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OF

PRACTICAL MEDICINE

BY

AMERICAN AUTHORS.

EDITED BY

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VOLUME IV.

DISEASES OF THE NERVOUS SYSTEM AND MIND—VASOMOTOR AND TROPHIC DISORDERS—DISEASES OF THE MUSCLES — OSTEOMALACIA — RACHITIS — RHEUMATISM — ARTHRITIS—GOUT—LITHÆMIA—OBESITY—SCURVY—ADDISON'S DISEASE.

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DISEASES OF THE PERIPHERAL NERVES.

DISEASES OF THE PERIPHERAL NERVES.

By FREDERICK G. FINLEY, M. D.

GENERAL PATHOLOGY OF LESIONS OF THE PERIPHERAL NERVES.

Etiology.—Most of the lesions of the peripheral nerves consist in various forms of traumatism. Many of the nerves are comparatively superficial, and are not infrequently divided by cuts or stabs, whilst the deeper ones are frequently divided or injured by gunshot or other penetrating wounds.

A more frequent cause of nerve injury is compression. Familiar examples are the pressure of a crutch on the musculo-spiral, or of paralysis of one or other of the arm nerves from pressure during sleep, an accident which is most apt to occur in intoxicated or in debilitated and bedridden patients. It seems probable that pressure destroys the continuity of the white substance of the nerve, and so sets up a secondary degeneration. Inflammatory changes in the nerve usually occur as the result of the injury and in the process of repair.

Compression frequently results from dislocations, fractures, the formation of callus, and by tumors, particularly malignant growths. During parturition the pelvic nerves of the mother are sometimes damaged, and the brachial or facial nerves of the infant,[1] from pressure or from the instruments or manipulations of the accoucheur. Neuritis in its various forms is one of the principal causes of motor paralysis, and will be discussed in a subsequent section.

Degeneration of the Peripheral Nerves.

Pathology.—The nutrition of the peripheral nerves is governed by certain nerve cells. In the case of a mixed nerve the motor filaments are connected with the large ganglion cells of the anterior cornua, whilst the trophic control of the sensory fibres is situated in the ganglion on the posterior nerve root.

If the nerve fibres are separated from the nerve cells, as in division of a nerve, there follows a degeneration of the whole peripheral portion

[1] Koster: *Deut. Arch. klin. Med.*, 1897, Bd. 58 (with bibliography).

below the point of section. The name of "Wallerian" degeneration is commonly applied to the changes which take place in the nerve, from the name of the physiologist who described them, and again the term of *secondary degeneration* is often used.

The process of nerve degeneration has been chiefly studied in animals, as opportunities for the examination of the processes in man are rare.

After division of a nerve the nuclei of the nerve fibres and the protoplasm about them enlarge, and compress and interrupt the continuity of the myelin and axis cylinders. The myelin breaks up into oily-looking globules, whilst the nuclei divide until two, four, or more are found in each internode. The myelin is absorbed, leaving empty and collapsed sarcolemma sheaths. Most observers agree that these changes occur simultaneously throughout the whole length of the peripheral nerve, although others regard the process as being a descending one, beginning at the cut end and spreading downward. The changes in the proximal end of the nerve do not extend beyond the first, or at most the second, internode above the point of section.

Regeneration succeeds the process of degeneration, and when complete leads to restoration of the function of the nerve. According to Ranvier, the process consists in an outgrowth of the central axis cylinders, which eventually become enclosed in a myelin sheath. Bowlby and others believe, however, that regeneration occurs in the distal end of the nerve even when completely divided. With our present views of the influence of trophic cells the latter view seems improbable, and, as pointed out by Gowers, the distal end of a cut nerve remains inexcitable to electric currents, thus rendering it improbable that perfect axis cylinders are formed in the distal segment. The process of regeneration is slow, and takes place in the second, third, and fourth month after division.

Symptoms.—The motor symptoms attending lesions of the peripheral nerves consist in paralysis of the muscles supplied by the affected nerve. The muscles are flaccid, and wasting occurs in the course of a few weeks. These effects vary with the degree of damage in the conducting power of the nerve. Very important data are obtained by the electrical examination of the affected nerves and of the muscles supplied by them. In this way the degenerative changes can be followed and their extent ascertained during life.

For the purpose of a complete electrical examination a faradic and a galvanic battery are required, in which the strength of the currents can be varied at will. The galvanic battery should be provided with a pole-changer and milliampèrameter to measure the absolute strength of the current. The electrodes, well moistened in salt solution, are placed one on an indifferent part of the body, as the back of the neck or the sternum, and the other on the nerve or muscle to be tested. It is convenient to provide the electrode placed on the nerve or muscle with a key to interrupt the current. Stintzing[1] recommends a circular electrode of a diameter of 2 cm. as the most convenient for applying to the nerve to be tested, whilst the large electrode placed on the sternum should have a measurement of 6×12 cm. This author has drawn up

[1] *Deut. Arch. klin. Med.*

useful tables showing the strength of currents required to produce contraction when applied to various nerves.

In health the application of a faradic current of sufficient strength to a nerve trunk causes a continuous contraction of the muscles supplied by it. The individual muscles may be stimulated through their nerves by applying the electrodes to the motor points. With the galvanic current, however, a single rapid contraction is obtained only on making or breaking the current or in changing its direction. The strength of the contraction also varies with the pole and with the opening or closing of the current. With a very weak current it is found that contraction is most rapidly produced by placing the negative pole or cathode on the nerve and closing the current. The formula applied is C. C. C., or cathodal closing contraction. On increasing the strength of the current contraction is next obtained by placing the anode over the nerve, contraction occurring sometimes with the opening, at other times with the closing, of the current, the result being expressed as A. O. C. (anodal opening contraction) or A. C. C. (anodal closing contraction). With a very strong current contraction, often tetanic in character, is obtained with the cathode on opening the current.

We have thus, under normal conditions, the reaction occurring in the following order:

Weak current,	C. C. C.
Stronger current,	A. C. C. A. O. C.
Very strong current,	C. O. C. or C. O. Te.

Both the faradic and interrupted currents applied to the muscle cause a rapid contraction due to stimulation of the peripheral portions of the motor nerve.

In nerve degeneration there is a marked difference in the reaction both of the nerves and of the muscles which they supply. The effect on the nerves is simpler, and will be considered first.

The rapid degeneration following any lesion of a nerve is accompanied by a diminution of irritability alike to both currents; or, in other words, a stronger current is required to evoke response to electrical stimuli on the diseased side. This diminution is present at the end of a few days or a week, and in severe cases irritability is completely lost at the end of a fortnight. In slight cases there is sometimes an initial rise of irritability, often more marked to momentary faradic shocks than to galvanism: the usual fall may never occur in such cases or there may be only a slight diminution in irritability.

In quite exceptional instances the behavior of the nerve to the two currents differs; thus a diminished or absent irritability to faradism, with an increased or unaltered irritability to galvanism, has been observed. The loss of nerve irritability is permanent where there is no recovery. In less severe cases, however, the nerve irritability to either current begins to return, being commonly preceded by a slight return of motor power. At the end of a few weeks in slight cases, up to several months or a year or more in severe ones, there is a gradual return of irritability.

In the muscles the changes of irritability are more complex, being not only of a quantitative, but also of a qualitative, character.

When the faradic current is applied directly to a muscle whose nerve is degenerated there is an absence of response, this form of electricity only evoking contraction through stimulation of the nerve endings. In cases of partial degeneration a somewhat stronger current than is required in health produces contraction. In point of time the changes in the faradic irritability of the muscles correspond with those in the nerve.

When the galvanic current, however, is applied to a muscle whose nerve is degenerated, a slow, wavelike contraction results, due to direct stimulation of the muscle itself, and quite different from the rapid and lightning-like contraction of health. In the first week a diminution of irritability corresponding with that of the nerve begins. About the end of the second week the slow contraction sets in, and is produced by a weaker current than is necessary to cause contraction under normal conditions. This increase in irritability is due to certain nutritive changes in the muscle. Not only is the irritability of the muscle increased, but it is often changed in quality. Instead of the cathodal closing contraction being most readily evoked, it often happens that the anodal closing contraction is produced by a current of equal strength or even by a weaker one. Again, the cathodal opening contraction may be greater than the anodal opening.

We then find in disease that contraction may occur in the following order:

1. $\begin{matrix} \text{C. C. C.} \\ \text{A. C. C.} \end{matrix}$ 2. A. O. C. 3. C. O. C.,

or 1. A. C. C. 2. C. C. C. 3. C. O. C. 4. A. O. C.

The slow, wavelike contraction is, however, much more constant than the qualitative changes, and when present is quite characteristic of nerve degeneration. In some very severe forms of nerve injury the galvanic irritability is rapidly lost, and the strongest currents which can be borne evoke no response.

These reactions in the muscle, the absence of faradic irritability, and with galvanism the slow contraction with change of quality, are spoken of as the reaction of degeneration. They indicate nerve degeneration, and are accompanied by loss of irritability in the nerve to both currents.

A partial reaction of degeneration is often seen in slight cases: it consists in a diminution only of the nerve irritability of the muscle, together with the slow contraction, increased irritability, and often changes in quality to the voltaic current.

The increased galvanic irritability of the muscle gradually diminishes and returns to the normal as the nerve recovers. There is recovery of power before the increased voltaic irritability passes off; with the return of faradic irritability the increased irritability in the muscle gradually lessens, and may fall below normal before returning to its original degree.

In cases of permanent paralysis, the galvanic irritability falls slowly after the rise, and, although persisting for a period of a year or two, gradually becomes extinct.

Sensation.—Lesions of sensory or mixed nerves cause loss of sensation in the areas which they supply. In disease or injury of a mixed nerve the sensory fibres usually suffer less and recover more rapidly than the motor; complete paralysis of the muscles may even be present without any defect in sensation. In explanation it has been assumed that sensory impulses are more readily conveyed than motor, or that the sensory fibres are regenerated more rapidly. In complete division of a nerve sensory symptoms, however, are sometimes slight or absent, and the theories mentioned are insufficient. The most rational explanation is that the anastomoses of the sensory fibres, which are very fine at the extremity of the limb, enable neighboring nerves to compensate for the paralyzed branches, this condition being termed *recurrent sensation.*

Both subjective and objective defects in sensation are frequent. *Pain*, either shooting along the course of a nerve or sometimes more diffuse, is present where there is any irritation. It is differently described as burning, boring, or throbbing, etc.: it may be more or less constant or paroxysmal.

More common than pain are various *paræsthesiæ*, such as numbness, formication, feeling of weight, heat, or cold, etc. In the extremities numbness is a very common symptom of a nerve lesion. Objectively various grades of anæsthesia may be observed, sometimes total, at other times very slight and only to be demonstrated by careful examination. Anæsthesia may coexist with pain, and is known as anæsthesia dolorosa.

Hyperæsthesia is not infrequent when there is nerve irritation, various sensory stimuli being felt more acutely than in health. Not infrequently sensation passes through different stages, hyperæsthesia being succeeded by diminished tactile sensation, and ultimately by anæsthesia. The different forms of sensation may be differently affected. Sensation to pain, touch, or heat and cold may be affected together or singly or in different combinations. It is, however, usual to find touch affected more than pain, the reverse being found in hysteria.

Heat and cold are sometimes confused, a condition termed paradoxical sensation. Loss of conducting power may be manifested by delay in transmission, particularly painful sensations.

Changes in sensation to galvanism of a qualitative character have been observed in lesions of the sensory nerves. Thus the anodal closing sensation has been found equal to, or stronger than, the cathodal closing sensation.

Reflex action is lost in the territory supplied by the affected nerve.

Vasomotor and trophic changes are frequent results of nerve lesions, the changes affecting the skin, bones, and joints.

The vasomotor fibres for the limbs run in the nerve trunks, and division of one of them causes an initial reddening and increase of surface temperature, In the course of a few days, or even in a few hours, the reddening and heat pass off, and the limb becomes colder than normal and often cyanotic. Owing to paralysis of the muscles the venous and lymphatic circulations are sluggish, and œdema is frequent.

The skin becomes thin and glossy, and slight injuries often produce obstinate sores or may even lead to gangrene. Various eruptions sometimes appear, of which herpes zoster is the most familiar. Pemphigus

is sometimes seen, the bullæ following the course of the injured nerve. The skin is dry, owing to atrophy of the sweat and sebaceous glands; the hair-follicles usually disappear, but exceptionally there is an increased growth. The nails become thickened, brittle, and rigid transversely or longitudinally. The bones in old-standing cases sometimes become thin, light, and easily fractured, whilst there is a tendency to pain, swelling, and fibrous ankylosis of the joints. In the fingers a glossy skin, with diminution in size from atrophy of the tissues, including the bone, is very characteristic.

Diagnosis.—In the diagnosis of peripheral lesions the paralyzed parts correspond with the distribution of the nerves, so that a thorough knowledge of their anatomical distribution is essential. In the sections on Neuritis and on Diseases of the Individual Nerves the differential diagnosis will be further dealt with (pp. 25, 38).

Prognosis.—In distinction to many other lesions of the nervous system affections of the peripheral nerves usually show a marked tendency to recovery. This tendency is naturally much influenced by the cause of the paralysis. When caused by pressure, contusion, etc. recovery is usually to be anticipated, although in some very severe injuries paralysis is permanent.

Much assistance in the prognosis may be gained by the electrical examinations. If, after the lapse of a week, it is found that the nerve irritability is increased or only slightly diminished, it may be concluded that the damage is slight and that recovery will occur within a few weeks.

Marked diminution of irritability at the end of a week is evidence of severe injury, and recovery cannot be anticipated before two or three months. Examinations made within the first three or four days do not yield accurate data for prognosis: later examinations give more definite results, and after the second week serious errors are not likely to occur.

Treatment.—In many cases the primary treatment is surgical. Where division of a nerve has occurred, it should, if possible, be treated by immediate suture. The results are usually very satisfactory, and failure seldom occurs when the aseptic technique is perfect. *Secondary* suture should be undertaken where there is evidence of division of a nerve. Bowlby[1] states that improvement is almost certain to follow, and that complete success may be anticipated in the majority of cases. This author records cases in which cure followed after division two years previously, and in one instance notable improvement after twelve years' standing.

After primary or secondary suture improvement first shows itself in recovery of sensation. In some instances there has been some return after twenty-four hours—a fact somewhat difficult to explain—although a period of some weeks or months usually elapses. Where there is marked atrophy of the muscles, it is usually several months before any notable return of power occurs.

In both primary and secondary suture Bowlby lays much stress on time as a healing factor. In many cases improvement or cure only occurs after a lapse of months or even two or three years.

In the treatment of paralysis from pressure the nerves in many

[1] Bowlby: *Injuries and Diseases of Nerves*, London, 1889.

instances recover on the removal of the cause. Thus in paralysis of the musculo-spiral from the use of a crutch disuse of the crutch may prove sufficient. In instances where cicatrices involve a nerve it should be freed from scar tissue and stretched. Recovery usually occurs spontaneously after compression by fracture, and even when the trunk is involved by callus. In other instances recovery has followed the removal of callus or spicules of bone pressing on a nerve trunk. Bowlby records many interesting examples of relief after the operations referred to.

Electricity is a valuable adjunct in the treatment of nerve lesions. Both the faradic and galvanic currents are used, the clinical rule being to use the one which most readily produces contraction without pain. When irritability to faradism is present, a current sufficiently strong to produce contractions may be applied, one pole being placed over the motor point and the other passed over the affected muscles. The irritability of the nerve is in this manner distinctly raised. After a few applications a weaker current evokes contraction, and it is probable that recovery is hastened.

The galvanic current is, however, principally used in nerve injuries. One pole may be placed on the back of the neck and the other moved over the affected muscles, especially their motor points, so as to produce contractions. Applications may be made daily or two or three times weekly for from ten to twenty minutes. Whilst undoubted benefit results, the mode of action is still obscure, but it may be stated that it is important to obtain muscular contractions.

Massage is a valuable measure in maintaining the muscular nutrition, and may be used in conjunction with electricity.

Deformity from overaction of opposing muscles should be prevented as far as possible by mechanical contrivances.

Drugs are not of very great value in most cases of peripheral paralysis. Iodide of potassium is used in neuritis, and strychnine is also administered with a view to hastening recovery. General tonics, such as iron, quinine, or arsenic, are useful in debilitated conditions.

NEURITIS.

Under the heading of "Neuritis" it is usual to include any affection of a nerve leading to paralysis of the parts supplied. Strictly speaking, many of the lesions consist in degeneration of the nerve fibres rather than inflammation, but for clinical purposes this distinction may be disregarded.

A single nerve or group of nerves may be affected, or, again, many nerves throughout the body may be involved, when the condition is spoken of as multiple neuritis.

Etiology.—The commonest cause of neuritis is some form of traumatism. It is met with after blows, wounds, or contusions, from prolonged pressure, or from compression by growths, such as syphilis or cancer, by the callus of a fractured bone, or even by violent contraction

of muscles through which certain nerves pass. Dislocations are frequently followed by paralysis, which is most commonly seen in the shoulder or arm from subluxation of the humerus.

Extension of inflammation from neighboring parts occasionally involves certain nerves. The cranial nerves are thus frequently compressed by basilar meningitis, and the spinal roots are occasionally involved in a similar way.

Cold has been gradually largely eliminated as a potent cause of neuritis, but in special instances, as in the facial and sciatic nerves, it is an important factor.

Rheumatic individuals, especially those who suffer from lumbago or chronic inflammation of fibrous structures, are frequently attacked by neuritis of the sciatic, and occasionally of the brachial, nerves. Gout, again, is an important predisposing cause.

Pathological Anatomy.—Inflammation may begin in the connective-tissue sheath, especially when a single nerve is affected (*perineuritis*), and thence sometimes spread to the interstitial tissue of the nerve. In other cases, especially in multiple neuritis, the nerve fibres themselves are primarily affected by a degenerative process (*parenchymatous neuritis*).

In the former case the nerve trunk is red and swollen at the damaged part, which is usually limited to a small extent. Several foci, however, may be present or even a considerable tract of a nerve involved. The sheath is infiltrated by leucocytes which sometimes extend along the connective tissue between the individual bundles or even the fibres of the nerve.

The axis cylinders suffer in proportion to the pressure exercised upon them, and undergo degeneration. In accordance with Waller's law, the degeneration extends down to the periphery of the nerve, owing to the fibres being cut off from their trophic cells.

In parenchymatous neuritis the changes begin in the nerve fibres themselves. The nuclei of the nerve fibres and the protoplasm about them enlarge, and compress and interrupt the continuity of the myelin and axes cylinders. The myelin breaks up into oily-looking globules, which float in the protoplasmic substance. The nuclei soon proliferate, whilst the products of degeneration are absorbed and leave collapsed sarcolemma sheaths, enlarged at places by nuclei with their surrounding protoplasm and remnants of myelin resembling oil globules.

Experimentally it has been shown that the electric irritability of the nerve is lost at a period corresponding in time with the break of continuity in the myelin and axis cylinders. In man irritability to electrical stimuli is lost during the second week, so we may infer that these changes occur at that period. The proliferation of nuclei is suggestive of inflammatory reaction, and this is indeed further borne out by the occasional appearance of leucocytes in the connective tissue of the nerve trunk.

Important secondary changes occur in the muscles. They undergo atrophy and are distinctly reduced in size. Microscopically the fibres become smaller and granular and their striæ less distinct. The connective tissue of the sheath and muscle proliferates, showing numerous areas of round cells. If recovery does not ensue, the newly-formed

connective tissue contracts and a condition of cirrhosis and permanent contraction results.

Symptoms.—The symptoms of neuritis are subject to considerable variation, depending on the acute or chronic character and the locality affected.

Acute perineuritis is sometimes ushered in by chilly sensations or a rigor with elevated temperature. The essential local symptom of an acute perineuritis is pain, referred along the course of the nerve. Its onset is usually acute, seldom gradual; it is of an extremely severe character, and is described as boring or burning; it is increased by any movement of the limb, and often subject to marked nocturnal exacerbation. Not only is the nerve itself exquisitely tender to pressure, but the bones and muscles may be unduly sensitive. When superficial the nerve occasionally presents distinct swelling.

Various subjective sensations are often present, such as numbness or tingling. In the early stages there is cutaneous hyperæsthesia, followed by diminution of sensation over the parts supplied by the nerve, and patches of total anæsthesia occur in severe cases. Slight inco-ordination is sometimes prominent, and if the arm is affected shows itself in inability to use the hand in delicate movements. Depending on the degree of damage to the nerve fibres, there is more or less loss of motor power in the muscles, with atrophy, flaccidity, and the electrical reactions characterizing nerve degeneration. In many instances, however, the loss of power is apparent rather than real, and is due to the pain induced by putting the muscles into action.

Many cases of neuritis are, however, not marked by pain, but present paralysis as the prominent feature. Painful cramps and fibrillary twitching may also occur. Various vasomotor and trophic symptoms are often observed. The skin may be hot, red, and moist, the surface temperature is elevated in the early stages, and œdema is not uncommon. In old-standing cases the limb is cold, the skin becoming thin and glossy, with loss of the hairs. The nails are stunted and ridged, and their growth slower than in health. Various eruptions, attributed to withdrawal of trophic influence, are sometimes present. Slight injuries may produce ulcers which are slow to heal, and bullæ or vesicles occasionally form in parts supplied by the affected nerve.

Ascending neuritis or neuritis migrans is not an uncommon affection. Here the inflammation ascends the nerve trunk, and if it reaches a plexus may be spread to a number of nerves. It results from some injury of a peripheral nerve, often traumatic, and extending up may cause considerable thickening and induration, involving consecutively one or more nerve trunks. Thus a trifling wound of a digital nerve occasionally gives rise to an ascending lesion involving the brachial plexus. The course of the affection is usually more insidious and chronic than a primary neuritis of the larger nerve trunks, and it often continues spreading for a period of many weeks.

Course.—Slight cases may recover in a few weeks, but severe ones last for several months, or complete recovery may exceptionally not occur for years. Permanent paralysis, although rare, is an occasional result. In the severe forms of the disease the condition passes into a chronic stage, and pain and weakness continue for months.

Diagnosis.—Although often confused with neuralgia, the local evidence of inflammation, such as tenderness of the nerve, should prevent such an error. When paralysis or anæsthetic areas develop the condition is unmistakable.

Prognosis.—The severity of the symptoms and the condition of the electric irritability of the nerve form the best guides to prognosis. Where we have the complete reaction of degeneration the condition will continue for months. In elderly people pain may last for years.

Treatment.—As in other forms of inflammation, complete rest is essential. In neuritis of a limb a splint is often serviceable to secure this end, and the part should be comfortable, supported by pillows to avoid pressure.

To relieve pain an icebag over the nerve is an excellent measure in the early stage. Hot fomentations of lead and opium (liq. plumbi subacet., tr. opii, āā. ʒij ; aq. ad Oj) are also very serviceable. It is probable that both these methods act by diminishing the vascularity of the nerve. Leeches, if applied early, are useful, and blisters are also used.

A brisk saline purgative, especially in gouty cases, is serviceable at the outset, and small doses of calomel or blue mass often exercise a markedly beneficial influence. Of other drugs, iodide of potassium (gr. v to xx) in a few cases gives marked relief, and salicylate of soda is also serviceable in relieving symptoms in a small number of cases.

In the chronic stage galvanism often relieves pain for hours. There is some recent evidence to show that this agent tends to prevent nerve degeneration, and it may be used early with this view. Blisters are of great value, and may be applied along the course of the nerve.

Faradism is of no value in the early stages, and only increases the pain. Later it is useful in relieving paræsthesiæ, and, used with a wire brush, sometimes alleviates pain.

Massage of the limbs and general tonics or change of climate are also indicated in the later stages of the disease.

MULTIPLE NEURITIS.

Synonyms.—Peripheral neuritis ; Polyneuritis.

Definition.—A symmetrical degenerative affection involving simultaneously many nerves.

Etiology.—Multiple neuritis is invariably due to some toxic agent.

Of all the causes, with the exception of lead and diphtheria, alcohol is by far the most frequent. It occurs in individuals who use stimulants to great excess and for prolonged periods. Although more common in spirit-drinkers, it is also produced by malt liquors. It is usually stated to be more frequent in women than in men, but in Ross's analysis of 90 cases only 41 were females. When we consider the much greater number of male alcoholics, we must admit a greater susceptibility in the female sex. Exposure to cold and improper food often co-operate in producing the disease.

Of the other diffusible stimulants, carbon monoxide gas evolved

from charcoal stoves, the use of roburite as an explosive, and the fumes of aniline and bisulphide of carbon are known to produce multiple neuritis.

Of the various infectious diseases, diphtheria is most frequently followed by paralysis, and injections of either the bacillus or its toxin have been shown experimentally to produce nerve degeneration. Judging from analogy, it seems probable that paralysis following other infectious processes is also due to toxins produced by various micro-organisms.

Neuritis is not very uncommon after typhoid fever, and it is occasionally seen after measles, pneumonia, influenza, scarlatina, varicella, variola, and erysipelas. Septic infection, with or without other manifestations of sepsis, is a rare cause of the disease. In a case recorded by Roth a man developed extensive and fatal peripheral neuritis almost six weeks after the healing of a stab wound. In other instances the affection has followed some local septic process. In a case recently under my care the only source of a multiple neuritis was cystitis following an old stricture. Dana[1] has recorded a somewhat similar origin from a pyelonephritis. It seems probable that syphilis[2] is a rare cause of multiple neuritis, and that recovery takes place with antisyphilitic remedies. Inasmuch as mercurial inunctions have been shown to be followed by this affection, care is requisite in distinguishing such cases from syphilitic ones.

Neuritis occasionally develops in the course of tuberculosis, and it is said to be frequent in a latent form. The occurrence of the disease in diabetes, although rare, is of interest in explaining the origin of obstinate neuralgia which occasionally occurs in this affection.

Rheumatic cases are those developing after exposure to cold. As numerous nerves are involved, it is probably correct to regard cold rather as a predisposing cause, lowering the resisting power and rendering the nerves susceptible to a poison allied to or identical with that of acute rheumatism. Although such cases are rare, their occasional occurrence is well established. In rare instances acute rheumatism is followed by multiple neuritis.

Cases of neuritis are not very infrequent in chorea, a condition closely allied to rheumatism. It seems probable, however, that arsenic, which is so generally used in this affection, is really the toxic agent.

The paralytic symptoms in beri-beri and leprosy are due to neuritis, and in the latter the bacilli settle in the nerve trunks.

Certain cases of paralysis occurring in malaria have been attributed to neuritis, but they are of extremely rare occurrence.

Of the metallic poisons, lead is by far the most frequent, whilst arsenic and mercury are occasional causes.

Various cachectic conditions, such as cancer or anæmia, frequently present pathological evidence of nerve degeneration, and it is not improbable that various symptoms, such as weakness and numbness, should be attributed to this factor. During or following confinement a few cases of multiple neuritis have developed.

[1] Dana: *Med. News*, 1889.

[2] For cases see Buzzard: *Trans. Clin. Soc.*, 1874 and 1880; R. W. Taylor: *N. Y. Med. Journ.*, lii; Tuckwell: *Lancet*, 1882, i.; Fordyce: *Journ. Cutan. and Genito-urin. Dis.*, 1891.

Arterial sclerosis[1] in elderly people sometimes leads to necrosis and inflammation of the nerve trunks, but the symmetry seen in the various forms of toxic neuritis is here less marked.

Finally, a number of affections, such as Raynaud's disease, perforating ulcers, and rheumatoid arthritis, are attributed by some writers to primary nerve lesions. Many if not all cases of Landry's disease are doubtless of this nature, but further observations on this point are required.

Pathological Anatomy.—The chief changes occur in the nerve fibres, which undergo parenchymatous degeneration precisely similar to that described under the heading of Neuritis of Individual Nerves. Associated with this there is, however, occasional evidence of inflammatory changes in the nerve sheaths, consisting in infiltration with leucocytes, and even visible swelling and redness in recent cases.

The changes in the nerves are more pronounced at the periphery, and indeed may only be visible within the muscles. The muscles undergo secondary changes similar to those described under Neuritis, and these effects are similar in whatever part of the nerve trunk the disease is situated. The nerves of the limbs are those showing the most advanced changes.

Degeneration has also been found in the ocular and bulbar nerves, but in some of these instances the lesions have been central, and in others careful investigation has failed to reveal any anatomical changes. In a few cases associated changes have been found in the spinal cord, consisting in patches of chronic myelitis or meningitis, especially in alcoholic cases; in the gray matter shrinking and vacuolation of the ganglion cells sometimes occur.

Symptoms.—The symptoms vary considerably in different instances. Gowers distinguishes three classes according to the prominence of motor or sensory symptoms or of inco-ordination. Although pure types of each class exist, it is usual to find two or more of them combined.

The disease usually sets in abruptly, but there may be a premonitory period extending over weeks or months, marked by numbness and tingling, especially in the hands and feet, and by muscular cramps. In the acute cases the onset is sometimes like that of an acute infectious process. The temperature rises to 102° or 104° F., with the usual febrile symptoms and occasionally slight albuminuria, splenic enlargement, or jaundice. Severe pains in the limbs, with slight articular swelling, are sometimes present, and may at first resemble acute rheumatism. Numbness and tingling in the extremities, and often some pains and muscular cramps, precede or accompany the loss of power which is early noticed in certain groups of muscles. The extensors of the hands and fingers and the anterior tibial group of muscles are those chiefly affected, and the resulting wrist- and foot-drop is very characteristic. Another symptom which is very characteristic is muscular tenderness, which is especially prominent in alcoholic cases, and is then often so marked that the slightest change of position or pressure on the muscles causes extreme pain.

Paralysis may develop rapidly, almost suddenly, and be complete in the course of a day or two. In other instances the onset is more gradual,

[1] Pitres and Vailliard: *Rev. de Méd.*, 1886.

and paralysis becomes complete in the course of two or three weeks, or paresis only may be present. Either the arms or legs may suffer, but always in a symmetrical fashion. Most commonly both upper and lower limbs are affected, and the legs are more often paralyzed alone than the arms. The muscles affected are chiefly those at the periphery of the limb, below the knee and elbow, particularly the homologous groups supplied by the musculo-spiral and external popliteal nerves. In slight cases the paralysis may be limited to the muscles supplied by the external popliteal nerve, but it often spreads to the muscles of the foot and those at the back of the lower leg. The thigh and upper-arm muscles are much less frequently affected, and those acting on the hip- and shoulder-joints still less seldom and only in cases of very extensive paralysis. Weakness of the muscles of the front of the leg gives rise to the "steppage" gait, the thighs being unduly flexed to enable the dropped feet to clear the ground.

In the arms, in addition to the extensors of the hand, the small muscles of the hand frequently suffer. In neuritis due to lead these two groups are most frequently attacked, and severe paralysis in these localities, without involvement of other groups of muscles, is seldom due to any other causes.

The muscles of the back, neck, thorax, and abdomen are not often attacked, and then only in severe cases. Paralysis of the diaphragm or other muscles of respiration is very apt to prove fatal.

Rapid heart action, with paralysis of the larynx, has also been met with from involvement of the pneumogastric, and attacks of dyspnœa or orthopnœa occur from paralysis of the diaphragm and other muscles of respiration.

The sphincters are very seldom involved without a lesion in the cord.

Apart from diphtheritic neuritis, the cranial nerves are not usually involved, but numerous instances are recorded in which one or more have been affected. The ophthalmoscope has in rare instances revealed optic neuritis, and paralysis of the third, fourth, and sixth nerves has also been observed, and slight weakness of these muscles may be evidenced by nystagmus. Inequality of the pupil and slow or absent reaction to light and accommodation has been occasionally observed.[1] Paralysis of one or both facial nerves, of the laryngeal and bulbar nerves, may also occur.

The nutrition of the muscles suffers; they become flabby and wasted, and in consequence of contractures of their opponents deformities are apt to occur which are troublesome to correct during convalescence. Of these foot-drop and flexion of the knees are the most frequent.

Sensory disturbance is commonly manifested by various subjective phenomena, such as numbness, tingling, or a feeling of pins and needles, felt especially in the hands and feet. Objective blunting of sensation is often completely absent, or, if present, incomplete. It shows itself in diminished tactile or painful sensations, in delayed sensation, and at times is incorrectly located. Marked hyperæsthesia is common during the earlier stage of the malady, and later on may be replaced by anæsthesia.

Fibrillary tremor of the affected muscles is often present. More or

[1] Reunert: *Deut. Arch. klin. Med.*, l. Case xxiv.; Oppenheim: *Zeit. klin. Med.*, xi.

less inco-ordination is not uncommon, the patient noticing that he has difficulty in using his hands for delicate manipulations or finding a certain amount of unsteadiness in his gait. In rare instances inco-ordination may form the most prominent feature of the disease, and the ataxia so produced is termed "neuro-tabes." Romberg's sign is sometimes well marked and increases the resemblance to locomotor ataxia.

A very important sign, always present in severe cases and seldom absent in mild ones, is absence of the knee jerk, which often disappears even before paralysis sets in. There are, however, mild cases in which it is present, and may even be increased in the early stages.

The skin reflexes vary considerably and are of little diagnostic value. Although occasionally increased, they are usually absent in severer forms of the disease.

Various vasomotor and trophic changes are often seen. Slight œdema about the ankles and back of the hands is common. Dejérine[1] has recently recorded a case in which all four limbs presented a marked œdema, but this is extremely rare. In a case recently under my care minute hemorrhages were associated with œdema of the lower extremities. Bedsores are rare, but glossy skin, loss of hairs, and trophic changes in the nails and articular adhesions are not uncommon.

Electrical Changes.—In acute cases with much pain and tenderness it is inadvisable to put the patient to the pain of an electrical examination. In other cases, however, the irritability of the nerve and muscles is of value both for diagnosis and prognosis. The changes found correspond with those in any condition of nerve degeneration. Owing to secondary changes in the muscles, a strong galvanic current may be required to evoke response in the muscles—a sign, according to Starr, of some value in the diagnosis of multiple neuritis. Many modifications of electric irritability occur and are referred to elsewhere.

Course.—Very severe cases may prove fatal in from one to two weeks from paralysis of the respiratory muscles, but these are exceptional. In all severe cases the course of the disease is very tedious, whilst recovery is gradual and extends over a period of months or even a year or more. The motor power returns slowly, the spontaneous pains cease, but the muscular tenderness often persists for a lengthened period. Ultimately complete recovery is the rule, even in extensive paralysis with contractures which have lasted as much as a year.

Complications.—Pulmonary complications of a pneumonic character are not infrequent when the respiratory muscles are affected, and are often the direct cause of death. Pulmonary tuberculosis, again, is frequent in alcoholic cases, and persistent fever should always suggest a careful examination of the lungs.

Other complications are the result of the original cause of the neuritis. In alcoholic cases cirrhosis of the liver, chronic nephritis with cardiac enlargement, and in lead-poisoning chronic cardio-vascular and renal complications, occur.

Special Forms.

Alcoholic Form.—In this form there is in women often difficulty in ascertaining the cause, and indeed the onset of symptoms may be the

[1] Dejérine: *Rev. de Méd.*, Jan., 1897.

first hint that the patient is addicted to alcoholic excess. There is frequently a premonitory period extending over several months in which there are cramps, numbness, or slight inco-ordination. Paralysis generally begins in the legs, and simultaneously or a little later in the arms, giving rise to foot-drop and wrist-drop. Ross and Bury refer to severe colic resembling that of lead, and also to various visceral neuralgiæ. The pulse is often rapid, weak, and irregular, whilst vomiting, due to associated gastric catarrh, is not uncommon.

Mental symptoms similar to those produced by prolonged alcoholic excess often occur. The memory is lost for recent events; the patient is frequently suspicious of attendants or friends and subject to various delusions. Other forms of mental aberration also occur, and may terminate in dementia. Convulsions are occasionally seen. There may be tremor of the limbs or of the lips and tongue, accompanied by thickness of speech simulating general paralysis. Any exacerbation of symptoms should awaken a suspicion that the cause is still operative, and too much caution cannot be exercised in preventing servants or attendants from secretly supplying stimulants.

Recurring Neuritis.—A few cases of recurring neuritis have been described, two and even three attacks having taken place. The interval between successive attacks has varied from a few months to several years.

The nerves affected in the second and third attack are usually those involved in the first. In Sorgo's case[1] the nerves of the arm were involved in the first two attacks, whilst the third, which proved fatal, affected in addition the diaphragm, left vocal cord, and the muscles of deglutition. Sherwood,[2] who gave the first description of the condition, also found the nerves affected were identical in two cases; in a case of Targowla's the facial nerves were affected on different sides in each attack.

Déjerine[3] has recorded two cases in which the ocular and bulbar nerves were affected with the limbs and trunk, and in which none of the known causes were discoverable.

Senile Form.—Under this name Oppenheim[4] has described a series of cases in which none of the ordinary toxic influences could be traced. He attributes the condition to arterial sclerosis with interference with the vascular supply of the nerves. Both arms and legs are usually involved; the motor and sensory paralysis is imperfectly developed, whilst pain is slight or absent. There is not the same tendency to recover as in toxic cases; the onset is insidious and the course chronic. Joffroy and Achard[5] found interstitial and parenchymatous changes in the sciatic nerves in a case of arterial sclerosis, which they traced to thrombosis of the vessels supplying the nerves. Such cases are apt to be associated or followed by gangrene of the extremities, due, like the neuritis, to primary vascular changes.[6] Of other forms of neuritis

[1] Sorgo: *Zeit. klin. Med.*, 32, Supplement.
[2] Mary Sherwood: *Virchow's Arch.*, 123, i.
[3] Déjerine: *Semaine méd.*, 1891, p. 177.
[4] Oppenheim: "Ueber die Senile Form d. Mult. Neuritis," *Berl. klin. Woch.*, 1893, p. 589.
[5] Joffroy and Achard: *Arch. de Méd. exp.*, 1889, p. 229.
[6] Dutil and Lamy: *Ibid.*, 1893, p. 102.

meriting a special description, the most important are those due to diphtheria and other fevers, metallic poisoning, beri-beri, and leprosy, all of which will be found described under their appropriate headings.

Prognosis.—From what has been already stated it will be gathered that the prognosis is in the majority of cases favorable. Complete recovery is the rule, but death may occur from involvement of the respiratory muscles or from pulmonary complications, particularly pneumonia or tuberculosis.

Sudden cardiac failure sometimes sets in, and is usually associated with degeneration of the heart muscle, and sometimes of the pneumogastric nerve.

Diagnosis.—Multiple neuritis may be mistaken for poliomyelitis affecting two or more limbs, as in both affections there are pains, weakness, and wasting of muscles and an acute onset. Where anæsthesia is present and pains persistent such an error is not likely to occur, but in cases of neuritis without pain or anæsthesia the distinction is less easy. The history of a cause such as alcohol or diphtheria, and the presence of muscular tenderness, are points indicating neuritis, whilst poliomyelitis is rare except in childhood; it is less symmetrical and recovery is incomplete.

Ataxia when prominent may lead to confusion with tabes dorsalis. When associated with weakness of the anterior tibial muscles there is no difficulty in pronouncing the condition one of neuritis. There are, however, cases of neuritis in which ataxia with sensory symptoms are present, and an entire absence of motor weakness. True lightning pains, the Argyll-Robertson pupil, and bladder disturbance are characteristic of tabes, and a high degree of ataxia is also suggestive of this condition. An acute and subacute onset of ataxia suggests neuritis, although it by no means excludes tabes. Déjerine and Sollier[1] have recorded a remarkable case of neuritis of fifteen years' standing with lightning pains, sensory disturbance, ataxia, the knee jerks preserved, slight myosis with a pupil reacting slowly to light, and not to accommodation, in which a diagnosis of tabes with involvement of the lateral columns was made. In such an instance a correct opinion would seem impossible.

Confusion with acute or subacute myelitis sometimes occurs. The features which characterize a spinal lesion are a girdle sensation, involvement of the bladder and rectum, and increased myotatic irritability with ankle clonus. Spasm of the muscles is again frequent in spinal lesions, and contrasts strongly with the relaxation in neuritis. The paralysis and sensory defects are more diffuse in spinal lesions, many groups of muscles being involved, as compared with the special groups in neuritis and the frequent localization of paræsthesiæ at the extremities of the limbs.

Treatment.—In the treatment of multiple neuritis it is of the first importance to recognize and arrest the cause. If alcohol is suspected, but not admitted, a trustworthy nurse should be put in charge or the patient placed in hospital. If stimulants are being used, they must be withdrawn, although in cases with a weak or rapid heart it may be advisable to do so gradually and substitute cardiac stimulants, such as

[1] Déjerine and Sollier: *Arch. de Med. exp.*, 1889, p. 251.

digitalis, strophanthus, or strychnine. In all but very slight cases absolute rest in bed is essential. The morbid process is probably intensified by movements, and any exposure is apt to cause exacerbations in the disease. To relieve the pains hot fomentations are very useful, but care must be exercised when marked anæsthesia is present to avoid blistering the limbs, as the nutritive functions are at a very low ebb. If the patient can bear the manipulation, a warm bath daily for fifteen to twenty minutes gives great relief. In less severe cases the affected parts may be wrapped in cotton wool. The various drugs used to relieve pain are often required. Morphine hypodermically is the most efficacious, but five grains of phenacetine or ten of antipyrine are sometimes sufficient. With a weak heart, however, the coal-tar preparations must be used with caution.

Atropine gr. $\frac{1}{120}$ to $\frac{1}{60}$ and cocaine gr. $\frac{1}{4}$ hypodermically are also recommended; also cannabis indica in pill form, gr. $\frac{1}{8}$ to $\frac{1}{4}$. (See Neuritis, p. 28).

It is of much importance to prevent contractures and false positions. The foot should be supported by a heavy sandbag or a suitable splint, and the knees and hips should be kept straight. If contractures occur, it is seldom necessary to divide tendons for their relief, as with manipulations and use they usually recover. Drugs have no specific influence on the morbid process in the nerves. Strychnine and arsenic are commonly recommended, but the latter must be used cautiously, inasmuch as even moderate doses have been known to produce the disease. Any benefit derived from such remedies is probably through their action as general tonics. Mercury is of no use in parenchymatous neuritis, but iodide of potassium is said to be of value in sensory forms of the disease, and it also proves of undoubted benefit in the neuritis of chronic metallic poisoning.

In cases due to cold, salicylate of soda is advised, and if there is any malarial influence, quinine should be freely given.

General tonics, such as iron and cod-liver oil, with a carefully regulated diet, are important.

Massage and electricity are both measures of much use when properly applied, particularly in the later stages of the disease. During the acute stage it is essential that neither of these measures should be used in such a way as to produce pain. A weak galvanic current is also of some use in relieving pain, the positive pole being passed over the painful nerves and muscles. After the acute stage is passed both measures may be used freely to maintain the nutrition of the muscles. The galvanic current used should be just sufficient to produce contraction of the muscles.

The faradic current is useful in combating various paræsthesiæ, and is also valuable in slight cases when it evokes muscular contraction.

TUMORS OF NERVES, OR NEUROMATA.

Definition.—Tumors in connection with nerves are spoken of as neuromata. When made up of nerve fibres they are termed true neuromata: it is, however, much more common to find these growths com-

posed of fibrous, myxomatous, or sarcomatous tissue, when they are termed false neuromata. The true neuromata may contain medullated or non-medullated fibres, and in a very few instances nerve cells have been present.

In syphilis and leprosy there are specific growths in connection with nerves, and in the latter they often constitute some of the cardinal features of the disease.

Single neuroma is commonly found in connection with the stumps of nerves in amputated limbs; any injury of a nerve is also a well-recognized cause. Several tumors sometimes exist on a single nerve.

SYMPTOMS.—The chief symptom is severe pain, situated both in the neighborhood of the growth and reflected along the affected nerve trunk. As the nerve fibres are seldom destroyed, paralysis and anæsthesia are rare. The tumors, which vary in size from a pea to a large growth, are often exquisitely tender to touch, and pressure has been known to produce spasm of the muscles supplied by the affected nerve. A few instances are recorded of growths of one of the spinal roots[1] proving fatal by pressure on the cord.

DIAGNOSIS.—The diagnosis is readily made when a growth lies in connection with a superficial nerve, particularly when associated with violent pain, either spontaneous or induced by pressure, along the course of the nerve. When deep seated, a neuroma is usually indistinguishable from various other forms of irritation or neuritis.

TREATMENT.—Removal is usually followed by relief. The growth can generally be dissected away from the nerve, but in some instances it has been found necessary to excise a portion of nerve and suture the ends.

Multiple neuromata are frequently congenital or appear in early life, but they may commence at any age. Prudden[2] in a valuable paper has shown that the disease is three times more common in males than females. The affection is hereditary in a few instances, or brothers[3] may be affected. Occasionally its subjects are members of a neuropathic family or are themselves weak-minded, idiots, or cretins. The sexual organs have been imperfect in some instances, and several cases have been associated with molluscum fibrosum or elephantiasis.[4]

PATHOLOGICAL ANATOMY.—Multiple neuromata vary in size from minute nodules to growths the size of a child's head. They are often present in enormous numbers, and may be in connection with almost any nerve in the body. The cranial nerves are sometimes attacked within the skull, and the growths have caused symptoms of cerebral tumor. Usually, however, these nerves are involved outside the skull, and extensive growths extending over long areas of nerve have been observed on the pneumogastric, hypoglossal, and other cranial nerves.

The spinal nerves may suffer in all parts of the body, great numbers of tumors often lying in connection with a single nerve, and large growths being attached to the limb plexuses. The sympathetic and visceral nerves are also attacked, but less frequently.

[1] Cases quoted by Bowlby: *Injuries and Diseases of Nerves.*
[2] Prudden: *Am. Journ. Med. Sci.*, vol. lxxx., 1880.
[3] Generisch: *Virchow's Arch.*, xlix. p. 15.
[4] Payne: *Trans. of Path. Soc.*, vol. xxxviii.

Painful subcutaneous tubercles containing nerve fibres and representing neuromata of the cutaneous nerves are sometimes associated with neuromata of the nerve trunks or they may occur alone. A good example of the former has recently been recorded by Reynolds.[1] The large growths are sometimes made up of a multitude of smaller ones.

In most instances the tumors are fibromata; less often they are of a myxo-fibromatous or sarcomatous nature. Cysts of considerable size may occur in connection with the larger tumors. It is sometimes possible to separate the nerves from the growths by dissection, and in some instances the fibres of the nerve pass uninjured through the growth; usually, however, they are damaged by compression. It is questionable whether there is ever any actual proliferation of nerve tissue: as Reynolds points out, it is an extremely difficult matter to count the fibres in any given section, especially when separated and altered by the new growth.

Symptoms.—In a considerable number of cases (12 out of 26 of Prudden's series) no symptoms are observed apart from the presence of tumors. Pain, sometimes of an exceedingly severe character, either spontaneous and occurring in paroxysms or induced by pressure, occurred in half of the same series of cases. Anæsthesia, however, is rare. Motor paralysis and trophic disturbances occasionally occur.

The tumors are sometimes felt as knobbed cords lying along the course of nerve trunks, or single tumors can occasionally be felt in connection with superficial nerves like the ulnar. Large growths are usually connected with the brachial, lumbar, or sacral plexuses.

In addition to the deeper-seated growths of the nerves, numerous small sessile growths, sometimes very sensitive to pressure, are often found in the subcutaneous tissues. In a case recorded by Duhring[2] elevated cutaneous nodules gradually developed about the shoulder and arm of a man æt. sixty, and were associated with violent paroxysms of pain in the arm, always increased by cold.

Death usually follows from intercurrent disease, and in quite a number of instances has occurred rapidly from severe diarrhœa and vomiting. Although the growths may exist for many years, these patients often die before attaining middle life.

Diagnosis.—In many cases the absence of symptoms renders the condition obscure. The presence of multiple growths in the course of a nerve trunk, especially with signs of interference with its function or severe pain, would suggest neuromata. Sarcomatous subcutaneous nodules closely resemble cutaneous neuromata, but this affection runs a much more rapid course. The association of molluscum fibrosum, elephantiasis, or of the neuropathic indications referred to above would support a diagnosis of neuromata.

Treatment.—No known treatment is known to influence this condition. In Duhring's case division of the plexus relieved the pain for several years.

Plexiform Neuromata.—These growths are composed of nerve fibres, and are therefore true neuromata. The statement made about multiple neuromata in regard to family and hereditary predisposition

[1] Reynolds: *Medical Chronicle*, Dec., 1896.
[2] Duhring: *Am. Journ. Med. Sci.*, vols. lxvi., lxviii., lxxxii.

applies also to this condition. They are noticed in early life, and some are probably congenital.

Symptoms.—These tumors occur especially about the forehead, temple, or neck, and form soft growths in which cords and nodules are sometimes felt. The skin covering them is unaltered, and apart from the disfigurement they rarely cause symptoms.

Treatment.—The growths have been excised to remove deformity. Recurrence took place in a case reported by Labbé.

DISEASES OF THE CRANIAL NERVES.

Diseases of the Olfactory Nerve.

Loss of the sense of smell is much more frequently due to local disease of the nasal mucosa than to actual lesions of the olfactory nerve. Trophic changes resulting from disease of the fifth nerve also lead to diminution or loss of the sense of smell.

The olfactory bulbs may be damaged by tumors in the anterior fossa of the skull and by syphilitic or other lesions in this locality. In hydrocephalus they may suffer from pressure. Falls on the back of the head have repeatedly caused loss of smell, owing probably to the delicate nerve filaments being torn from the bulbs, whilst fractures or penetrating wounds have also produced the same result. Tabes is seldom accompanied by anosmia. It has been suggested that a cerebral tumor sometimes sets up a neuritis comparable to that seen in the optic nerve, so that loss of smell has of itself no localizing value. Powerful olfactory stimuli, such as offensive smells, are said to sometimes produce permanent loss of smell. A few cases are recorded in which the loss of smell was congenital.

Not only is the sense of smell lost in disease of the olfactory bulb, but also the perception of flavors, so that patients complain of losing taste. In testing smell, volatile substances like camphor or asafœtida should be used; ammonia stimulates the fifth nerve, and thus cannot be employed.

In rare cases of central disease anosmia is sometimes present, and the centre is supposed to be in the uncinate gyrus.

Treatment.—The treatment of anosmia depends on the cause. When due to nerve disease it is unsatisfactory unless syphilis is the causal agent. Strychnine may be tried, and is known experimentally to stimulate the nerve.

DISEASES OF THE OPTIC NERVE.

The Optic Nerve.

In this section we can only deal with certain affections of the nerve of special interest to the physician.

The fibres from each optic nerve pass back to the chiasm, where a

partial decussation takes place. The fibres passing to the optic tract are derived from the temporal side of the retina of the same eye and the nasal half of the opposite eye; the fibres from the nasal portion of each retina are placed toward the centre, whilst those from the temporal portion are situated at the periphery of the chiasm. A reference to the diagram will render this arrangement clear. The optic tracts pass to

FIG. 1.

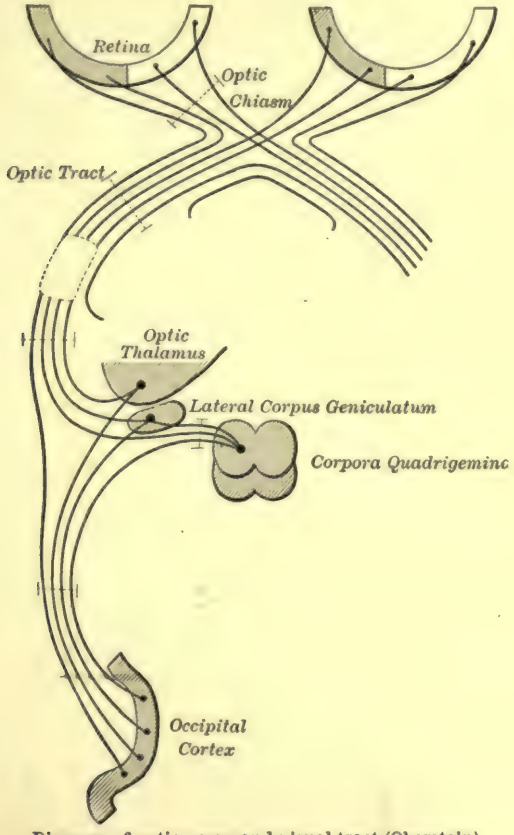


Diagram of optic nerve and visual tract (Oberstein).

the nuclei in the posterior part of the optic thalami, and thence to the occipital cortex.

ETIOLOGY.—Disease may attack separately the optic nerve, chiasm, or tract. These parts may all be damaged by tumors, which is by far the most frequent cause, or by meningitis, syphilitic lesions, or hemorrhage.

The nerve may suffer from disease at the back of the orbit or within the skull. Rheumatic neuritis following exposure, sometimes associated with neuritis of other cranial nerves, is a rare affection.

Unilateral blindness sometimes follows severe head injuries, and is probably due to laceration or hemorrhage.

The chiasm often suffers in tumors of the pituitary body and in the

enlargement of this structure in acromegaly. Lying on the floor of the third ventricle, it has been compressed in cases of hydrocephalus.

Optic neuritis is almost always a symptom of other grave disease, being especially common with cerebral tumor or abscess.

Symptoms.—1. Disease of an optic nerve, if complete, causes blindness on the same side. The pupillary reaction is absent, but contraction can be brought about by throwing a light on the healthy eye. Within a few weeks optic atrophy is detected by the ophthalmoscope.

In cases of partial damage to the nerve there are scotomata or narrowing of the field of vision.

2. Disease of the optic chiasm, when complete, causes blindness of both eyes and complete loss of the pupil reflexes to light. It is, however, more common to find temporal hemianopia or blindness of the temporal half of each field of vision. This symptom is quite characteristic of lesions of the chiasm.

Total blindness in one eye, with temporal hemianopia of the opposite side, is sometimes found, and indicates an extension of the lesion to the lateral portion of the chiasm or to the optic tract or nerve, so as to involve all the fibres passing to the eye of the same side.

An extremely rare symptom of the disease of the chiasm is nasal hemianopia. It can only result from lesions of each side of the chiasm. Gowers refers to a case of tabetic atrophy in which this sign was present.

3. *Optic Tract.*—Lesions in this situation are accompanied by hemianopia on the side opposite the lesion.

Diagnosis.—Lateral hemianopia is occasionally met with as a transient symptom of migraine, and it is also said to occur as a rare symptom of hysteria. In the vast majority of cases it is, however, due to organic affections, and its presence should be looked for in all cases of chronic cerebral disease.

The visual fibres of the optic nerve pass to the pulvinar or posterior part of the optic thalamus, and thence radiate to the occipital cortex. Lateral hemianopia may result from disease in any part of this tract. With lesions in the optic thalamus there is frequently hemianæsthesia owing to the proximity of the posterior limb of the internal capsule. Lesions in the cortex or subcortical region of the occipital lobe are often associated with sensory aphasia, particularly word-blindness.

The pupillary reaction to light affords important indication of the site of the disease. This test is known as *Wernicke's hemianopic pupillary reaction.* Contraction of the pupil to light depends on the integrity of the optic nerve of the centre in the geniculate bodies and of the fibres of the third nerve passing to the iris. If a light is thrown from one side on the blind portion of the retina and contraction takes place, we are justified in assuming that this reflex arc is intact, and that the lesion is situated higher up, either in the optic radiation or occipital cortex. An absence of contraction would point to a lesion of the reflex arc. The test is, however, somewhat difficult to carry out, and not always satisfactory.

Treatment.—Syphilitic lesions are alone amenable to treatment. In cases of doubtful origin a course of antisyphilitic treatment is advisable.

OPTIC NEURITIS.

ETIOLOGY.—Optic neuritis is in rare instances due to exposure to cold and wet, either alone or associated with neuritis of other cranial or even limb nerves. According to Zimmermann, this rheumatic form is monocular.

The great majority of cases are, however, symptomatic of coarse cerebral disease, more particularly of tumor. Tumors of any part of the brain may induce optic neuritis, and that it is independent of the size of the growth is shown by the fact that, on the one hand, very small growths may be associated with intense neuritis, and on the other, large growths may occur without it.

According to both Gowers and Bramwell, optic neuritis occurs in about 80 per cent. of cerebral tumors. Cerebellar growths are most commonly associated with it, and after these, growths situated in the motor cortex. The frequency of syphilitic gummata or meningitis as a cause of neuritis is well recognized, and must be carefully borne in mind.

After tumor, meningitis is the most common cause of neuritis, but it usually develops after other unequivocal signs, and consequently is of only occasional value in diagnosis. Abscess of the brain, again, is an occasional cause, but the disease is so rapid that it often proves fatal before sufficient time has elapsed for the occurrence of neuritis.

Multiple neuritis, myelitis, chorea, and chlorosis are in rare instances accompanied by optic neuritis.

Infectious diseases, especially typhoid, scarlatina, measles, and influenza, are in exceptional cases followed by neuritis, and Panas has described two cases in which it came on during chronic gonorrhœa. In lead-poisoning and in the excessive use of tobacco or alcohol neuritis is occasionally set up.

Neuro-retinitis is often present in the advanced stages of Bright's disease, and occurs in diabetes and leucæmia.

Suppression or other disturbance of the menstrual function has preceded severe optic neuritis.

Several cases of the disease occasionally occur in members of a family, usually, however, sparing the female members.

PATHOLOGICAL ANATOMY.—Microscopically, the trunk of the nerve shows an interstitial neuritis. At the papilla the swelling and infiltration are most marked, due probably to its looser structure favoring exudation. Elschnig[1] in a recent careful study of the subject found that signs of inflammation of the nerve trunk were present in all cases where the ophthalmoscope had revealed neuritis during life, and in some cases the nerve trunk was affected without papillitis.

In the condition known as choked disk, the most extreme form of optic neuritis, an œdema is superadded to signs of inflammation.

The pathogeny of optic neuritis has given rise to much discussion. The old view, that it is due to increased intracranial pressure, is now almost altogether abandoned. The facts that it does not occur in cerebral hemorrhage, and that a small growth is just as apt to induce it as a large one, are strongly against the pressure theory. Parinaud's view,

[1] Elschnig: *Graefe's Arch.*, 1895, vol. xli.

that a primary œdema occurs from pressure followed by inflammatory changes, is apparently refuted by the careful anatomical observations of Elschnig already referred to.

Recent observations tend to show that optic neuritis is due to certain toxic substances in the blood or in the cerebro-spinal fluid. Cases following fevers or due to poisonous agents, such as lead, point strongly to a toxic origin, and in meningitis or abscess bacterial poisons may also be supposed to play an important part in its production. Other nerves are so frequently affected by such agents that it is not surprising that the optic should also be vulnerable.

The difficulty of this theory is to account for the great frequency of neuritis in cases of tumor. The very frequent occurrence of inflammatory changes in the neighborhood of tumors, either in the brain substance or meninges, supplies, however, a source for toxic substances, and again, as Deutschmann and Leber believe, irritating substances produced in the abnormal tissue growth may perhaps act as exciters of inflammation. In meningitis the direct extension of inflammation to the optic nerve doubtless plays an important part, but that this is not essential is shown in cases of toxic neuritis without the intervention of meningitis.

The fact that trephining, with or without the removal of a tumor, often relieves optic neuritis does not militate against the view adopted that the changes are due to toxic substances, as these would drain off with the cerebro-spinal fluid after operation.

Symptoms.—In the early stages, even of intense neuritis, symptoms may be entirely absent, and reliance must be placed on the ophthalmoscopic examination.

The disk becomes slightly hyperæmic and blurred at its margin, and the central vein slightly dilated. In extreme instances (choked disk) the disk is much swollen, enlarged, and the edges are ill defined and striated. The central vein is greatly dilated and the arteries contracted. In a few cases there are hemorrhages and small white spots in the retina or about the macula, rendering the appearance precisely that of albuminuric or diabetic retinitis.

Although usually bilateral, neuritis of a single eye may occur in cases of cerebral disease; it is common to find the affection more advanced on one or other side.

Vision may be little affected or the field may be somewhat contracted. In other cases there may be only perception of light, depending on the damage to the nerve fibres themselves.

In retrobulbar neuritis inflammatory changes in the nerve trunk may be present to such an extent as to seriously interfere with vision, and yet changes in the disk may be slight or absent. The most characteristic symptom is a central scotoma at or near the fixing point for green. It may be readily tested by holding up a small green object before the eye, and is seen in its most typical form in tobacco amblyopia.

Prognosis.—High degrees of neuritis only too often pass on to atrophy and blindness. Slighter cases may recover, but very frequently leave some loss of vision.

Treatment.—In all cases the treatment of the cause is of the first importance. Unfortunately, many of these are of so grave a nature as to preclude any hope of benefit.

Toxic agents, such as tobacco and alcohol, should be forbidden. If there is evidence of syphilis, inunctions of mercury, followed by iodide of potassium, should be used; and, indeed, it is very common to adopt this treatment in all cases not due to acute cerebral disease.

OPTIC ATROPHY.

ETIOLOGY.—Optic atrophy may be either primary or result from neuritis. The former is most commonly seen in tabes, but also occurs in insular sclerosis and general paralysis. It is not uncommon to find cases apart from any obvious cause or in certain obscure nervous diseases, and it also occurs in rare instances in several members of a family. It has been attributed to cold, sexual excesses, alcohol, lead, and specific fevers.

SYMPTOMS.—In primary atrophy the disk has a grayish appearance, the edges are sharply defined, and the vessels present no marked change. In cases following neuritis the disk is white and the arteries small. It is, however, not always possible to distinguish the two forms.

Loss of sight is proportionate to the atrophy. Complete blindness or mere perception of light is present in advanced cases. In less severe cases the field of vision is contracted, often in an irregular manner, and showing sharp angles in a perimetric tracing; the acuity of vision is also diminished and color-blindness is very frequent. Occasionally a central scotoma with eccentric defects of the field of vision occurs early.

PROGNOSIS.—The outlook is bad in primary cases, usually advancing to complete blindness in one or two years.

TREATMENT.—Rest in a dark room, iodide of potassium, and mercury are used in the early stages, and later hypodermics of strychnine (gr. $\frac{1}{30}$ to $\frac{1}{20}$ daily), and galvanism. All these measures, however, usually fail to give any marked relief.

PARALYSIS OF THE OCULAR NERVES.

THE muscles of the eyeball are innervated by the third, fourth, and sixth nerves.

The third or motor oculi supplies the levator palpebræ superioris, the superior, inferior, and internal recti, the inferior oblique, the circular fibres of the iris, and the ciliary muscle. The fourth or patheticus is distributed to the superior oblique, and the sixth to the external rectus.

ETIOLOGY.—Paralysis of the ocular nerves results from lesions of the nerve trunks or of their nuclei. Defective action may also result from disease in the cortex or the fibres leading thence to the nuclei. In the present section affections of the nerves below the nuclei will be alone considered.

The nerves are sometimes injured by various forms of traumatism, such as fractures of the skull or penetrating wounds. Syphilis is the

most frequent cause of ocular paralysis; the lesion may be a gumma of or in the neighborhood of the nerves, chronic meningitis, or periostitis.

Rheumatic paralysis results from exposure to cold, and cases occurring without any obvious cause are commonly placed in this class.

Paralysis of one or more of the ocular nerves is often a symptom of cerebral disease, and is of value in localizing the affection at the base of the brain. Owing to its long course the sixth nerve is very apt to be involved.

Areas of softening, hemorrhage, or sclerosis, cerebral tumors, and tubercular meningitis may all produce paralysis of the ocular nerves. In such cases other evidence of central disease is rarely lacking.

Following diphtheria, the ciliary muscle is frequently paralyzed, and with it sometimes motor branches to the ocular muscles, and in rare cases of multiple neuritis these nerves sometimes suffer.

In tabes dorsalis paralysis, temporary or permanent, of an ocular nerve is not uncommon. The whole or part of the third nerve or any of the others may suffer.

Combined palsies are not uncommon, especially from intracranial disease. Lesions at the sphenoidal fissure, such as aneurysm of the internal carotid, has been known to affect all the ocular nerves, together with the ophthalmic nerve.

Recurring paralysis is a rare and peculiar form in which symptoms like migraine, severe unilateral headache and vomiting, are followed in a day or two by paralysis of the third, or less seldom of the sixth, nerve. These attacks recur at intervals varying from a month up to several years. The paralysis lasts for a few days or weeks; in frequently recurring cases it passes off rapidly. It is more common in women, and often begins in childhood.

Symptoms.—There are a series of symptoms which are common to paralysis of any ocular muscle. There is *limitation of movement* in the line of action of the paralyzed muscle, and later *strabismus* from overaction of its unopposed antagonist. In slight cases these deviations may be so little as to escape notice. The limitation of movement in the direction of the paralyzed muscle is termed the *primary deviation*. When an attempt is made to fix an object with the affected eye, the healthy eye deviates in the same direction as the line of action of the paralyzed muscle; the deviation of the sound eye is termed secondary deviation, and results from the unduly strong nervous impulses passing down from the centre to muscles habitually acting together. Its amount can be ascertained by covering the healthy eye and getting the patient to fix with the affected eye. If the shade is now changed to the paralyzed eye, the healthy eye moves back to its fixing point. This secondary deviation is sometimes a useful sign in detecting paralysis when the primary deviation is slight.

Another effect of paralysis is *erroneous projection*. It is manifested by inability to touch any given object when using the affected eye, the finger passing to the same side as the line of action of the paralyzed muscle. It is most marked in using the affected eye alone, and disappears when it is closed. A sense of *giddiness* is sometimes associated with erroneous projection.

Another important sign of paralysis, of much value in diagnosis, is *diplopia*, or double vision. The image seen by the healthy eye is termed the true, and that by the diseased eye the false, image. In slight cases the double image is not perceived when the eye is at rest, but it becomes well marked on attempting to put the affected muscle into action. The false image is displaced in the line of action of the paralyzed muscle, and the distance between the images increases when the eyes are directed toward the paralyzed muscle. When the false image is on the same side as the affected eye, it is termed simple or homonymous diplopia, and, if reversed, crossed or heteronymous diplopia.

Paralysis of the Motor Oculi or Third Nerve.—In this condition all the muscles of the eyeball, with the exception of the superior oblique and external rectus, are paralyzed. There are drooping of the upper lid and external strabismus, and the only movement possible of the orbit is outward. Compensatory action on the part of the frontalis may raise the lid to a slight degree, but this power varies in different individuals.

The circular muscle of the iris and that of accommodation are also paralyzed, and in consequence the pupil is moderately dilated and fails to respond to light, whilst near objects, such as small print, can only be indistinctly seen. Isolated muscles supplied by the third are occasionally paralyzed, sometimes in peripheral disease, as in neuritis after diphtheria. In central lesions, such as tumors, isolated branches sometimes suffer, especially that to the levator palpebræ.

Bilateral paralysis is rare, but may result from disease situated in the interpeduncular space. As a rule, the paralysis of one nerve is then only partial.

Paralysis of the Patheticus or Fourth Nerve.—In this affection there is slight defect in movement of the eyeball down and out. The diagnosis rests on the relative position of the true and false images.

Paralysis of the sixth nerve is evidenced by limitation of the outward movement of the eye, and later by an internal strabismus due to the unopposed contraction of the internal rectus.

TREATMENT.—In cases where there is any suspicion of syphilis mercurial inunctions, followed by large doses of iodide of potassium, should be used. In tabes a true syphilitic paralysis occasionally occurs, and in all tabetic cases an antisyphilitic treatment should be instituted.

In cases due to neuritis, as from cold, hot local applications are used at the onset, and blisters behind the ear or on the occiput are strongly recommended by some authors. Iodide of potassium, sometimes with small doses of mercury, is also beneficial. Strychnine is useful after the acute stage has been passed. General tonics are indicated when the general health is defective. In many cases treatment is of little use, owing to the cause being an irremediable organic cerebral lesion.

An opaque glass is useful if diplopia is troublesome, or the same end may be obtained by the use of prisms.

Spasm of the Ocular Muscles.—Tonic spasm of the muscles is common in irritating lesion of the nerves, especially tubercular meningitis, and is occasionally a symptom of hysteria. Clonic spasm or nystagmus is seen in insular sclerosis and sometimes in brain disease. It also occurs in albinos and coal-miners.

DISEASES OF THE FIFTH NERVE, OR TRIFACIAL.

The fifth is the great sensory nerve of the face and fore part of the scalp. Of its three main trunks the ophthalmic supplies the orbit and lachrymal glands, the skin of the forehead and scalp as far as the vertex, the tip of the nose, and the anterior part of the nasal mucous membrane. The superior maxillary supplies the skin over the malar bone, the root of the nose, the infraorbital region, and the upper lip, also the greater part of the nasal mucous membrane, the palate, the upper part of the pharynx, and the upper teeth. The inferior maxillary division is distributed to the skin of the temple, lower lip, and chin, and parts of the ear and external auditory meatus, and also the lower teeth and the mucous membrane of the mouth and tongue.

The motor division of the nerve supplies the muscles of mastication, the masseter, two pterygoids, and the temporal.

Paralysis.—The whole nerve may be affected or any of its main trunks. Owing to its deep position, it seldom suffers from primary neuritis or from cold like the facial, and it is only damaged by deep penetrating injuries, especially bullet wounds.

The whole nerve may be involved by coarse lesions of the pons, such as softening or tumor, or by disease in the middle or posterior fossæ at the base of the skull, such as chronic meningitis or bone disease, and the possibility of such lesions being due to syphilis must always be borne in mind.

Of the separate divisions of the nerve, the first is sometimes involved by aneurysms of the carotid or by orbital growths, and the second and third may be damaged in the spheno-maxillary region by parotid tumors or bone disease.

The motor portion of the nerve is in rare instances affected in tabes, poliomyelitis, and bulbar paralysis: it is probable that the lesion is nuclear in such cases.

Sensory Symptoms.—The most prominent and distressing symptom is severe pain in the area of distribution of the nerve, more or less constant in character, and sooner or later accompanied by diminished sensation or anæsthesia, or in the irritative stage by hyperæsthesia.

Anæsthesia is quite characteristic, and may come on without pain, but more commonly both pain and loss of sensation exist together. The various mucous membranes share in the loss of sensation. The conjunctiva and cornea are insensitive, and may be touched without producing the usual reflex. The nasal mucous membrane no longer responds to painful stimuli, such as ammonia vapor, and later the sense of smell is defective or lost, owing to diminished secretion and dryness in the membrane. The mouth, hard and soft palate, and the tongue are also insensitive as far as the middle line.

In some cases, but not invariably, taste has been lost on the tongue and palate of the affected side. It is not known whether the variations which occur are due to individual differences of distribution or to instances of partial disease of the nerve in the pons.

Vasomotor changes are rare, but trophic affections are not uncommon. The most important is inflammation of the cornea, which becomes opaque and tends to ulcerate. Meissner believes that it is specially in

irritative disease of the Gasserian ganglion that such changes occur. The secretions of the mucous membrane and of the lachrymal and salivary glands are diminished.

Motor Symptoms.—Paralysis of the muscles of the jaw is rendered obvious by the absence of contraction of the masseter in forcibly closing the mouth. If the lower teeth are protruded in front of the upper, the jaw deviates toward the paralyzed side, owing to the action of the healthy external pterygoid. Food is masticated on the healthy side, and consequently the tongue is often furred on the affected side.

Later atrophy and flattening of the temporal and masseter muscles may be observed in the temporal and zygomatic fossæ.

Paralysis of the four small muscles supplied by the nerve (mylo-hyoid, digastric, tensor tympani, and tensor palati) does not give rise to any obvious signs.

Diagnosis.—This is easy when there is distinct anæsthesia in the course of the nerve or of one of its trunks, or if the motor part is affected.

The presence of anæsthesia serves to distinguish the pain from that of neuralgia, and again hyperæsthesia, when present, is more marked and persistent.

In cases where the only symptom is pain due to irritation of the nerve the distinction from neuralgia may be uncertain or impossible until some less equivocal sign arises.

When possible the site and nature of the lesion should be determined. If the pons is affected, there may be paralysis of the limbs on the opposite side or paralysis of the conjugate movements of the eyes toward the diseased side.

The sixth or seventh nerve, if paralyzed with the trunk of the fifth, points to disease in the middle fossa of the skull, or if the first division only is diseased with the ocular nerves, the lesion can be located in the sphenoidal fissure or the back of the orbit.

It is more difficult, and often impossible, to determine the pathological nature of the disease. If we can exclude lesions of the pons and traumatism, the most probable cause of the lesion is either syphilis or tumor, the latter being usually of a malignant character.

Treatment.—A trial may be made of iodide of potassium in doses of 10 to 30 grains thrice daily, or even in larger doses. If the lesion is syphilitic, relief will be obtained.

If pain is severe, morphine is the most certain remedy, although the various coal-tar preparations may be tried.

Galvanism or faradism is useful in combating various paræsthesiæ which are sometimes present, and these agents are also used for the paralyzed muscles.

Division of the nerve or of one of its branches may prove serviceable in relieving pain in obstinate cases, provided the section can be made behind the lesion. In a case of pain and anæsthesia of the ophthalmic nerve G. E. Armstrong successfully divided this branch within the skull. with relief to the symptoms.

Spasm.—Spasm in the muscles supplied by the facial is common, but the great majority of cases do not depend on disease of the nerve. Cases of spasm following paralysis have been already referred to, and

a few cases are reported in which spasm, clonic or tonic in character, has resulted from the pressure of tumors or aneurysms on the nerve trunk at the base of the skull.

DISEASES OF THE SEVENTH NERVE.

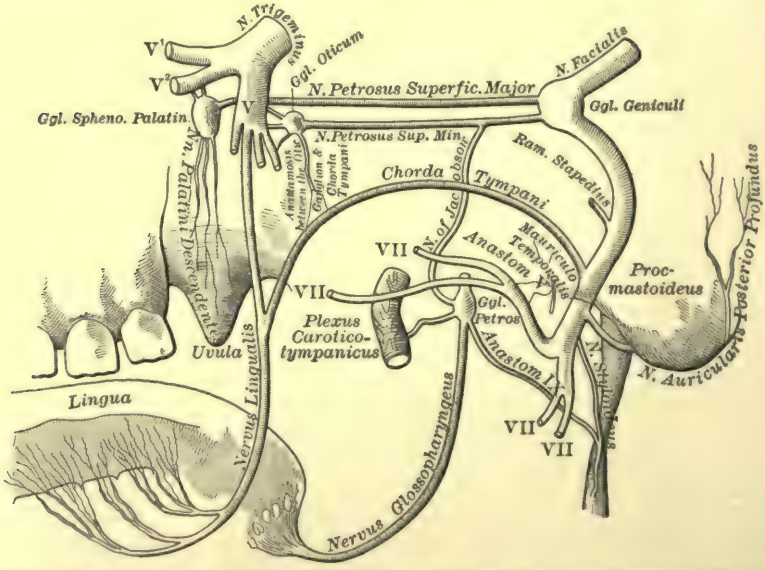
Facial Paralysis; Bell's Paralysis.

Etiology.—Owing to its exposed position and its course through the bony aqueduct of Fallopius, the seventh is one of the nerves most frequently paralyzed.

In a large majority of cases the affection is due to neuritis, often termed "rheumatic," and traceable to direct exposure to cold or draughts, as in sitting by an open window. In many cases, however, there is no history of direct exposure to cold.

In purulent middle-ear disease, especially when necrosis or caries is present, the nerve is often involved by the inflammatory process.

Fig. 2.



Course of the facial nerve, and its connection with the fifth and glosso-pharyngeal (Leube).

Traumatism, again, is responsible for certain cases. In fractures of the skull passing through the petrous bone, the nerve is often crushed or torn. It is occasionally divided by stabs or during surgical operations, especially in the parotid region, and it has been paralyzed by the pernicious practice of boxing the ears. In obstetric cases it is occasionally damaged by pressure of the forceps, and even double paralysis has occurred in infants from this cause.

The relative frequency of these causes may be judged by the statistics of Philip,[1] who found that 72.3 per cent. of his cases were of a rheumatic nature, 6.2 per cent. due to ear disease, and 5.4 per cent. to traumatism.

Among other causes may be mentioned tumors in the parotid region and tumors or meningeal inflammation at the base of the brain.

In multiple neuritis one or both facial nerves have been involved, but only in exceptional instances. Thus in alcoholic and diphtheritic paralysis the facial is occasionally attacked, and a few instances are recorded in multiple neuritis after influenza. Westphal has reported a case in which both nerves were affected from this cause. Syphilis plays an occasional part; the mechanism through which it operates may be a gumma or interstitial neuritis of the nerve trunk or meningitis. In spinal disease, especially tabes, facial paralysis occurs in rare cases. Gowers records two cases of instantaneous onset which he regards as due to hemorrhage into the Fallopian canal.

Apart from obstetrical cases, the disease is very rare in childhood, and most commonly occurs between twenty and forty.

That a certain personal predisposition to the disease exists is shown by the fact that only a small portion of individuals who are exposed to draughts suffer. Bernhardt found in 33 per cent. of cases an hereditary tendency or a history of a previous attack.

It has usually been assumed that facial paralysis is due to an inflammatory exudation compressing the nerve in its bony canal. Anatomical proof of this is, however, wanting, but that such a result is possible is proved by a case of May's, in which a patient with leucocythæmia was affected by facial paralysis due to a lymphoid infiltration of the nerve in the Fallopian canal.

In a recent case quoted by Bernhardt the patient died from hydrochloric-acid poisoning, and gave an opportunity of examining a facial paralysis of eight weeks' standing. There was merely a degeneration of the nerve, but it is obvious that an exudation might have existed and been absorbed in this time.

A recurrence of the disease is very unusal, but isolated instances of three or even four attacks are recorded. This immunity from second attacks is indeed such a marked feature that it has been assumed that the disease is due to an infectious process conferring protection from recurrence.

Symptoms.—The appearance of an individual suffering from facial paralysis is extremely characteristic. The affected side is immobile and expressionless. The furrows on the forehead and face are smoother than on the healthy side. The labio-nasal fold is shallower; the mouth is drawn toward the healthy side, and its angle is lower than in health. These signs are much less marked in the young, in whom the elasticity of the skin diminishes the deformity when the muscles are at rest.

Owing to paralysis of the orbicularis palpebrarum, the eye is open and looks larger than its fellow, from retraction of the lid.

On attempting to laugh or smile the affected side is fixed and immobile; the mouth is markedly drawn to the healthy side, and there is

[1] Philip: quoted by Bernhardt in Nothnagel's *Specielle Pathologie.*

total inability to wrinkle the forehead or to close the eye, which remains open during sleep. In attempting to corrugate the forehead the eyeball turns up, owing to associated action of the superior rectus and the occipito-frontalis. The lips fail to meet on the affected side, so that there is inability to whistle, to blow out a candle, or to expectorate in a straight line. Saliva is also apt to dribble from the side of the mouth. In drinking there is a tendency for the fluid to escape from the paralyzed side. The tears are also apt to run over on the cheek, leading eventually to erythema or eczema of the skin, and, owing to paralysis of the buccinator, food collects between the cheek and the gum. Speech is at times slightly affected in pronunciation of labial sounds. The tongue is protruded in the median line, but there may be an apparent deviation owing to the deformity of the mouth. The platysma and the external muscles of the ear are paralyzed, and their lack of movement can be occasionally demonstrated in cases where they are under control of the will.

When the nerve is damaged between the geniculate ganglion and the origin of the chorda tympani taste is lost on the anterior third of the tongue, and this feature is usually easily demonstrated. Judging from the physiological function of the chorda, diminished secretion of saliva might be anticipated, but such has not been conclusively proved. It is often stated that hearing is more acute, owing to paralysis of the stapedius leaving the tensor tympani free to act and tighten the membrana tympani. In a few cases an abnormal sensitiveness to sound, especially the deeper tones, is present. The sense of hearing is, however, often impaired, owing to disease of the middle ear, which is the cause of the paralysis, and again disease at the base of the skull may damage the auditory nerve. The sense of smell may be diminished, owing to weakness of the ala nasi.

Paralysis of the palate and deviation of the uvula to the healthy side is said to occur in disease above the geniculate ganglion. It is then assumed that fibres pass from the geniculate ganglion by the great superficial petrosal and Vidian to the spheno-palatine ganglion, and thence by the descending palatine nerves to the palate. Both Gowers and Hughlings-Jackson state that they have never seen paralysis of the palate in isolated palsy of the seventh.

It is highly probable that the palate is innervated from the spinal accessory, and it is certainly frequently paralyzed by tumors or other lesions at the side of the medulla.

Later Symptoms.—As in all forms of peripheral paralysis, muscular atrophy follows the nerve degeneration, but owing to the subcutaneous fat this change is not perceptible to the eye.

Secondary contracture again occurs in severe cases, and comes on several months after the onset of paralysis. The labio-nasal fold is in this way often deeper than on the paralyzed side, so that at the first glance the healthy is mistaken for the paralyzed side. This impression is corrected by putting the muscles into action, when the affected side, although showing a deeper labio-nasal fold, moves less than its fellow.

Spasmodic twitchings of the paralyzed muscles is another after-effect of facial palsy. Peculiar associated movements again are seen—*e. g.* if the patient winks or closes the eye, a simultaneous deviation of the

angle of the mouth occurs. Increase in the reflex activity of the face, showing itself by contractions or striking or picking the skin of the face, also occurs. All these features are observed chiefly in severe cases in which recovery is imperfect.

Electrical Reaction.—The electrical reactions of the nerve and muscles in facial paralysis correspond with the reactions seen in nerve degeneration elsewhere.

In slight cases stimulation of the nerve with either current shows diminished irritability, coming on usually toward the end of the first or during the second week. There is frequently, however, an increased irritability, lasting for a few days to a couple of weeks, and sometimes not followed by any subsequent fall. According to Erb, very slight cases show no electrical changes, but careful examination usually reveals some alteration either in the direction of increase or decrease of nerve irritability.

Corresponding changes are found in the muscles. There is increased irritability to faradism depending on changes in the terminal portion of the nerve, and later diminished irritability proportionate to that of the nerve. With galvanism, however, there may be increased irritability and qualitative changes.

In severe cases the nerve irritability to both currents is completely lost at the end of a fortnight, whilst the muscles respond only to galvanism. They then show the slow contracture, often with qualitative changes, so characteristic of marked nerve degeneration.

Diagnosis.—Peripheral paralysis of the facial must be distinguished from paralysis due to disease of the motor tract in the brain or the nucleus. The former or cerebral paralysis is usually associated with weakness of the arm and leg, and the upper fibres of the nerve to the forehead and eyelid commonly escape, so that the patient can wrinkle his forehead and close the eye. In the early stages, however, of cerebral disease the upper muscles of the face are occasionally involved, but they recover more rapidly, being innervated from both hemispheres.

In diseases above the nucleus the electrical reactions are unaltered, or at the most show only a slight increase of nerve irritability. Another feature of the cerebral form is that emotional movements are less impaired then voluntary ones.

In nuclear paralysis the orbicularis oris escapes, the fibres innervating this muscle arising from cells in close proximity to those for the tongue.

Facial palsy with loss of hearing in the absence of ear disease and paralysis of the sixth nerve usually indicates disease at the base of the skull, although the latter nerve is sometimes affected by a simultaneous rheumatic paralysis. Loss of taste proves the site of disease to be between the geniculate ganglion and the origin of the chorda just above the stylo-mastoid foramen. The chorda escapes in lesions below the exit of the nerve.

A purulent discharge from the ear usually indicates an aural origin of the disease. In a case recently under my care, however, a patient in a stuporose condition presented a complete facial paralysis, with a stinking discharge from the ear. An exploratory incision over the mastoid revealed a fracture of the skull. The nerve had doubtless been

injured by the fracture, and the purulent discharge resulted secondarily from sepsis.

PROGNOSIS.—In rheumatic cases the electrical reactions are the surest indications of the extent of damage to the nerve. Where the irritability of the nerve is unaltered or increased after a week, recovery may ensue in two or three weeks. If, however, distinct diminution of nerve irritability is present in eight or ten days, recovery will be postponed for six or eight weeks.

With a complete reaction of degeneration recovery cannot be expected for several months. Any return of nerve irritability indicates an early return of some power in the muscles. It is fortunately only in rare cases that there is complete and permanent paralysis.

TREATMENT.—The treatment depends on the cause. If there is any evidence of syphilis, iodide of potassium should be administered in doses of 10 grains and pushed to 20 or 30 or more. The presence of suppurative middle-ear disease demands careful cleansing with the syringe and free exit for any discharge. In rheumatic cases, which form by far the most numerous class, warm fomentations should be applied below and behind the ear for a few days, and a blister one inch square should also be applied in all but the very mildest cases. If there is much neuralgic pain, a few doses of antipyrine (gr. 10) or phenacetine (gr. 5) may be administered as analgesics. Small doses of iodide of potassium (gr. 2 to 5) should also be administered, and a little later a pill of quinine, gr. 1–2, with strychnine, gr. $\frac{1}{30}$–$\frac{1}{40}$, may be added.

There seems to be little doubt that the systematic application of weak galvanic currents hastens the return of the voluntary power and also tends to preserve the nutrition of the muscles. The positive electrode is placed over the exit of the nerve behind the ear, and the negative is moved over the face. The current used should be from 2 to 4 milliampères, applied for ten or fifteen minutes two or three times weekly. In slight cases faradism may be used, but it is useless in severe cases where the muscles do not respond.

When contracture sets in electricity is of little value. Massage and warm douches of the affected side have been recommended, and faradism of the healthy side to stretch the contracted muscles. To none of these plans, however, can much value be attributed.

DISEASES OF THE AUDITORY NERVE.

DEAFNESS due to disease of the nerve trunk or of its end organ in the labyrinth are grouped together under the term "Nerve-deafness."

Disease of the labyrinth is by far the commonest cause. The lesions may consist in acute or chronic inflammation, often spreading from the tympanum, syphilis (congenital or acquired), and in elderly people degenerative changes. Sudden onset with vertigo suggests hemorrhage, and extravasation in the middle ear has been found in such cases.

Lesions of the nerve trunk may result from tumors, meningitis (especially the epidemic form), and syphilis. A neuritis analogous to that

of the optic nerve has been assumed in certain cases of cerebral tumor. In tabes and in elderly people primary atrophy may occur.

SYMPTOMS.—Nerve deafness is recognized by the tuning fork. In cases of deafness due to disease of the external and middle ear bone conduction is preserved, whilst aërial conduction is diminished or lost. In cases of nerve deafness, however, the tuning fork is not heard, either through the air or when placed against the bone.

In cases of slight nerve deafness there is rather more difficulty in recognizing the condition. In health the vibrations of the tuning fork are heard longer through the air than by the bone, and if the sound cannot be distinguished by the air after it has ceased through the bone, there is diminished conduction through the middle or external ear.

In cases of slight deafness through the air, and where the tuning fork is heard longer through the air than by the bone, there is an indication of nerve deafness.

Nerve deafness associated with facial paralysis indicates disease at the base of the skull or at the internal auditory meatus.

TREATMENT.—Apart from syphilitic lesions little benefit can be obtained from treatment.

DISEASE OF THE GLOSSO-PHARYNGEAL NERVE.

OWING to its numerous connections and the absence of any case of isolated lesion, less is known of the functions of this than of any other cranial nerve.

It is probable that paralysis of the nerve produces difficulty in deglutition, owing to paralysis of the middle constrictor of the pharynx, and there is loss of sensation of the roof and wall of the pharynx and of the Eustachian tubes.[1] Physiological experiment seems to show that the nerve checks continuous contractions of the pharynx, as there is tonic spasm of the pharynx after it is divided.

The question as to whether it is a special nerve of taste is still sub judice. In a case of Pope's[2] this nerve with the pneumogastric was damaged by a thrombus in the vertebral artery. In addition to loss of power of deglutition there was loss of taste at the back of the tongue and diminution anteriorly.

DISEASES OF THE PNEUMOGASTRIC OR VAGUS NERVE.

THE pneumogastric or vagus nerve has a very long course, and is distributed to the pharynx, œsophagus, larynx (superior and inferior branches), lungs, heart, and stomach, and also sends twigs to the spleen and intestines.

[1] Birkett: *Montreal Med. Journ.*, vol. xix. [2] Pope: *Brit Med.. Journ.*, 1889, ii.

Paralysis.—Causes.—The nerve may be injured within the cranium by growths in its neighborhood or from syphilitic meningitis or aneurysms.

The nucleus suffers in cases of acute or chronic bulbar palsy, but other nerve nuclei are always affected as well.

Outside the skull the nerve has been compressed by tumors, divided in surgical operations, and included in ligatures passed round the carotid artery. Wounds of the nerve are usually accompanied by laceration of the great vessels of the neck, which prove rapidly fatal from hemorrhage.

Symptoms.—Division of the nerve in animals is followed by rapid cardiac action, whilst respiration is slower and deeper. Stimulation of the lower end of the cut nerve produces slowing or arrest of the cardiac action, whilst the respirations are increased in rate by stimulation of the upper end. From these facts it is inferred that the efferent fibres distributed to the heart exert an inhibitory action, whilst the afferent fibres stimulate discharges from the respiratory centre.

That analogous symptoms occur in disease is evidenced by the comparatively few reported cases of division during operation. An increase in the pulse and slowing of respiration has occurred, the condition usually terminating fatally. Similar symptoms have been repeatedly noticed after diphtheria, and are almost invariably fatal.

Irritation of the nerve may reverse these symptoms, the most noticeable feature being slowing of the cardiac action. Several well-known cases are recorded in which the heart could be slowed at pleasure, one of the best marked being that of Czermak, who did so by compressing the nerve against a small growth in the neck. In a case under my own observation evidence of an intrathoracic tumor was accompanied by a pulse of sixty and respiration of thirty, probably due to irritation of the nerve.

The most important evidence of paralysis of the pneumogastric is found in the larynx, which is treated in another section.[1]

The pharynx and œsophagus are paralyzed most commonly from intracranial affections of the nerve, and especially in lesions of the nuclei. Slight difficulty in swallowing results, which becomes marked, from bilateral disease. Owing to associated paralysis of the larynx in bulbar lesions, particles of food are apt to pass into the respiratory tract and set up an inhalation pneumonia. The vagus supplies motor branches to the stomach and intestines, and increase and loss of appetite and thirst have also been attributed to derangement of the functions of the nerve.

Vomiting may occur from direct irritation of the nerve, but is much more common from various reflex or central channels.

Irritation of the sensory branches distributed to the heart has produced the symptoms of angina pectoris. Fatty degeneration of the heart following disease or injury of the nerve is probably of trophic origin.

[1] Vide *Diseases of the Larynx*, vol. ii. pp. 75, 76.

DISEASES OF THE SPINAL ACCESSORY NERVE.

This nerve consists of an accessory portion (arising from the medulla), which joins the vagus and is distributed to the laryngeal muscles, and probably to the soft palate through the pharyngeal plexus. The external or spinal portion supplies the sterno-mastoid and trapezius muscles.

The nerve suffers from the same causes which affect the vagus within the cranium. The external portion may be divided outside the skull from wounds or compressed by tumors or bone disease. It is in rare instances the seat of a primary neuritis.

Symptoms.—Horsley[1] has shown that stimulation of the root of the nerve in the monkey produces movements of the soft palate, and it is probable that there is a similar distribution in man. It is unquestionable that disease of the side of the medulla is frequently associated with paralysis of the palate, vocal cord, and tongue on the same side, there being little doubt that the spinal accessory is the nerve for the first two.

When the external portion of the nerve is diseased there is paralysis of the sterno-mastoid and upper portion of the trapezius, followed by wasting. Owing to paralysis of the sterno-mastoid, there is defective action in rotating the head to the opposite side. Paralysis and wasting of the trapezius are most obvious on deep inspiration, the prominent boundary of the posterior triangle of the neck being lost and the scapula hanging lower than its fellow.

Treatment.—The treatment of paralysis of the pneumogastric and spinal accessory nerves should be directed as far as possible toward the cause. Syphilitic lesions are alone markedly influenced by drugs.

DISEASES OF THE HYPOGLOSSAL NERVE.

The hypoglossal nerve is purely motor and supplies the muscles of the tongue.

Paralysis of the nerve is very common in cerebral lesions involving the fibres from the cortex to the nucleus. Hemiplegia is a familiar example. Nuclear lesions are usually bilateral, but unilateral disease has been observed in tabes and syringomyelia.

In contrast to paralyses from cerebral disease, peripheral paralysis is uncommon. The nerve has been affected within the skull from tumors or meningitis (especially the syphilitic form) about the medulla, and by caries of the posterior fossa. Weir Mitchell has recorded a case in which the nerve was divided by a pistol wound within the skull. Outside the skull the nerve has been divided by penetrating wounds and damaged by deep-seated tumors or from pressure from caries of the atlas. Very rarely the only explanation has been a rheumatic neuritis.

Symptoms.—In unilateral paralysis of the tongue the organ usually lies in its normal position when in the mouth, but the paralyzed side is higher than the other. In a case of Birkett's,[2] however, the tip was directed to the healthy side.

[1] Horsley: *Brit. Med. Journ.*, 1888, ii.

[2] Birkett: *loc. cit.*

When protruded it is directed toward the paralyzed side, owing to the action of the genio-hyoglossus muscle. At the same time the median raphé becomes concave toward the paralyzed side from contraction of the unaffected intrinsic muscle.

In nuclear and infranuclear lesions there is distinct atrophy of the diseased side, and the electrial reactions correspond with those of nerve degeneration. Owing to wasting of the muscles the mucosa is thrown into prominent folds. Taste is unaffected and speech but slightly affected in unilateral disease.

Diagnosis.—Paralysis of the hypoglossal due to cerebral disease is associated with paralysis of the limbs and face on the same side. In the medulla there is a cross paralysis of the hypoglossal and the limbs.

Paralysis of the hypoglossal with the soft palate and vocal cord of the same side (spinal accessory) usually indicates disease at the side of the medulla.

Treatment.—Mercurials and iodide of potassium may be tried, especially if there is a history of syphilis. Electrical treatment, especially galvanism, is sometimes serviceable.

DISEASES OF THE PHRENIC NERVE.

Paralysis of the diaphragm is most frequently seen from involvement of both phrenic nerves in multiple neuritis. It occurs especially in diphtheritic paralysis, but is also seen in alcoholic and lead neuritis and in tabes. Disease of the vertebræ or of the spinal meninges at the level of the third and fourth cervical vertebræ has involved both nerves.

The nerve has been divided by wounds in the neck as it lies on the scalenus anticus muscle. It has also been damaged by the pressure of growths or gummata, and in the thorax by aneurysm. It is rarely the seat of a primary neuritis.

Symptoms.—Bilateral disease is much more common than unilateral, and is detected by the sinking in of the abdominal wall during inspiration and of the opposite condition in expiration. The liver border has been noticed higher during inspiration, and it is possible that the absence of Litten's diaphragmatic phenomenon may be utilized in diagnosis.

Dyspnœa becomes very marked during exertion, but beyond a slight increase of the respirations is not noticeable at rest. When one nerve only is attacked the morbid symptoms are present only on one side, and are readily overlooked.

Slight bronchial attacks are apt to prove serious, owing to the difficulty of expectoration.

A fatal issue is common in alcoholic or diphtheritic neuritis when the phrenic nerves are involved.

Treatment.—Absolute rest is essential, and even speaking is liable to overtax the respiratory movements. Otherwise the treatment must be directed toward the course and in accordance with the general rules of peripheral paralysis.

DISEASES OF THE BRACHIAL PLEXUS AND NERVES.

Neuritis.—ETIOLOGY.—*Neuritis* of the brachial plexus is a somewhat rare disease. It occurs chiefly in rheumatic and gouty individuals, and five sixths of the cases occur over the age of fifty. Unlike other forms of neuritis, it is as common in women as in men.

The morbid changes consist in an inflammation of the nerve sheaths, and from clinical grounds there is reason to believe that some cases commence about the nerve roots—*radicular neuritis.*

SYMPTOMS.—The chief symptom is severe pain, usually somewhat gradual in its onset, but sometimes sudden and severe from the beginning. At first it is commonly referred along some of the nerves, and then settles down in the plexus above the clavicle or in the axilla. Although always greatly increased by movement, it is also spontaneous in the severer cases.

Hyperæsthesia of the skin is common with the pain, and the nerves above the clavicle are extremely tender and sensitive to the touch. Anæsthesia is seen in some cases, and is present at the extremity of the limb.

Œdema and trophic changes in the skin and nails are also common, and articular adhesions with deformity occur in the shoulder, wrist, and hands.

The motor fibres are seldom sufficiently damaged to cause considerable loss of motor power, but slight atrophy of the muscles, especially of the arm and hand, is common, and occasionally a group of muscles may show considerable atrophy, with the reaction of degeneration. The loss of motor power is often more apparent than real, owing to the pain induced by any attempt at movement.

DIAGNOSIS.—The disease is very often mistaken for neuralgia, rather from an ignorance of the symptoms than from their equivocal character.

Pain is the distinguishing feature of both conditions. In neuritis the nerve trunks are persistently tender, the pain is increased by movement, and slight wasting with anæsthesia may be added. The tender points of neuralgia are shifting and transitory.

Pain of a paroxysmal character, due to neuritis of the left arm, may be mistaken for the reflected pain of aneurysm or angina pectoris. In these conditions, however, there is an absence of tenderness of the nerve trunks in the neck. In the reflected pain of spinal disease, such as tumors or bone disease, there is often positive evidence of bone or cord lesions, and in addition there is not the tenderness of the nerve trunks.

PROGNOSIS.—In most cases the symptoms persist for months or even a year. The pain is often slow to disappear, and adhesions forming in the joints may permanently impair the use of the limbs. The muscles at times do not completely recover, and wasting and weakness may be permanent.

TREATMENT.—The treatment is similar to that of neuritis in general. The limbs may be bandaged across the chest to secure rest, and all measures, such as massage, which increase pain are to be carefully avoided in the acute stage. Later on the stiffness may be combated by hot douching or rubbing.

Morphine hypodermically is usually required to relieve pain, and cocaine is also used for the same purpose.

Paralysis of the Nerves of the Brachial Plexus.—Paralysis of all or nearly all the nerves of the brachial plexus results from many morbid lesions, and either the nerve roots or the cords of the plexus may be affected.

The nerves are most commonly paralyzed by some form of traumatism. They are occasionally damaged by carrying weights on the shoulder or by dislocation of the shoulder; in fractures of the clavicle or humerus they are sometimes compressed, torn, or involved by callus. Abscesses and tumors of the neck, wounds, and in obstetrical cases the pressure of forceps on the child's neck, may all produce paralysis. Primary or migrating neuritis again has been already referred to in producing the disease.

Symptoms.—In complete paralysis the arm hangs by the side, the humerus being rotated in, the forearm supinated, and the palm of the hand directed back. The muscles are powerless, flaccid, and wasted; the reaction of degeneration is present, and the limb is often cold and blue, and trophic changes, such as glossy skin and changes in the nails, may be present.

There are commonly numbness and loss of sensation in the limb, but the sensory nerves suffer much less than the motor. If complete, sensation of the whole limb is involved except the inner part of the arm, supplied by the intercosto-humeral nerve, and the skin over the deltoid supplied by the third and fourth cervical segments.

Duchenne-Erb's Paralysis, or Paralysis of the Upper-arm Type.—Erb particularly described a form of paralysis affecting the deltoid, biceps, brachialis anticus, supinator longus, and sometimes the spinati and supinator brevis.

The arm hangs by the side, the forearm is pronated, and wasting of the affected muscles occurs. There is in a few cases anæsthesia over the outer part of the arm and forearm, over the distribution of the sensory branches of the circumflex and musculo-cutaneous nerves. It is probable that loss of sensation occurs in most cases, but is temporary and escapes recognition. This paralysis is referred to lesions of the fifth and sixth cervical nerves, and Erb found a point between the scalene muscles at which electrical stimulation put these muscles into action.

It is due to injuries, such as carrying weights on the shoulder, to neuritis, and in infants is one of the forms of obstetrical palsy.

Paralysis of the Lower-arm Type.—This form involves paralysis of the seventh and eighth cervical and first dorsal nerves. It usually results from injuries or wounds of the shoulder, and two cases are recorded by Pfeiffer from pulmonary and vertebral tumors.

The muscles involved are those of the thenar and hypothenar eminences and the interossei supplied by the first dorsal; also the muscles of the forearm, with the exception of the supinators and the triceps. An area of anæsthesia along the inner side of the hand and forearm, supplied by the ulnar and internal cutaneous nerves, is usually present, being much more frequently met with than the corresponding anæsthesia of the upper-arm type.

In cases where the nerve roots are damaged there is oculo-pupillary

disturbance, evidenced by contraction of the pupil, narrowing of the pupil, and recession of the eyeball, and exceptionally flattening of the face. The pupillary fibres, as shown by M. Klumpke,[1] pass in the ramus communicans of the first dorsal nerve to the sympathetic. Vasomotor disturbance is not present, as these fibres pass to the sympathetic from the second to the sixth dorsal nerves.

These types are often mixed in actual cases: in total paralysis partial recovery may occur and paralysis of one or other sets of muscles remain.

DIAGNOSIS.—Complete or partial paralysis of the arm is often the result of poliomyelitis in children. This affection is marked by an acute febrile onset, and sensation is not affected.

PARALYSIS OF THE LONG THORACIC NERVE TO SERRATUS MAGNUS.

THIS nerve is formed by the union of branches from the fifth and sixth cervical nerves. It passes through the scalenus medius and behind the brachial plexus to the side of the chest, ending in the serratus.

The nerve is most frequently damaged by direct injury in the neck, such as the pressure of packages carried on the shoulder, or indirectly by muscular efforts when it is compressed by the scalenus medius, through which it passes. It has been divided by punctured wounds in the posterior axilla. Paralysis is most common on the right side and in males. The nerve is sometimes the seat of a primary neuritis.

SYMPTOMS.—The onset is usually marked by pain about the shoulders. The paralysis of the serratus is most obvious when the arm is held straight forward. The vertebral border of the scapula then becomes prominent; the *scapula alata* and the lower angle are tilted up and in. The arm can still be raised above the head by the trapezius, but this movement is weakened, and the arm tires easily in such a movement as brushing the hair. In a schoolmaster recently under observation much difficulty was felt in raising the arm to write on the blackboard. At rest the scapula is rather higher and nearer the spine than its fellow, owing to the unopposed action of the trapezius, rhomboids, and levator anguli.

Double paralysis of the serratus occurs in muscular dystrophies and in scapular forms of spinal muscular atrophy, rarely from bilateral peripheral traumatism.

Recovery from peripheral paralysis is often tedious, and paralysis is sometimes permanent.

TREATMENT.—In the treatment the limb should be supported by a sling, and movement of the shoulder prevented in the earlier stages of the malady. Counter-irritants, if required, should be placed over the scalene just outside the lower end of the sterno-mastoid muscle. Galvanism is serviceable as in other forms of nerve injury.

[1] Klumpke: *Rev. de Méd.*, 1885, July-Sept.

Paralysis of the Suprascapular Nerve to the Supra- and Infraspinatus Muscles.

The suprascapular nerve is seldom damaged alone, and, like the long thoracic, is occasionally paralyzed by injuries about the neck and shoulder.

In severe cases there is marked atrophy of the infraspinatus, and paralysis of this muscle interferes with outward rotation of the humerus, so that difficulty is felt in such movements as writing or sewing.

Weakness of the supraspinatus allows a little drooping of the shoulder and throws more work on the deltoid, so that this muscle readily tires.

Neuritic pains over the scapula, followed by some loss of sensation, frequently occur.

Paralysis of the Circumflex Nerve.

Derived from the posterior cord, this nerve supplies the deltoid, the teres minor, the skin over the shoulder, and the joint.

Paralysis most frequently results from injury, especially dislocations and fractures of the head of the humerus.

Owing to its close proximity to the shoulder-joint, it is occasionally involved by articular inflammations or may be attacked by a primary neuritis.

The chief symptom is loss of power in elevating the arm to a right angle, the supraspinatus being too feeble to compensate for the loss of the deltoid. Flattening of the shoulder is very conspicuous, owing to atrophy of the deltoid. The sensory fibres often escape, but if affected produce pain and later anæsthesia over the shoulder.

The shoulder-joint inclines to the formation of adhesions, owing, probably, to the withdrawal of the influence of its trophic nerves.

Double paralysis is rare. A case has recently been described by Raymond resulting from sleeping with the arms extended above the head.

DISEASE OF THE MUSCULO-SPIRAL NERVE.

The musculo-spiral nerve is formed from the three primary trunks of the brachial plexus. Passing between the outer and inner heads of the triceps, it winds to the outer side of the humerus in contact with the bone, where it divides into the radial and posterior interosseous; the triceps, supinator longus, and extensor carpi radialis longior are supplied directly by the nerve trunk, whilst the other extensors and the supinator brevis derive their nerve supply from the posterior interosseous. The radial supplies the skin over the outer part of the back of the hand.

Paralysis of this nerve is more frequent than that of any other nerve of the arm. Lying on the bone, it is subject to pressure, and frequently suffers from the use of crutches or from various injuries. Gowers refers to three cases in which the nerve was paralyzed after violent contraction of the triceps. The nerve is frequently affected by being lain on during sleep or from sleeping with the arm over the back

of a chair. These sleep palsies are especially common in drunkards, owing to the deep sleep and anæsthesia produced by alcohol.

A few cases are recorded after the use of the Esmarch bandage, and recently paralysis has been observed after injections of ether in the back of the forearm.

Paralysis of the extensors of the wrist is extremely common in the neuritis of chronic lead and alcoholic poisoning.

Symptoms.—The most noticeable feature of disease of the musculo-spiral nerve is wrist-drop, owing to paralysis of the extensor muscles. The triceps is often paralyzed in crutch palsy, but usually escapes when the nerve is damaged lower in the arm. The supinator longus and extensor carpi radialis longior are usually affected, but sometimes escape when the nerve is injured below the origin of the branches to these muscles.

Fig. 3.

Wrist-drop from paralysis of the musculo-spiral nerve (Leube).

There is loss of power in extending the wrist and fingers and in extending the arm when the lesion is high up, owing to paralysis of the triceps. The power of flexion of the hand is much diminished, owing to the loss of support of the extensors.

In severe cases wasting of the muscles takes place, and a prominence on the back of the hand develops, owing to protrusion of the synovial sacs and perhaps of the metacarpal bones.

Sensation even in severe cases of paralysis is seldom much affected —a fact which is explained by the theory of "vicarious sensation," the skin supplied by the radial being innervated by other nerves: although some loss of sensation is at times experienced over the radial side of the back of the hand and fingers and the ball of the thumb, it is more common to find various forms of paræsthesiæ.

From lead paralysis the distinction is made by the history of a cause, such as sleep or crutch palsy, by the more rapid onset, by the unilateral character, and often by the involvement of the supinator longus.

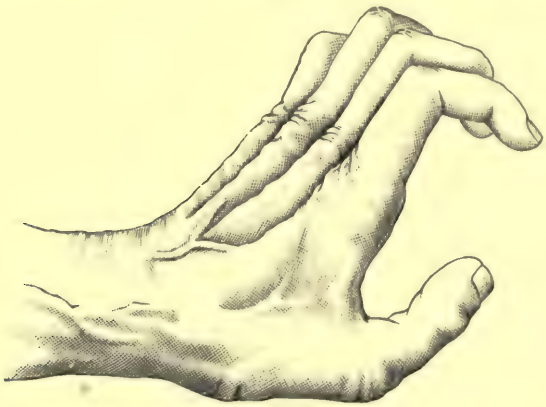
DISEASES OF THE ULNAR, MEDIAN, AND MUSCULO-CUTANEOUS NERVES.

THE muscles of the front of the forearm and hand are supplied by the ulnar and median nerves. The former supplies the flexor carpi ulnaris, the two inner heads of the flexor profundus digitorum, the interossei and two inner lumbricales, the adductors and inner head of the flexor brevis pollicis, and the muscles of the hypothenar eminence. The other flexors and pronators and small muscles of the thumb are supplied by the median. The cutaneous branches of the ulnar supply the ulnar side of the hand, front and back, and half the ring finger, and on the dorsum the basal phalanx of the middle finger.

The median supplies on the palmar surface the radial side of the hand and three and a half fingers, and often the terminal phalanges of the first and second fingers on the dorsum.

Ulnar Nerve.—Owing to its position, the ulnar nerve is often

FIG. 4.



Paralysis of ulnar nerve (Leube).

paralyzed from pressure or from injuries to the elbow, and its superficial course at the wrist renders it specially liable to wounds. Like the other brachial nerves, it may be affected by a primary neuritis.

In complete paralysis of the ulnar nerve the wrist is flexed imperfectly and toward the radial side, whilst the third and fourth fingers can only be incompletely closed.

The most important defect results from paralysis of the interossei. These small muscles flex the first phalanx and extend the two terminal ones, as in the upstroke in writing. The loss of this movement is less noticeable in the first and second fingers, as the first two lumbricales which supplement the interossei are supplied by the median. There is also inability to separate the fingers, to adduct the thumb, and to use the special muscles of the little finger forming the hypothenar eminence.

Considerable flattening of the hypothenar eminence from atrophy is always present. In old-standing cases the hand is drawn toward the radial side, the first phalanges are hyperextended by the unopposed common extensor, and the two terminal phalanges flexed, which, com-

bined with atrophy of the hypothenar muscles and interossei, give rise to the peculiar deformity of the hand described by Duchenne as the *main en griffe*, or claw hand. (Figs. 4, 5.)

Sensation, as in disease of the other nerves, varies considerably. There may be only certain subjective sensations, as numbness or pins and needles, or there may be loss of sensation over one and a half fingers and the adjacent parts of the hand, front and back.

Median Nerve.—Paralysis of this nerve usually results from injury to the arm, and is commonly associated with damage to other nerves.

FIG. 5.

Paralysis of ulnar nerve (Leube).

Cases of isolated palsy are usually the result of injury to the forearm or division at the wrist.

In complete paralysis there is inability to completely pronate the forearm, flexion at the wrist is weakened, and the hand drawn toward the ulnar side. Flexion of the fingers is lost, except the terminal phalanges of the third and fourth fingers, whilst the thumb is adducted and extended, lying alongside the hand. Owing to paralysis of the long flexor of the thumb, the second phalanx cannot be flexed, but the most important defect is the loss of power to oppose the thumb and fingers, due to paralysis of the opponens pollicis.

There is a tendency to over-action of the interossei, owing to their opposing muscles being paralyzed, and in consequence the terminal phalanges are hyperextended or even dislocated backward. Atrophy of the thenar eminence is prominent, and more or less atrophy also occurs in the forearm. When the nerve is injured at the wrist the muscles of the forearm escape. Various grades of altered sensation occur over the cutaneous area supplied by the nerve, although, as with other mixed nerves, sensation may be unaffected.

Musculo-cutaneous Nerve.—This nerve supplies the biceps, coracobrachialis, and brachialis anticus, and sends cutaneous branches to the outer side of the forearm.

Only a few cases of paralysis are reported, and the chief symptom is due to loss of power of the biceps.

Numbness on the outer side of the forearm is also usually present.

DISEASES OF THE LUMBAR PLEXUS.

THE lumbar plexus is formed from the first, second, third, and part of the fourth lumbar nerves. Its most important branch is the anterior crural supplying the iliacus, quadriceps extensor, sartorius, and pectineus, also the skin over the front and inner side of the thigh, and by its long saphenous branch the skin on the inner side of the leg and foot. The obturator from the third and fourth nerves is distributed to the adductors, obturator externus, and gracilis, and skin of the upper and inner sides of the thigh, and to the knee- and hip-joints. The other branches of the plexus are the ilio-inguinal, ilio-hypogastric, external cutaneous, and motor twigs to the psoas and quadratus lumborum.

Paralysis of one or more of these branches results from the pressure of deep-seated abdominal growths, bone disease, or psoas abscess. They are sometimes affected by spontaneous neuritis.

Anterior Crural Nerve.—The superficial position of this nerve at Poupart's ligament renders it liable to injury from wounds, dislocations of the hip, or fractures of the pelvis. Bernhardt has noted strain as a rare cause of paralysis. The nerve may also suffer in disease of its roots from spinal lesions.

SYMPTOMS.—When the lesion is situated about the level of Poupart's ligament the extensors of the leg are paralyzed, and waste later on. Various movements are impaired, but the patient can still stand and walk, although in doing so there is a tendency to fall from contraction of the flexors of the knee.

When the nerve is damaged high up flexion of the hip is weakened, and, if the branches from the plexus to the psoas are involved by the morbid process, abolished.

Sensation may be affected over the lower two thirds of the front and inner side of the thigh (middle and internal cutaneous) and along the inner side of the leg and foot (long saphenous). The knee jerk is lost early in most cases.

The Obturator Nerve.—The obturator nerve, although rarely affected alone, is occasionally injured from operative interference during parturition, and possibly in some cases of obturator hernia.

SYMPTOMS.—The power of adduction is lost and outward rotation weakened.

Sensation may be impaired on the inner and upper part of the thigh, and reflected pain in the knee-joint is a classical symptom of irritation of the nerve.

The external cutaneous nerve is occasionally affected alone. The

symptoms are sensory, consisting in numbness, pain, and anæsthesia on the outer side of the leg.

The superior gluteal nerve, arising from the lumbo-sacral cord, is rarely diseased alone.

Paralysis of the two smaller gluteal and tensor facial muscles produces a loss of power of abduction and internal rotation. The unopposed external rotators contract, so that the toes point outward.

DISEASES OF THE LEG NERVES.

THE **sacral plexus** is formed by the lumbo-sacral cord, the first three and part of the fourth sacral nerves. Its chief trunk is the great sciatic, but it also gives off the small sciatic, supplying the skin on the back of the thigh and upper half of the leg, together with several small muscular and cutaneous branches.

The **great sciatic nerve** supplies the flexors of the legs and terminates by dividing into the internal and external popliteal branches. This nerve is frequently the seat of neuritis (see Sciatica), and has been divided by wounds in the thigh. Paralysis has also followed the operation of stretching the nerve.

SYMPTOMS.—The symptoms of paralysis vary according to the seat of injury. If affected in the pelvis or above the origin of the branches to the hamstrings, there is loss of power in the flexors of the legs, as well as paralysis of all the muscles below the knee. The patient is still able to walk, the knee being fixed by the quadriceps extensor and gracilis, and the limb being thrown forward by the flexors of the thigh. The toes reach the ground first, and the forward movement of the paralyzed leg is very short.

External Popliteal Nerve.—The external popliteal nerve, owing to its superficial position in close contact with the head of the fibula, is by far the most frequently paralyzed of all the nerves of the leg. It is liable to contusions as it winds round the head of the fibula, and it is the nerve most frequently attacked in multiple neuritis.

SYMPTOMS.—In paralysis there are foot-drop and inability to flex the ankle. The foot is adducted, owing to paralysis of the peronei and contraction of the unopposed tibialis posticus. Contraction of the interossei produces flexion of the first and extension of the two terminal phalanges. The gait is peculiar, and is termed "steppage," the patient flexing the thigh unduly to enable the foot to clear the ground. After the lapse of a few weeks atrophy of the affected muscles takes place. The electrical reactions are also characteristic of nerve degeneration. Sensation is lost over the outer side and dorsum of the foot.

The **internal popliteal** and its branches supply the muscles of the back of the leg and sole of the foot. Owing to its deep position it is not easily damaged by trauma.

SYMPTOMS.—There is loss of power of the calf muscles and deep flexors, so that extension of the ankle is impossible in such movements as walking. Contraction of the muscles on the front of the leg produces pes calcaneus.

The **external plantar nerve** supplies the interossei, and paralysis of

these muscles produces a deformity similar to that seen in the fingers in paralysis of the ulnar nerve. Anæsthesia of the sole of the foot, which is supplied by the plantar nerves, may be present in lesions of the internal popliteal nerve.

Diagnosis.—Acute poliomyelitis usually attacks the muscles supplied by the external popliteal, and less often those supplied by the anterior crural. The spinal disease is usually readily recognized by a history of an acute onset occurring in childhood.

Muscular atrophy of spinal origin is progressive and bilateral. Lesions of the cauda equina may cause paralysis of the nerves within the spinal canal and simulate peripheral disease. In such cases the disease is usually, but not always, bilateral.

Treatment.—The rules governing paralysis of other nerves are applicable to those of the leg. Various mechanical contrivances to prevent deformity by contractures or to support the limb are useful. For a description of these the reader is referred to works on orthopædic surgery.

SCIATICA.

The term "sciatica" is applied to painful affections of the sciatic nerve. Although usually due to a perineuritis of the nerve, its occasional dependence on neuralgia or on the pressure of tumors or inflammatory exudation in the pelvis must be borne in mind.

Etiology.—The disease is about four times more frequent in men than women. Unknown in childhood, it is extremely rare under twenty and is most common from forty to sixty.

Rheumatic and gouty diatheses predispose to sciatica. It is particularly common in individuals subject to that form of rheumatism which attacks muscles and fibrous structures, and it is therefore not uncommon to find a history of lumbago or of pain about the fibrous attachments to the ilium. A gouty history is sometimes traced in the family, or there is in other cases a history of articular or other forms of gout.

Exposure to cold, especially when the individual is overheated, is an important cause. It thus very commonly affects the laboring classes. Exposure from standing in water, from wet feet, or from sleeping in damp clothes has been repeatedly followed by this condition. Another occasional antecedent is over-exertion of the muscles, as from the use of a sewing-machine or from a long walk, and prolonged pressure from sitting on a hard stool sometimes induces it. The pressure of tumors in the pelvis or rectum, impacted fæces, peri- or parametritis, or aneurysms may induce the symptoms of sciatica. Such cases must be carefully distinguished from primary sciatica.

Pathological Anatomy.—Opportunities of examining the nerves are rare, but changes indicative of perineuritis, sometimes extending to the interstitial tissue of the nerve, have been found. Whilst most intense at the sciatic notch or about the middle of the thigh, the signs of inflammation of less degree sometimes extend along considerable tracts of the nerve or its branches.

The constant character of the pain, the tenderness of the nerve

trunk, and the muscular wasting are also features indicating that the affection is a neuritis and not merely neuralgic.

Symptoms.—The principal symptom, and by far the most distressing, is pain. Beginning gradually, seldom suddenly, it soon becomes more intense and often of an extreme character. Its points of greatest intensity are at the sciatic notch, in the middle of the thigh, and about the posterior superior iliac spine. It frequently radiates along one or both main branches of the nerve, more frequently the peroneal branch of the external popliteal, producing severe pain, especially about the head of the fibula, the external malleolus, and the dorsum of the foot. Whilst the pain is sometimes diffused, it often follows the course of the nerve. It is variously described as burning or boring, dull or acute, and in different paroxysms may affect the whole area of distribution or shift from one part to another.

Any movement causes an exacerbation of pain, and the limb is held slightly flexed at the hip- and knee-joints. Some cases, however, seem to derive more ease by moving about. Nocturnal exacerbation is usual, but the pain remains during the day.

Neighboring nerve territories are sometimes affected; anæsthesia over the back of the thigh indicates involvement of the small sciatic; pain with flabbiness of the muscles on the front of the thigh is probably due to a neuritis ascending the lumbo-sacral cord and involving the lumbar plexus; secondary involvement of the cord has also been observed in extremely rare instances.

When the disease is well established the nerve trunk becomes tender to pressure, especially at the sciatic notch, in the thigh, and about the head of the fibula. This tenderness is sometimes best elicited by putting the nerve on the stretch, as by asking the patient to stand and bend the body forward, or by seating him on a chair with the knee at a right angle and pressing on the ham with the fingers.

Numbness, tingling, or other subjective sensations are sometimes present, and small areas of anæsthesia or hyperæsthesia may occur.

The muscles show a certain amount of flabbiness and wasting, and in old-standing cases a difference of from one to two inches in the measurements of the thighs and lower legs is not infrequent. The buttock is also somewhat flatter than on the healthy side. Muscular cramp and twitchings are occasionally seen. Slight alterations in the electrical reactions are sometimes present, but very rarely amount to the reaction of degeneration.

Herpes along the course of the nerve has been described, but is very unusual.

The duration of the disease is notoriously uncertain. Slight cases may recover in a few weeks, whilst others drag on for from three to six months or even a year. Relapses are apt to occur, and still more common is recurrence. Bilateral disease is not common, but both nerves may suffer at different periods.

Diagnosis.—The pain is usually readily localized in the nerve by its site and the presence of tenderness.

Indications of rheumatism or of gout, such as the presence of Heberden's nodes, and in the latter case of tophi also, should be looked for. A careful examination of the rectum and vagina sometimes reveals

the presence of a growth and clears up a doubtful case. Hip-joint disease is usually readily distinguished by the tenderness produced on percussing the heel or acetabulum.

Although neuralgia of the sciatic may occasionally occur, it is certainly rare. It is distinguished by the more transitory character of the pain, by its subjects being usually weakly and anæmic or subject to neuralgia elsewhere, and by the absence of any sign of nerve injury, such as muscular wasting or patches of anæsthesia.

TREATMENT.—The most important part in the treatment of sciatica is rest in bed. In severe or obstinate cases a good deal of success has been attained by adopting Weir Mitchell's method of keeping the limb immobile with a long splint, such as is used for fractures of the thigh. In many of the mild cases the patient walks about, but there is always a risk of severe symptoms setting in.

Counter-irritation is of signal service, and often materially shortens the course of the disease. Small blisters may be applied every other night along the course of the nerve, or mustard may be used, but is less serviceable, and in the acute stage hot lead-and-opium fomentations. Weir Mitchell speaks highly of ice in combination with the long splint. The application of the thermo-cautery is a very valuable measure and not very painful. Gowers recommends small doses of mercury in the acute stages.

In rheumatic cases salicylate of soda is sometimes very efficacious, but more often fails. Saline purgatives should be freely given in gouty individuals.

Pain when severe requires the use of sedatives. Morphine hypodermically is the surest, but should be avoided as long as possible. Other sedatives used in neuritis may also be tried. Acupuncture sometimes gives temporary relief, whilst sedative and stimulating liniments, such as equal parts of belladonna and chloroform, are sometimes used, but are usually inferior to other forms of counter-irritation.

Galvanism (10 to 20 ma.) is sometimes very serviceable in relieving pain, especially in the later stages. When the disease is very obstinate nerve-stretching may be resorted to, but the results, as a rule, are not very brilliant.

COCCYDYNIA is a painful affection of the coccygeal nerves, probably depending on irritation or neuritis. It is met with chiefly in women, and is usually attributed to a fall or to injury during labor. It may, however, originate without any obvious cause or from exposure to cold. The occasional presence of growths in the rectum should not be overlooked as a possible cause.

SYMPTOMS.—The chief symptom is pain in the region of the coccyx, increased by sitting or walking and sometimes by micturition or defecation. There is tenderness to pressure over the bone.

The COURSE is prolonged, and it is often rebellious to treatment.

TREATMENT.—The usual forms of counter-irritation, such as are used in sciatica, may be tried. If they fail, excision of the coccyx has been followed by relief in many instances. Subcutaneous section of the tissues attached to the borders of the bone is also efficacious.

DISEASES OF THE SPINAL CORD.

DISEASES OF THE SPINAL CORD.

THE DIAGNOSIS AND LOCALIZATION OF SPINAL-CORD DISEASES.

By M. ALLEN STARR, M. D.

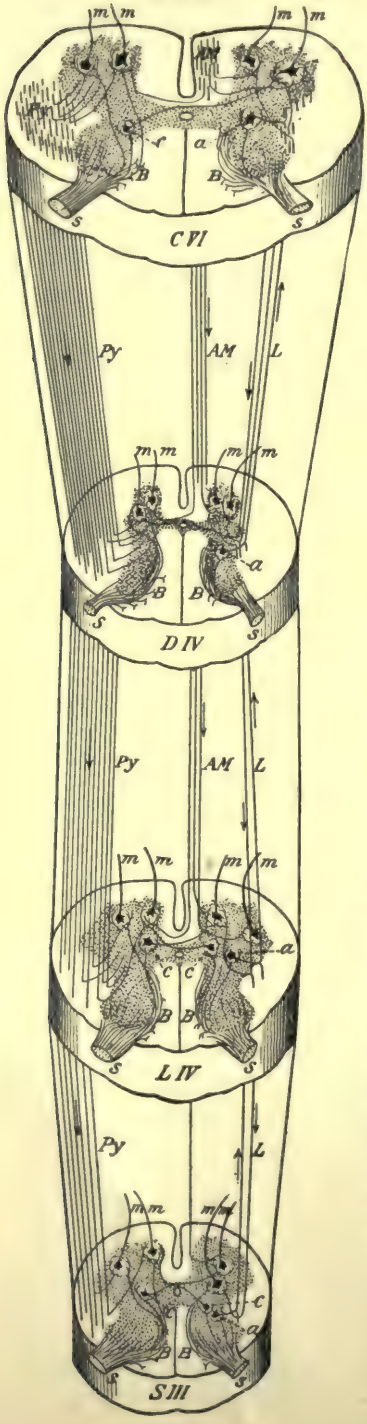
The general practitioner approaches a case of nervous disease from the side of the symptoms. It is therefore important to analyze each symptom and to show its pathological significance; for if a defect of function is carefully studied with the anatomical basis of that function clearly in mind, the symptom will be found to point directly to the situation of the pathological process; the degree and location of the symptom will indicate the extent of the lesion; and the combination of symptoms will lead to the diagnosis of the exact form of disease.

This is particularly true of diseases of the spinal cord, for this organ is made up of thirty-one segments, each segment consisting of a mass of gray matter connected with a pair of spinal nerves which pass to a definite region of the body; and each segment is joined to the others and also to the brain by means of nerve tracts running through the various columns which surround the gray matter of the segment.

The spinal cord is an organ which has two distinct functions: namely, (1) the function of controlling directly the various parts of the body with which it is joined by means of its pair of nerves; and (2) the function of communicating impulses to and from the brain. In considering, therefore, the functions of the cord and the symptoms which arise from disturbance of these functions it is necessary to understand not only the function of each spinal segment so far as its motor mechanisms, its sensory connections, its vasomotor and trophic functions, and the distribution of its particular pair of spinal nerves, are concerned, but also the functions of the columns of the cord which pass through the particular segment concerned, and transmit motor impulses downward from the brain and sensory impulses upward toward the brain.

In this article an attempt will be made to consider carefully each symptom which may present itself in any case of spinal-cord disease—to refer these symptoms one by one to the anatomical structure whose function is impaired, and thus to determine what the pathological import of each symptom may be. This will demonstrate how readily the localization of spinal affections may be determined, and from this localization and the combination of the symptoms it will be easily possible to arrive at a diagnosis of any form of spinal-cord disease.

Fig. 6.



Symptoms.—The symptoms of spinal-cord disease are paralysis, changes in reflex activity, disturbance in the control of the sphincters, alterations of gait and of posture, defects of sensation, ataxia, pain, and trophic disorders.

Paralysis is a condition of weakness or total loss of power in a muscle. It may be limited to a single muscle, it may affect a group of muscles, or it may affect a limb in its entire muscular apparatus. Paralysis may be due to an interference with the transmission of voluntary impulses from the motor centres of the brain to the motor cells (neurons) of the spinal cord, which impulses pass in the motor tracts of the spinal cord—viz. in the lateral pyramidal and anterior median columns. Paralysis may also be caused by a destruction of the motor neurons situated in the anterior gray horns of the spinal cord, whose axis-cylinder processes (axons) pass directly to the muscles through the anterior nerve roots and the motor nerves of the body. There are, therefore, two forms of spinal paralysis, quite clearly distinct from one another according as the lesion affects the first (cortico-spinal) or the second (spino-muscular) parts of the motor mechanism.

The diagram (Fig. 6) shows these two parts of the motor mechanism. The motor neurons of the brain send their axons downward through the pyramids of the medulla, where a partial decussation takes place, the majority of the axons from one pyramid crossing into the opposite lateral pyramidal tract of the cord (Fig. 6, *Py*),

Diagram of the spinal cord, showing the motor mechanism: *Py*, lateral pyramidal; *AM*, anterior median columns transmitting voluntary impulses from the right pyramid of the medulla to the motor cells of the anterior horns of the cord, whence motor nerves issue in the motor root (*m*); *s*, sensory nerve sending its fibres into the posterior horn and into the root zone of the column of Burdach (*B*), whence fibres pass forward to reach the commissural cells (*c*) and the association cells (*a*), and the motor cells (*m*); *L*, fibres of the lateral limiting layer, consisting of association neurons between various levels of the cord, being branches of the cells (*a*).

and the remainder passing directly into the anterior median column (Fig. 6, *AM*). As these axons pass downward they terminate at various levels in the anterior gray horns of the spinal cord, their terminations taking the form of fine brush-like expansions which surround the motor neurons of the cord (Fig. 6, *m*), each filament coming into contact with the protoplasmic processes (dendrites) of those neurons, but not being continuous with those dendrites. As these motor axons pass downward to their termination they give off small fine branches (collaterals) at right angles to their course, and these collaterals terminate in the same manner in brushes around the motor neurons of the cord. Thus a voluntary impulse starting from a motor neuron of the brain may reach several motor neurons of the cord. As the motor tract passes downward through the cord it becomes smaller and smaller, but few fibres remaining in the anterior median column below the dorsal region, while some fibres of the lateral pyramidal tract extend to the very last segment of the cord.

This motor tract is much more highly developed in those animals which make use of the digits of their extremities. Thus an elephant has no pyramid, while man has the most highly developed pyramidal tract. Hence it is evident that this tract transmits those voluntary impulses which are concerned in the finely adjusted movements of volition.

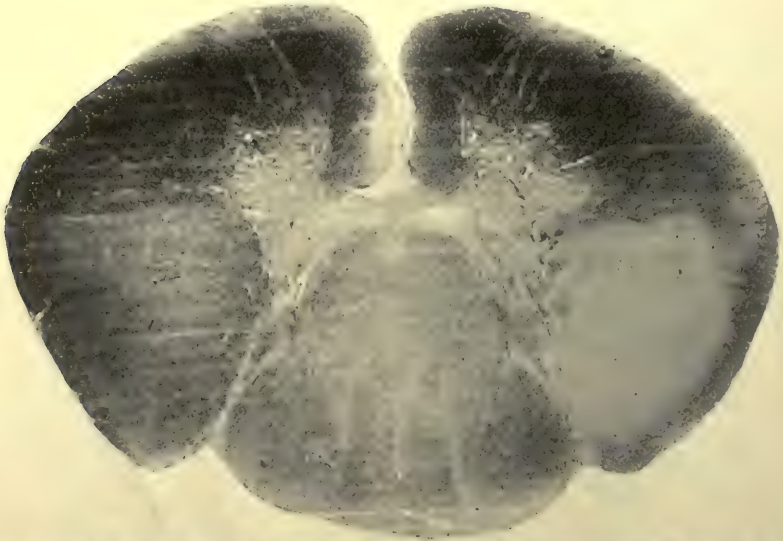
Each axon in this tract, being a process of a motor neuron of the cerebral cortex, is dependent upon that neuron for its nutrition. If the cortical neurons are destroyed or if the axon is separated at any point in its course from its neuron, it degenerates to its terminal brush. Hence a degeneration of the motor tracts of the cord may be due to disease in the brain, as in hemiplegia, or may be due to any transverse lesion of the cord which separates the axons from their neurons. The following figures (7–10) of specimens demonstrate descending degeneration of the motor tracts in the cord. Figs. 7 and 8 show descending degeneration consequent upon hemiplegia, of slight and of severe type, the anterior median column on the side of the brain lesion and the lateral pyramidal tract upon the other side being degenerated. Figs. 9 and 10 show descending degeneration in both lateral pyramidal and anterior median columns consequent upon a transverse myelitis located at some distance above the level from which the section is taken.

Paralysis of the cortico-spinal type, which is always due to a disease in this first element of the motor tract, has the following characteristics: The muscles paralyzed are partially and not absolutely paralyzed. All the muscles of the limb affected are about equally involved; the limb is stiff, its joints being moved with difficulty and slowly on account of the rigid condition of the muscles. The muscles are thus in a state of slight tonic contraction, and are hypersensitive to mechanical irritation, so that tapping a muscle produces a quick contraction, and tapping its tendon causes a prompt response. The muscles show no tendency to atrophy, though from disuse they may gradually become somewhat small. They are never flabby; their nutrition is good; there is no change in their electric excitability. In this form of paralysis the circulation of the limb is sometimes impaired. There is slight blueness and coldness in the extremity and a tendency to œdema. Sensory dis-

FIG. 7.

Section of the spinal cord, at the sixth cervical segment, showing descending degeneration in the left lateral pyramidal and the right anterior median columns, after a small lesion in the motor tract of the right cerebral hemisphere.

FIG. 8.

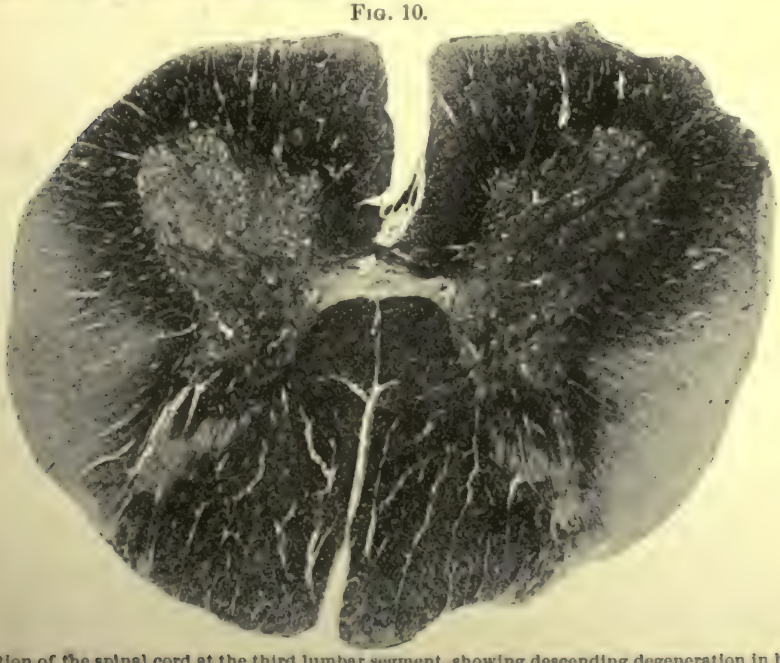


Section of the spinal cord, at the fifth cervical segment, showing descending degeneration in the left anterior median and right lateral pyramidal tract, after extensive lesion in the left cerebral hemisphere. There is slight degeneration in the left pyramidal tract and in the posterior columns.

FIG. 9.

Section of the spinal cord (somewhat distorted) in lower cervical region, showing bilateral descending degeneration in both anterior median and lateral pyramidal tracts. This section demonstrates the unequal size of corresponding columns on the two sides of the spinal cord. When the anterior median column is large in extent the opposite pyramidal tract is correspondingly small. Integrity of the direct cerebellar columns is evident.

FIG. 10.



Section of the spinal cord at the third lumbar segment, showing descending degeneration in both lateral pyramidal tracts. A few fibres are degenerated in the anterior median columns. At this level there are no direct cerebellar columns.

turbances do not necessarily attend this form of paralysis, and, if they are present, are an indication of disease elsewhere than in the motor tract.

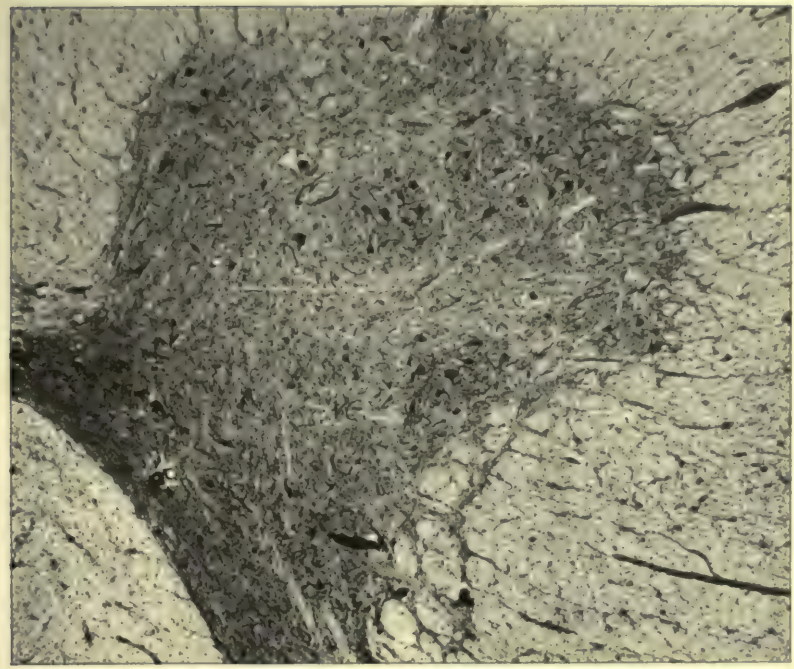
This type of paralysis is seen after all forms of cerebral disease, and then usually affects the arm and leg upon one side (hemiplegia). It is also seen in both lower limbs after a transverse lesion of the cord, such as may be produced by Pott's disease, by transverse myelitis, by softening from thrombosis, by hemorrhage, or by tumors of the spinal cord. If this transverse lesion is in the cervical region, the arms are also involved. It is also seen in primary lateral sclerosis and in syphilitic paraplegia, in which conditions it is limited to the legs.

The second or spino-muscular type of paralysis has an entirely different set of characteristics. The muscles affected are usually entirely paralyzed at the outset, and if they recover at all recover slowly and imperfectly. While all the muscles of the limb may be affected, as a rule they are not, a few muscles here and there upon the member being apparently paralyzed, while others retain their power; or if all the muscles are paralyzed at first, some will recover to a greater extent than others. The limb is never stiff, but hangs helpless, yielding to the force of gravitation, its joints being relaxed and the articular surfaces no longer being held in close approximation by the tonic contraction of the muscles; hence these joints are all more freely movable than in health. The muscles are relaxed and flabby. They do not respond to mechanical irritation by tapping with a hammer, and tapping their tendons does not cause contraction; the so-called tendon reflex is lost. The muscles atrophy very soon after they are first affected, and this atrophy may go on rapidly until but little of the muscle is left. There is an early appearance of the reaction of degeneration in the muscle; that is, it no longer responds when either faradic or galvanic excitation is passed through its nerve, and it no longer responds when faradism is applied directly to it. The circulation in the limb is always impaired in this form of paralysis. The vessels are relaxed, the blood pressure is decreased, there is a slow capillary circulation, the limb is blue and cold, and all the chemical processes seem to be delayed; hence the surface temperature is much colder than in the first type of paralysis. Soon after the onset the limb is likely to be covered with a clammy perspiration, and later on, when this is no longer present, it is very difficult to heat it up to the temperature of the other side. Sensory disturbances do not necessarily attend this form of paralysis.

This type of paralysis is due to a lesion of motor cells in the anterior gray horns of the cord (Fig. 6, *m*) or to an affection of the nerve trunks containing the axons of these motor neurons. It occurs in infantile spinal paralysis or anterior poliomyelitis, acute or chronic; in amyotrophic lateral sclerosis; in myelitis, either localized in a few segments or extensive throughout the cord; in syringomyelia when the lesion invades the anterior horns; in tumors and hemorrhages within the cord; and in softening of the cord due to embolism or thrombosis.

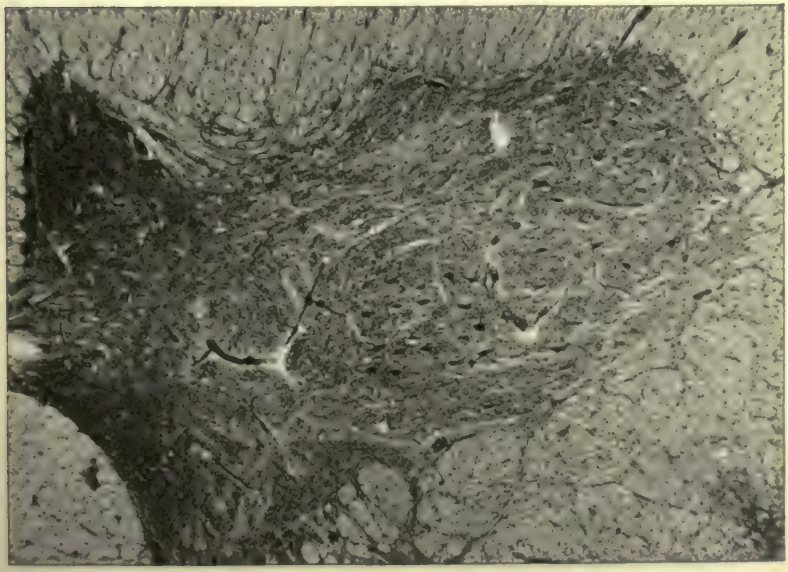
The reason for these characteristics of this second type of paralysis will be better understood if the anatomical structure of the second part

FIG. 11.



The groups of cells in the spinal cord at the fifth cervical segment.

FIG. 12.



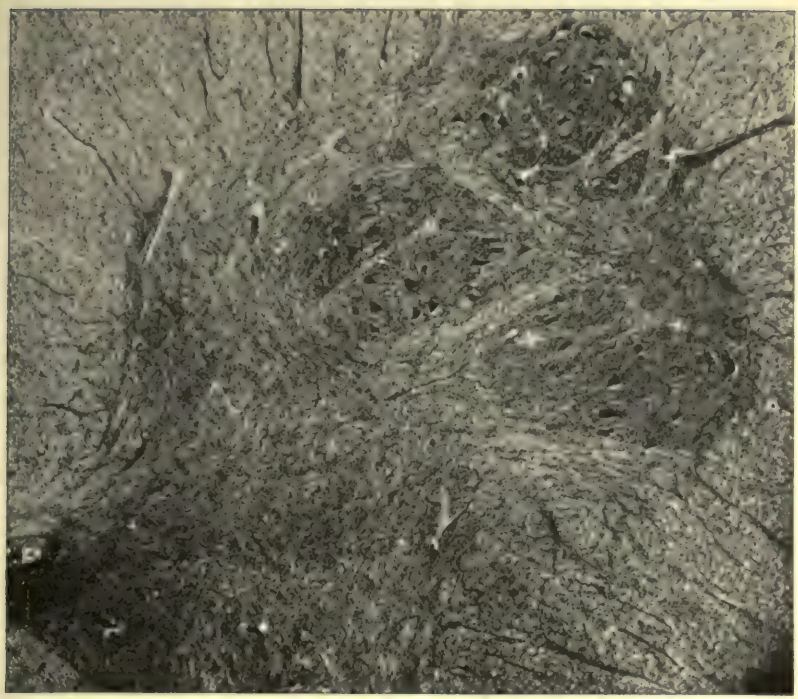
The groups of cells in the anterior horns of the spinal cord at the fifth cervical segment.

of the motor tract is considered. The motor neurons of the cord lie, as already stated, in the anterior part of the gray matter. These motor neurons are not scattered irregularly through the gray matter, but are collected into groups, the larger number of groups lying in the cervical and lumbar enlargements. The number of these groups varies greatly in different segments of the cord, as is shown in Figs. 11 to 16, and the groups have a varying extent longitudinally, so that while some groups are limited to a single segment, others extend through several segments. Each group of neurons controls a single muscle or a group of muscles which act simultaneously and in harmony. The fundamental movements of flexion and extension of the larger muscles of the limbs are represented in the large groups of the median, anterior, and lateral parts of the anterior horn. The accessory movements of the fingers and toes which are peculiar to monkeys and to man are represented in the smaller groups lying nearer the central portion of the gray matter. In Figs. 11 to 16 groups of cells at six different levels of the cord are shown, and it will be seen that they differ in their situation and extent at different levels. A careful study of comparative anatomy and of the lesions occurring in anterior poliomyelitis, in which disease single groups of cells are affected, has enabled us to determine the exact level in the cord of the various groups of cells representing the various muscles of the body. These are shown in Table I., which gives each segment of the cervical, lumbar, and sacral regions, with a list of the muscles represented in each segment. It will be noticed that some muscles are represented in two or even three segments, while other muscles are represented in but one. It is evident, therefore, that if the lesion in the cord is limited to one segment, it will paralyze completely two or three muscles which are represented by motor neurons in that segment only, and it will paralyze partially other muscles which are represented not only in that segment, but also in adjacent seg-

FIG. 13.

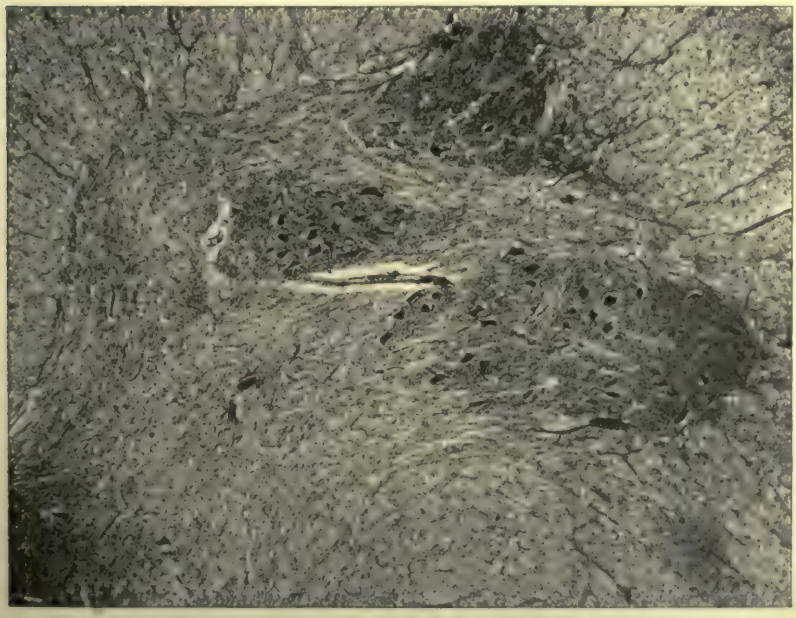
The groups of cells in the dorsal region of the spinal cord.

FIG. 14.



The groups of cells in the third lumbar segment of the spinal cord.

FIG. 15.



The groups of cells in the fifth lumbar segment of the spinal cord.

ments. Hence the apparent irregular distribution and degree of the paralysis in various muscles upon the limb in cases of infantile paralysis.

TABLE I.—*Showing the Muscles represented in Groups of Cells in the Various Segments of the Spinal Cord.*

II., III. Cervical.	IV. Cervical.	V. Cervical.	VI. Cervical.	VII. Cervical.	VIII. Cervical.	I. Dorsal.
Diaphragm. Sterno-mastoid. Trapezius. Scalenus.	Diaphragm. Lev. ang. scap. Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps.	Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps. Supin. brev. Serratus mag. Pect. (clav.). Teres minor.	Biceps. Serratus mag. Pect. (clav.). Pronators. Triceps. Brach. ant. Long extensors of wrist.	Pronators. Triceps. Brach. ant. Long extensors of wrist and fingers. Pect. (costal). Latis. dorsi. Teres major. Long flexors of wrist and fingers.	Long flexors of wrist and fingers. Extensor of thumb. Intrinsic muscles of hands.	Extensor of thumb. Intrinsic muscles of hands.

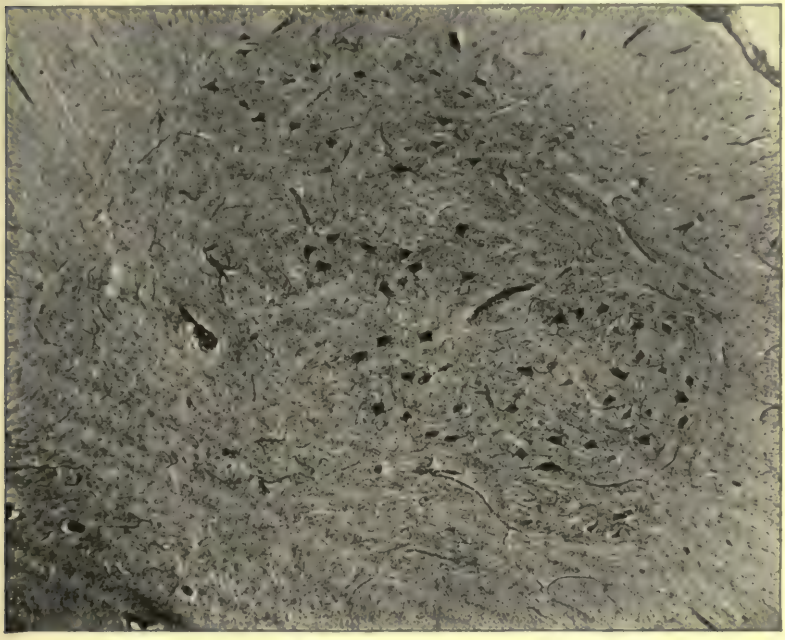
I. Lumbar.	II. Lumbar.	III. Lumbar.	IV. Lumbar.	V. Lumbar.
Quadr. lumb. Obliqui. Transversalis. Psoas. Iliacus.	Psoas. Iliacus. Sartorius. Quad. ext. cruris.	Quad. ext. cruris. Obturator. Adductores.	Obturator. Adductores. Glutei.	Glutei. Biceps femoris. Semi-tend. Popliteus.

I. Sacral.	II. Sacral.	III. Sacral.	IV. and V. Sacral.
Biceps femor. Semimemb. Ext. long. dig. Gastroc. Tibialis post.	Gastroc. Tibialis post. Tibialis anticus. Peronei. Intrinsic muscles of foot.	Peronei. Intrinsic muscles of foot.	Sphincter ani et vesicæ. Perineal muscles.

Certain special forms of the second type of paralysis are quite commonly recognized. Thus we have in anterior poliomyelitis the upper-arm type of paralysis in which the deltoid, biceps, supinator longus, and muscles about the shoulder-blade are affected together, the muscles moving the wrist and fingers escaping. Reference to the table will show that this form is due to a lesion in the upper part of the cervical

enlargement. We also have a lower-arm type of paralysis, in which the muscles which move the fingers and wrist are alone invaded, the supinator longus which lies among them escaping entirely. Reference to the table will show that this is due to a lesion of the lower cervical enlargement. The same distinction can be made in paralysis of the leg,

FIG. 16.



The groups of cells in the second sacral segment of the spinal cord.

there being a thigh type, a leg type and a foot type, according as the lesion is in the upper lumbar, mid-lumbar, and sacral segments. By reference to the table, therefore, it will be possible in any case of paralysis of the second type to arrive at a localization of the lesion or at an estimate of its extent in the cord when the muscles paralyzed are exactly determined. The electrical examination will assist in this determination, inasmuch as the muscles whose groups of cells are destroyed will have lost their faradic excitability, while the muscles whose groups of cells are intact will show no change in electrical reactions.

That the motor neurons of the cord have a distinct influence upon the nutrition of the muscle and upon the circulation in it is shown by the rapid atrophy and the vasomotor disturbances in the muscle which also attend this type of paralysis, and to which allusion has already been made.

It has already been stated that this type of paralysis may be due to a lesion in the axons arising from the motor neurons of the cord. It is to be remembered, however, that after their exit from the cord these axons are divided up into nerves, being distributed in various directions through the brachial and lumbar and sacral plexuses. A differential diag-

nosis can always be made between lesions of the spinal cord and lesions of the nerve trunks by the distribution of the paralysis; for the muscles which are paralyzed together from a lesion of a single nerve are different from the muscles paralyzed together from a lesion of the spinal cord. Thus the deltoid is often paralyzed from a lesion of the circumflex nerve alone, but is never paralyzed alone from lesion of the cord. Thus the extensors of the wrist, together with the supinator longus, are paralyzed in lesions of the musculo-spiral nerve, but, as already stated, these muscles are never paralyzed together in small lesions of the spinal cord, their groups of cells lying far apart. And what is true of the nerves of the brachial plexus is also true of the nerves going to the lower extremity; hence, while the characteristics of paralysis in lesions of the spinal cord and of the motor nerve trunks are the same, the association of paralyzed muscles with one another in the two conditions differs. A further point of differentiation is also found in the distribution of anæsthesia occurring with lesions of the nerve trunks, anæsthesia being frequently present in lesions of the nerve trunks, and not necessarily present in lesions of the motor neurons of the cord. Furthermore, when it is present in a lesion of the cord, as will be shown later, the distribution of the anæsthesia in the skin differs entirely from the distribution of anæsthesia after a lesion of the nerve.

It is thus possible, from a study of paralysis and of its essential characteristics and distribution in any case, to reach a diagnosis and a localization of the lesion, and to differentiate between lesions of the motor tracts of the cord or of the motor neurons of the cord or of the nerve trunks.

There are cases, however, of a general disease of the cord, such as a general myelitis involving both the white tracts and the gray matter, in which a third type of paralysis is observed. This presents some of the characteristics of both the former types, and, were the diagnosis to rest upon the examination of the paralysis alone, it might present certain difficulties. Fortunately, there are other symptoms always present to guide us in the diagnosis; for in a general inflammation of the cord the changes in reflex action and in the control of the bladder and rectum, and the tendency to very severe trophic disturbances, are so marked as to leave no doubt regarding the actual condition. In this third type of spinal paralysis the paralysis resembles more closely the second type than it does the first, there being the same total paralysis, the same atrophy, and the same reaction of degeneration in the paralyzed muscles. But the distribution of the paralysis is more extensive—is, in fact, frequently total in both legs and almost complete in both arms, and, although there is no rigidity of the limbs, there is sometimes an increase in the reflex activity and in the mechanical excitability of the muscles in the early stage before they are finally lost. This is particularly noticed in the disease amyotrophic lateral sclerosis in which both the lateral tracts and the anterior horns are simultaneously progressively destroyed. In this disease the first type of paralysis is present in the legs for several months, and the second type is present in the arms, associated with an increased mechanical excitability of the muscles, but finally this is lost, and the arms present the typical second type, which gradually and finally extends also to the legs.

In cases of injury of the spinal cord by fractures and dislocations of the vertebræ the spinal cord may be seriously bruised or it may be absolutely disintegrated. The symptoms of paralysis appear to differ somewhat in these two conditions, as has been shown by Thorburn[1] and by Kocher.[2] When the spinal cord is injured, but not destroyed, there is total paralysis below the level of the injury, with a condition of rigidity of the limbs and an increase of tendon reflexes. There is also a loss of control of the bladder, which may take the form either of retention of urine or of spontaneous evacuation of the bladder. There is likely to be some distention of the abdomen by gas, due to a paralysis of the intestinal wall.

When the spinal cord is absolutely divided or destroyed at any level, there is total paralysis below this level, the limbs being completely relaxed and not in a state of rigidity. The paralysis is symmetrical upon the two sides. The tendon reflexes are absolutely lost. There is always a retention of urine, which has to be relieved by catheter; there is tympanites with distention of the abdomen; there is a paralysis of the vaso-constrictors leading to a dilatation of the subcutaneous veins, and consequently to an increased temperature and to priapism. There is an increase in the genital reflex, obtained by pinching the testicle. There is a loss of sensibility to pain and temperature, and usually to touch also, at a definite level of the surface, as shown in the diagram, Plate I. In any case in which these characteristics are absent after an injury of the spine, it is certain that the cord has not been completely destroyed at the level of the lesion.

Spasmodic contractions of the muscles of an involuntary kind are associated with paralysis in many forms of spinal-cord disease. Like the paralysis, they can be divided into two categories. When the condition present is spastic paralysis and the muscles are rigid and reflexes exaggerated, patients often notice a spontaneous trembling of the entire limb due to alternate contraction of the two sets of opposing muscles. This is usually attended by an extensive spasm, so that the legs are stiffened and shake more or less violently. The condition is occasionally so extreme as to warrant the term *spinal epilepsy*, first applied to it by Brown-Séquard, although this term is misleading and should be discarded. This symptom is seen in lateral sclerosis from any cause, and is indicative of a lesion in the lateral columns of the cord. When it is present the lesion does not involve the spino-muscular element of motion.

Fibrillary twitchings of the muscles are much less painful than spasm of the entire muscle, but give considerable discomfort. Such fibrillary twitchings are present in diseases of the anterior horns of the cord, especially in chronic anterior poliomyelitis and in syringomyelia. Individual fibres of the muscle alternately contract, producing a little wave-like movement which goes on in the muscle itself, but does not involve a sufficient number of the muscle fibres to produce any contraction of the muscle as a whole. Such fibrillary twitchings can be

[1] Thorburn: *A Contribution to the Surgery of the Spinal Cord*, Philadelphia, 1893.

[2] T. Kocher: "Die Verletzungen der Wirbelsaule, Zugleich als Beitrag zur physiologie des Menschlichen Ruchenmarkes." *Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie*, vol. i. p. 181–460, Jena, 1896.

elicted by percussion of the muscle or by exposure of the limb to cold. They always indicate a disease in the motor cells controlling the muscle, as they are absent in all forms of muscular dystrophy in which the disease is exclusively a muscular affection; hence such fibrillary twitchings are always associated with the second type of paralysis. They occasionally accompany the third type of paralysis, in which the motor cells are also affected.

Disturbance of reflex action is a symptom of great importance in spinal-cord diseases, the increase in reflex activity or the suspension of reflex activity being both of great significance. The diagram (Fig. 6, p. 102) shows the anatomical basis of a simple reflex act. Whatever theory may be held with regard to the exact nature of the tendon reflexes, whether they are due to pure mechanical irritation of the fibres of the muscle, whose tone is maintained by spinal impulses, or whether they are due to a transmission of impulses through the spinal cord, the facts here stated hold true.

A reflex act is the immediate result of a sensory impression received in the spinal cord, and it takes place without necessarily producing any conscious perception and without any voluntary guidance. The sensory nerve enters the spinal cord through the posterior nerve root and divides into two parts, which separate in a Y-shaped division, one branch passing upward and the other downward in the root zone or column of Burdach (see Fig. 6, *B*). As these branches pass up and down they give off at right angles to their course small twigs (collaterals), and these collaterals, as well as the terminal filaments of the branches, terminate in brush-like expansions in the gray matter of the cord at various levels. Thus a sensory impulse entering in a posterior nerve is distributed to a considerable extent of the gray matter of the cord. Some of the terminal filaments pass forward to end about the motor neurons of the anterior horn of the same side upon which they enter; others pass forward and cross through the posterior commissure to terminate about the motor neurons in the anterior horn of the opposite side: these subserve reflex motor acts. Others terminate about the large cells in the median gray matter, which are the intrinsic or association cells of the cord, and transmit the impulses to other levels where they reach other motor cells (Fig. 6, *a*); others end about cells whose function is to control vasomotor and trophic reflex acts. Thus a single sensory impulse entering the cord may be widely distributed and awaken many reflex acts. Impulses entering the cord through the sensory nerve, and thus reaching various mechanisms of the cord, set up an activity in the various cells presiding over these mechanisms, and thence motor impulses pass outward to the muscles or to the viscera and result in their contraction, and hence in motion. The number of these reflex acts constantly going on in the body is enormous. In fact, in many of the lower animals the spinal cord is so much more developed than the brain that it is evident that almost the entire nervous mechanism acts without conscious perception or voluntary control. The whole regulation of nutrition, of circulation, of digestion, of reproduction, and of excretion is regulated by the spinal cord independently of the brain. This is proven by the fact that in man, when consciousness is suspended in sleep, in coma, or by extensive injuries of the brain cortex, as in paresis

and senile dementia, these vegetative functions go on in a normal manner. And it is well known that infants born with defective brains, or with almost no brain at all, may live for several months.

While the majority of these reflex acts are known to have mechanisms in the cord, there are only a few reflexes which are recognized and can be tested in health and disease. These reflex acts are—first, the tendon reflexes; second, the skin reflexes; third, the automatic functions of the bladder and rectum.

(*a*) *Tendon reflexes* are produced by tapping the tendon of a muscle near its insertion and thus producing a sudden contraction of the muscle. Thus the tendons about the wrist and elbow, the patellar tendon at the knee, and the Achilles tendon at the ankle can be excited in a state of health. We now know the exact level in the spinal cord which contains the reflex mechanism necessary to the existence of these various reflex acts. In Table II. the various spinal muscular reflexes are given, the method of producing the reflex, and the level of the segment controlling each.

TABLE II.—*Localization of Muscular Reflex Acts in the Spinal Cord.*

Reflex acts.	Localization in segment.
Pupillary reflex through the sympathetic: Dilatation of the pupil produced by irritation of the neck.	Fourth cervical to first dorsal.
Scapular reflex: Irritation of the skin over the scapula produces contraction of the scapular muscles.	Fifth cervical to first dorsal.
Biceps and supinator longus: Tapping their tendons produces flexion of the forearm.	Fifth and sixth cervical.
Triceps reflex: Tapping tendon produces extension of forearm.	Sixth cervical.
Tapping extensor tendons at the wrist causes extension of the hand.	Sixth to eighth cervical.
Tapping flexor tendons at the wrist causes flexion of the hand.	Seventh to eighth cervical.
Palmar reflex: Stroking palm causes closure of fingers; finger clonus.	Eighth cervical to first dorsal.
Abdominal reflex: Stroking side of abdomen causes retraction.	Ninth to twelfth dorsal.
Genital reflex: Squeezing the testicle causes contraction of the abdominal muscles.	First to third lumbar.
Patellar tendon: Striking tendon at knee causes extension of the leg; "knee-jerk."	Second and third lumbar.
Foot clonus: Extension of Achilles tendon causes flexion of the ankle.	First to third sacral.
Plantar reflex: Tickling sole of foot causes flexion of toes.	First to third sacral.

In the process of disease these reflex acts may be lost or they may be exaggerated. A loss of the reflex implies a lesion in the reflex mechanism, either in the sensory nerve, which would necessarily result in a coincident condition of anæsthesia, or in the sensory reflex fibres within the cord, which might give rise to this symptom alone, or in a destruction of the motor mechanism, which would give rise to a coincident condition of paralysis. If, therefore, by the distribution of the anæsthesia or of the paralysis it is possible to exclude in any case a

lesion of a nerve trunk, the loss of reflex necessarily implies a lesion of the spinal cord. This lesion will necessarily be limited to the segment controlling a reflex tested. Therefore, in the examination of any case of spinal disease it is incumbent to try each of the reflexes in turn, and, if any one of them is lost, to direct particular care to the examination of the other functions of the segment of the cord in which the lesion is indicated. The diseases in which the reflex activities are suspended are locomotor ataxia from the very earliest stage, and also those cases of general paresis in which there is an early complicating sclerosis of the posterior columns of the cord, syringomyelia, anterior poliomyelitis, general myelitis in the later stages, transverse myelitis at the level of the reflex arc, disseminated sclerosis when the patch of sclerotic tissue happens to lie in the reflex arc, tumors and hemorrhage in the cord at the site of the reflex arc. It is evident that a loss of tendon reflexes is a common accompaniment of the second type of paralysis.

An exaggeration of the tendon reflexes is also frequently observed as a symptom of spinal disease. This implies a suspension of the inhibitory impulses coming from the brain in a state of health, which impulses pass downward through the lateral columns in the motor tracts. The inhibition being removed, the spinal cord reacts more quickly and intensely to sensory impulses coming into it from without. In this condition a phenomenon known as *clonus* may be elicited in almost any one of the tendons of the longer muscles. Clonus consists of a rapidly repeated series of contractions in a muscle set up by a sudden over-extension of the tendon of that muscle. Thus if the patient's leg be allowed to rest upon the left hand of the examiner, and the right hand grasping the foot presses it suddenly backward, thus extending the Achilles tendon, if ankle clonus is present a series of vibrations of the foot will be produced, due to a repeated contraction of the muscles of the calf of the leg. A similar clonus may be elicited in the quadriceps femoris by a forcible pressure downward upon the patella when the limb is relaxed and extended. A similar clonus may occasionally be elicited in the tendons of the elbow, of the wrist, and of the fingers. The existence of clonus, like that of exaggerated reflexes, is an indication of disturbance of function in the lateral columns of the spinal cord, and is therefore commonly associated with the first type of spinal paralysis. It is also an early indication of any pathological process which interferes with the transmission of motor impulses from the brain to the cord. Thus in the early stage of Pott's disease, when slight pressure is made upon the cord or some disturbance of the nutrition of the cord occurs opposite the caries, the reflexes are commonly exaggerated in the segments below. Exaggeration of the reflexes is present in lateral sclerosis, in descending degeneration of the lateral columns below the level of the lesion, in syphilitic paraplegia, and in the early stage of acute myelitis when the pathological process irritates the motor neurons before they are destroyed. It is particularly noticeable in the arms in amyotrophic lateral sclerosis in the early stage of the disease, but as the process advances reflexes are lost in the arms, while they continue to be exaggerated in the legs. An increase of tendon reflexes is not infrequently observed in hysteria.

While it is true that a compression of the spinal cord of slight or

intense degree, or a partial destruction of the spinal cord in its upper region, always produces an increase of the tendon reflexes in the parts below the lesion, it has been observed by Miles and Bastian that in some cases where by injury (fracture or dislocation of the vertebræ) the spinal cord has been entirely divided or so completely crushed that all its elements have been destroyed, the tendon reflexes below the point of destruction have been abolished. This is not thought to be due to the concussion of the cord consequent upon the injury, as it may persist for many weeks. It has been ascribed to a setting up of abnormal inhibitory impulses from the point of destruction downward, due to the irritation of the lesion. This, however, is not accepted by Bastian, and no satisfactory explanation of the condition can be offered. In any case of localized crush of the cord from an injury the absolute loss of tendon reflexes is thought to indicate a total destruction of the cord, and in such a case any operation at the site of injury would be futile.

(*b*) *The skin reflexes* are not as well understood as the tendon reflexes. When certain areas of the skin are slightly stroked or tickled there occurs a slight movement of the skin at a part not under the point of irritation, but near to it, which seems to be due to the contraction of involuntary muscular fibres lying under the skin. These skin reflexes are best seen in animals, especially horses and cows, for the sting of a fly is capable of setting up a little fibrillary twitching in the skin of their bodies in almost any part. The following table (III.) gives a list of the skin reflexes found in man, the method of their production, and the level of the spinal cord that is supposed to control them, though it is not certain that they are under the control of a direct spinal mechanism:

TABLE III.—*Localization of Skin Reflexes in the Spinal Cord.*

Reflex acts.	Localization in segment.
Epigastric reflex: Stroking breast causes dimpling of the epigastrium.	Seventh to ninth dorsal.
Cremasteric reflex: Stroking inner side of thigh causes retraction of scrotum.	First and second lumbar.
Gluteal reflex: Stroking buttock causes dimpling in the fold.	Fourth to fifth lumbar.

The skin reflexes are usually lost in those diseases in which the tendon reflexes are exaggerated. They are also lost in brain diseases, but never in hysteria. They are never exaggerated.

The reflex mechanisms controlling the bladder and rectum are complex and are located in the fourth and fifth sacral segments of the spinal cord. The sensory impulses setting up this mechanism come from the mucous membrane of the bladder or of the rectum, and, passing inward to the cord, produce two separate effects: one is the active motor impulse of contraction in the muscles which empty these organs; the other is an inhibitory impulse arresting the action of those muscles which normally exert a constriction at the opening of these organs. Such evacuation of their contents by a reflex act may occur without the knowledge of the individual or without his control when disease cuts off the lower part of the cord from its communication with the brain, as in a transverse myelitis of the dorsal region. We then have a con-

dition known as active incontinence, in which the organs are emptied spontaneously at intervals in a normal manner. The mechanism itself, however, may be destroyed by any lesion of the sacral region of the spinal cord. Under these circumstances, the reflex arc being broken and the motor cells controlling muscular action being destroyed, the organs are no longer controlled. They then fill up to their utmost limit and evacuation has to be attained by outside aid. In some individuals a distention of the bladder finally overcomes a constrictive action of the sphincters, and then there is a constant leakage, producing a passive incontinence of urine, but the rectum shows no tendency to empty itself. In some individuals the constrictive action of the sphincters seems to be unusually strong, and occasionally a distention will lead to a rupture of the bladder rather than to its evacuation by water pressure. Sometimes there is a permanent weakness of the sphincter and a constant dribbling of urine without any distention of the bladder.

Disturbances in the action of the bladder and rectum constitute the chief sources of danger in spinal-cord disease, for a retention of urine is liable to set up catarrhal conditions, and finally cystitis, and the evacuation by means of a catheter is liable to lead to the infection of the bladder by germs unless the catheter is absolutely aseptic. Such a cystitis when once set up very often leads to pyelitis, nephritis, and death; hence the greatest care is to be taken of the bladder in spinal-cord disease. The evacuation of the rectum must also be carefully attended to, even though this involves its emptying by digital manipulation, as is commonly the case. Disturbance in the action of the bladder and rectum occurs in almost all forms of spinal-cord disease, and therefore is not diagnostic of any one special pathological lesion or of its location. It rarely occurs in hysteria, though retention is occasionally present.

The Gait in Spinal Disease.—In various forms of spinal-cord diseases the position and gait assumed involuntarily by the patient in walking are abnormal. In diseases which produce the first type of paralysis—*e. g.* lateral sclerosis—the gait is a stiff one: the hips and knees are held rigid and adducted, so that in walking the knees appear to touch or even to overlap, and the feet are not lifted from the floor, and hence are dragged or are pulled forward with effort, the great toe scraping the floor. At the same time the tendency to ankle clonus frequently leads to trepidation of the entire body from the shaking of the foot. These patients cannot step out freely, and hence their steps are very short, but quite regular in length. They rise from a chair or sit down slowly, there being an apparent resistance to the flexion and extension of the joints. Their motions are not awkward and can be guided accurately. This has been named the *spastic gait.*

In diseases involving the lumbar enlargement of the cord and affecting the gray matter alone or the entire cord, and producing the second type of paralysis, the gait is the *paralytic gait.* As the muscles grow weaker the support of the joints becomes imperfect, so that the aid of canes and crutches is needed to support the hip, and there is a tendency for the knee to be over-extended and for the ankle to turn. The paralysis of the muscles of the thigh makes the ordinary lifting of the feet from the floor impossible, and hence the leg is dragged along the floor, the inner side of the foot touching the floor, and the limb dragging rather loosely

as it is pulled forward between the crutches. There is none of the stiffness of the spastic gait and none of the uncertainty of the ataxic gait. If the anterior tibial group of muscles is paralyzed completely, there will be a drop-foot, and if this precedes paralysis of the thigh or is alone present, then the patient in stepping forward lifts the leg higher than normally in order to avoid stubbing the toe. This has been termed the *stepping gait*, as the patient has the appearance of stepping over an obstacle in his way or of raising his legs as if about to go up stairs. Such patients are soon unable to rise from a chair or to ascend steps.

In posterior sclerosis (locomotor ataxia) the gait is uncertain, the steps being of irregular length, the patient not appearing to place the foot upon the ground in the position in which he desires to, and hence he loses his balance frequently and makes irregular efforts to preserve it. He rises with difficulty from a chair, spreads his feet far apart, and sways for a moment after rising in order to get his balance. As the disease advances the gait becomes more irregular, with a tendency to long steps, to too great lifting of the foot from the floor, too sudden and violent placing of the foot downward upon the floor, so that the gait has been called a *stamping gait*. The feet are uniformly placed too far apart, as the patient seeks a wide base of support. The irregularity of the gait is increased by an attempt to walk backward or to walk forward with the eyes closed. This has been named the *ataxic gait*.

The Position Assumed in Bed.—When the patient is confined to his bed after an injury of the spinal cord, such as occurs from fractures and dislocations, the position assumed in bed differs according to the level of the injury.

If the sacral segments are crushed, the patient is unable to move the feet and ankles, and hence the feet lie in an extended position, assuming the position of drop-foot. The thighs and knees can be moved in this condition.

If the lesion involves the lower half of the lumbar enlargement, the patient lies with the thighs drawn up, the legs flexed, and he is unable to straighten the legs voluntarily or to lift the feet.

If the lesion involves the entire lumbar enlargement, the patient lies with the thighs, legs, and feet extended, and cannot move them.

If the lesion is in the dorsal region, the lower extremities are paralyzed more or less completely, but there is no tendency to drop-foot, and the limbs offer some resistance to passive motion, the tendon reflexes being exaggerated and the muscles somewhat rigid, unless there has been a total destruction of the cord, under which circumstances the muscles are relaxed and the tendon reflexes lost.

If the lesion involves the first dorsal and last cervical segments, the hands will be in a position of *main en griffe*, but the elbows and shoulders can be freely moved.

If the seventh cervical segment is destroyed, the forearms are partially flexed and lie upon the body with the hands pronated. Voluntary movements of the wrist are impossible, but the elbow and shoulder can be moved.

If the lesion lies at the sixth cervical segment, the arms are abducted from the side, the forearms are supinated, wrists and fingers being paralyzed.

If the lesion is at the fifth segment, the arms lie extended and relaxed at the side of the body, all motion being impossible.

These forced positions are assumed because of the fact that at any level of the cord the muscles controlled at and below the lesion will be paralyzed, and those controlled by the cells just above the lesion will be actively contracted by the state of irritation into which their motor centres are thrown, or if two centres of opposing action are irritated together, the stronger will overcome the weaker, and the resulting position, once assumed, cannot be corrected voluntarily; hence the fixed position once assumed is maintained.

Disturbances of sensation are important symptoms of spinal-cord disease. Any irritability of the sensory areas of the cord may lead to a hypersensitive condition of the skin, to the perception of ordinary impulses as extraordinary ones, and hence to the too keen appreciation of any sensation which may be started up. The irritation in the cord may be so great as to lead to hallucinations of sensation; that is, to the perception of sensations in the skin which are set up in the cord and do not really come from the skin (similar in origin to the tingling felt in the little finger on compressing the ulnar nerve at the elbow). Hence in any disease of the cord during the early stage of congestion, or in the irritation produced by beginning sclerosis, patients commonly complain of tingling and numbness, of sensations of burning or of cold, of sensations of pain, of fulness, of pressure, and of weight. These sensations are referred to the skin or to the limbs, and to the particular parts of the skin and limbs which correspond to the segment of the cord which is irritated. These sensations are classed together under the term "paræsthesiæ."

Any destruction of the sensory areas of the cord will lead to a suspension of sensations which are classed together as anæsthesiæ. Sensations are of several kinds—namely, sensations of touch, the loss of which is tactile anæsthesia; sensations of pain, the loss of which is analgesia; sensations of temperature, the loss of which is thermo-anæsthesia; and sensations of muscular sense, the loss of which leads to imperfect co-ordination or ataxia. As these sensations reach the spinal cord through the sensory nerve roots, any disease of these nerve roots affects uniformly all forms of sensibility. But the sensory nerve roots transmitting these sensations separate from one another after their entrance into the cord, and pursue different courses in their way upward to the brain. Hence it is possible for one form of sensation to be lost while the others are preserved in diseases affecting different tracts in the spinal cord. Thus in diseases of the root zone of the column of Burdach, through which all sensations pass, we have anæsthesia, analgesia, and ataxia. In diseases of the central gray matter of the cord we have analgesia and thermo-anæsthesia without anæsthesia or ataxia. In diseases of the posterior columns of the cord we may have ataxia alone or associated with anæsthesia. The course of these various impulses into the cord and upward through the cord is not as yet absolutely determined, but so far as it is known it is shown in the diagrams (Figs. 17, 18, pp. 91, 92).

The sensory fibres are the axons of neurons situated in the posterior spinal ganglia, and if they are cut off from connections with these

ganglia by any lesion of the nerve root or any lesion within the cord, they will degenerate from the point of lesion onward. A study of the degenerations following lesions of the nerve roots and following transverse lesions of the spinal cord has given us some knowledge of the course of the sensory tracts.

The fibres of the posterior nerve root on entering the cord divide in a Y-shaped manner, one branch turning downward and the other upward. The branch which turns downward is short. It descends in the portion of the column of Burdach lying anterior and external to the root zone, and terminates by sending its fibres (collaterals) into the gray matter of the posterior horn. The special function of these fibres is unknown. The branches that turn up are of various lengths, and some fibres ascend all the way to the medulla; these are the long sensory tracts. The great majority of the branches which turn upward terminate soon after their entrance into the cord in the segment into which they enter or in the segments just above it; these are the short sensory nerve fibres. They terminate in branches around the cells of the gray matter in the posterior and anterior horns and in the central gray. A few of the fibres also turn upward in a small zone lying near to their entrance, and between the tip of the posterior horn and the periphery of the cord known as Lissauer's column.

FIG. 17.

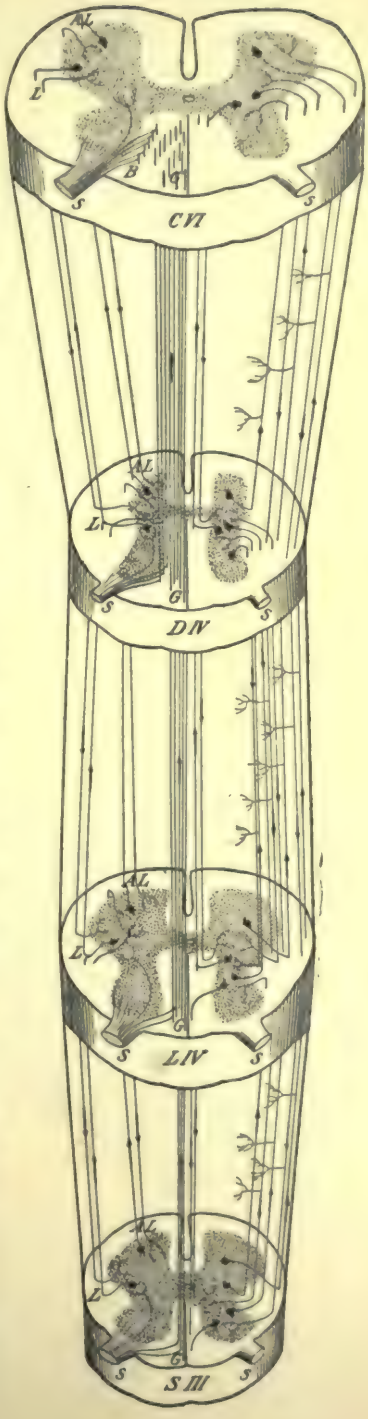
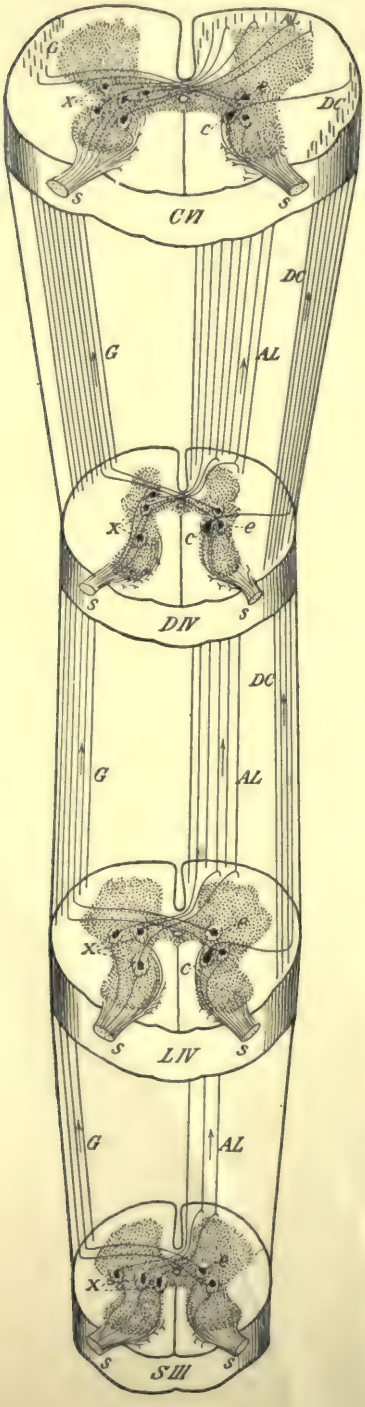


Diagram showing long sensory fibres in the posterior columns of the cord: *S*, sensory nerves whose fibres turn upward after entering the root zone. Each successive nerve root from below upward presses the fibres already ascending inward and backward, so that in the cervical region the fibres which have come from the sacral region lie in the column of Goll near to the posterior septum; the fibres from the lumbar region lie in the column of Goll external and anterior to those from the sacral region; the fibres from the dorsal region lie at the lateral part of the column of Goll; and the fibres from the cervical region lie in the column of Burdach. This diagram also shows association neurons of the cord whose axons are passing upward and downward in the marginal portion of the posterior column and in the lateral (*L*) and antero-lateral (*AL*) columns of the cord.

FIG. 18.



The course of the long sensory tracts is pretty well known, and is well illustrated in the figures here given (Figs. 17 and 18). When a single nerve root is injured or destroyed, the area of degeneration at its point of entrance into the cord is quite extensive, but at higher levels in the cord the area of degeneration grows smaller, and at the junction of the cord with the medulla it is quite limited in extent. This is well illustrated in Figs. 19 to 21, which show the area of degeneration in a case of unilateral tumor involving the second and third lumbar nerve roots.[1] It will be seen that at the second lumbar segment the entire nerve-root zone in the column of Burdach is degenerated. In the mid-dorsal region this degeneration is limited to a small strand in the column of Goll, and in the cervical region to a small strand also in the column of Goll and near to the median line. This case confirms the results of physiological experiments in which the posterior roots have been divided in monkeys.[2] Such a case demonstrates that of the large number of nerve fibres entering in any one posterior nerve root, but a few extend all the way up to the medulla. If a series of cases of transverse lesion of the cord is studied, the transverse lesion in different cases be-

[1] Case reported by Prof. Wm. Osler: *Journ. Men. and Ner. Disease*, 1889, p. 499.

[2] *Beiträge zur Anatomie des Centralnervensystems insbesondere des Ruckenmarkes*, von Prof. Dr. J. Singer und Dr. E. Munzer in Prag, Wien, 1890.

Diagram showing the course of long sensory columns in the spinal cord: *S*, sensory nerve roots whose fibres enter the root zone and the gray matter. On the right side of the diagram these fibres terminate about the cells of the column of Clark (*C*), whence fibres pass into the right direct cerebellar column (*DC*), and thus upward to the cerebellum. Sensory fibres also terminate about the intrinsic cells of the cord (*e*), whence fibres cross to the opposite side and ascend in the column of Gowers (*G*). On the left side of the diagram sensory fibres are seen to terminate around intrinsic cells of the gray matter (*X*), whence fibres cross over to the opposite side of the cord and ascend in the anterolateral column (*AL*).

ing situated at different levels, it is found that the ascending degeneration in these long fibres within the posterior columns of the cord varies in extent in different cases. The higher the transverse lesion the larger the area of degeneration produced. This is demonstrated in Figs. 22

FIG. 19.

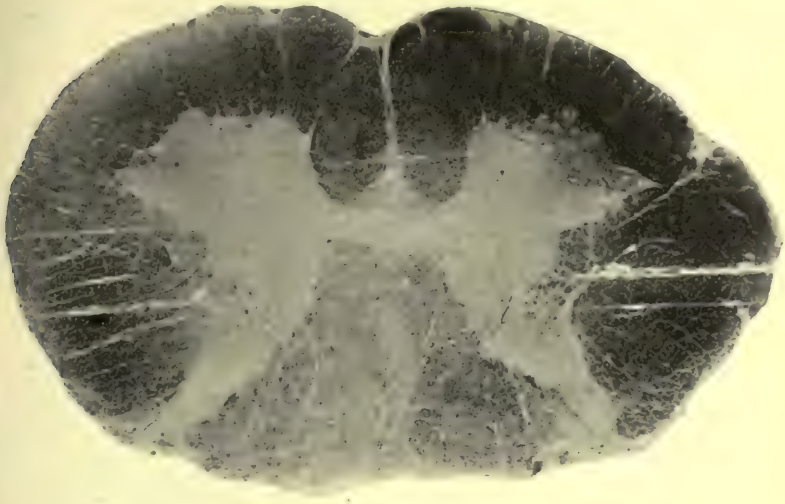


FIG. 20.

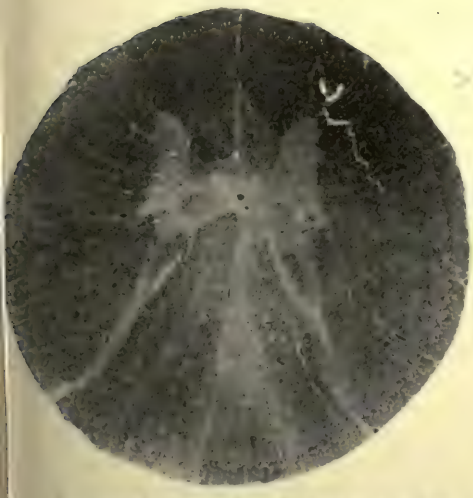
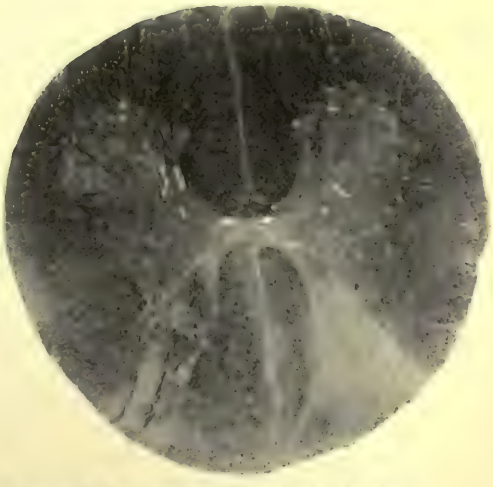


FIG. 21.



Sections of the spinal cord at the cervical (Fig. 19), dorsal (Fig. 20), lumbar (Fig. 21) levels, showing ascending degeneration, unilateral, in the posterior column after a gumma involving the second and third lumbar nerve roots. The relative extent of the degenerated fibres at the different levels is shown in the sections.

and 23, showing the area of ascending degeneration following a lesion of the lower lumbar region (Fig. 22), as contrasted with the area of degeneration following one in the upper dorsal region (Fig. 23). A study of such cases has made it possible to determine the relative position in

the columns of Goll and Burdach occupied by the long sensory fibres coming up from the various segments below. This is shown in Fig. 17. While the lesion in the posterior root zone causes an ascending degeneration in the posterior columns only, it has been found that a transverse lesion of the cord causes an ascending degeneration in the direct cerebellar column, the antero-lateral ascending tract, or column of Gowers, and many fibres of shorter or longer extent in the column of Burdach and in the antero-lateral columns. As degeneration only occurs in a fibre which is cut off from its neuron, it is evident that the neurons of

FIG. 22.

Ascending degeneration in the mid-dorsal region after transverse myelitis at the first lumbar segment. The degeneration affects the posterior median columns, together with the marginal zone at the base of the posterior commissure, also the direct cerebellar columns, the columns of Gowers, and many fibres in the antero-lateral columns.

which these degenerated fibres are branches lie in the cord itself and not in the posterior spinal ganglia. It has been stated already that the majority of the fibres entering a posterior root zone are short fibres. These terminate in fine brushes about the cells lying in the posterior gray matter and median gray matter of the spinal cord. Numerous neurons lie in these regions of the gray matter, and these neurons send out their axons into the lateral columns of the cord, where they turn upward toward the medulla, forming the long and short sensory columns whose existence is proven by the study of degenerations. Figs. 17 and 18 show the situation of these cells in the posterior and median gray matter of the cord with their axons passing into the various columns.

In Fig. 17 it will be noticed that some cells send their fibres into the columns of the cord on the side on which they lie. These are termed tautomere neurons. In Fig. 18 it will be seen that some cells send their fibres across the median line into the opposite columns of the cord. These are termed *heteromere neurons*. In both figures the numerous fibres from the sensory nerves entering the gray matter and terminating around these neurons are seen. It is thus evident that the course of the sensory impulses coming in through the short sensory nerve fibres is very complex. And it is evident that while

FIG. 23.

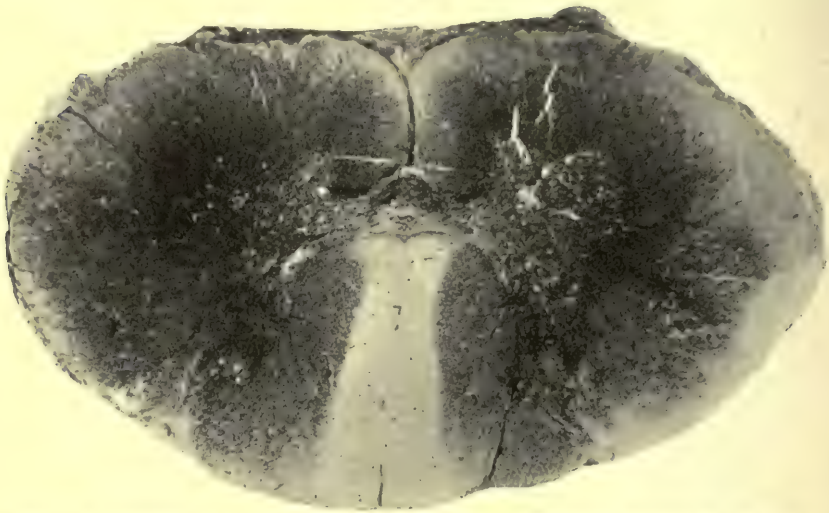
Ascending degeneration at the first dorsal segment after transverse lesion of the fifth dorsal segment. The ascending degeneration affects all of the posterior columns excepting the root zone of the column of Burdach. Also the direct cerebellar columns and columns of Gowers, and many fibres in the antero-lateral columns. A few degenerated fibres are seen around the margin of the anterior median column.

some sensory impulses pass upward on the same side on which they enter, many sensory impulses are sent across the cord and pass up on the opposite side.

It has been already stated that sensations entering the cord are of various kinds. It is not yet possible to assign a special path to each of these various sensations, but it seems probable that the sensations of muscular sense pass upward in the long fibres of the posterior columns and in the direct cerebellar tract of the same side upon which they enter; that the sensations of temperature and of pain pass in the short fibres to the central gray matter of the side on which they enter, are then taken up by new neurons which transmit them across the cord and upward in the antero-lateral tract; that the tactile impulses enter the posterior columns and also the gray matter of the cord, and are taken up by neurons, some of which send their fibres into the columns

of the same side; but the majority send their fibres across the median line into the antero-lateral columns of the opposite side (Fig. 18, *AL*). It can be positively stated that a condition of ataxia implies a lesion of the posterior column of the cord; that a condition of analgesia and thermo-analgesia implies a lesion of the central gray matter and of the ascending antero-lateral fibres; and that a condition of tactile anæsthesia implies widespread degeneration in the antero-lateral and posterior columns of the cord.

FIG. 24.



Ascending degeneration at the fifth cervical segment after transverse myelitis at the eighth dorsal segment.

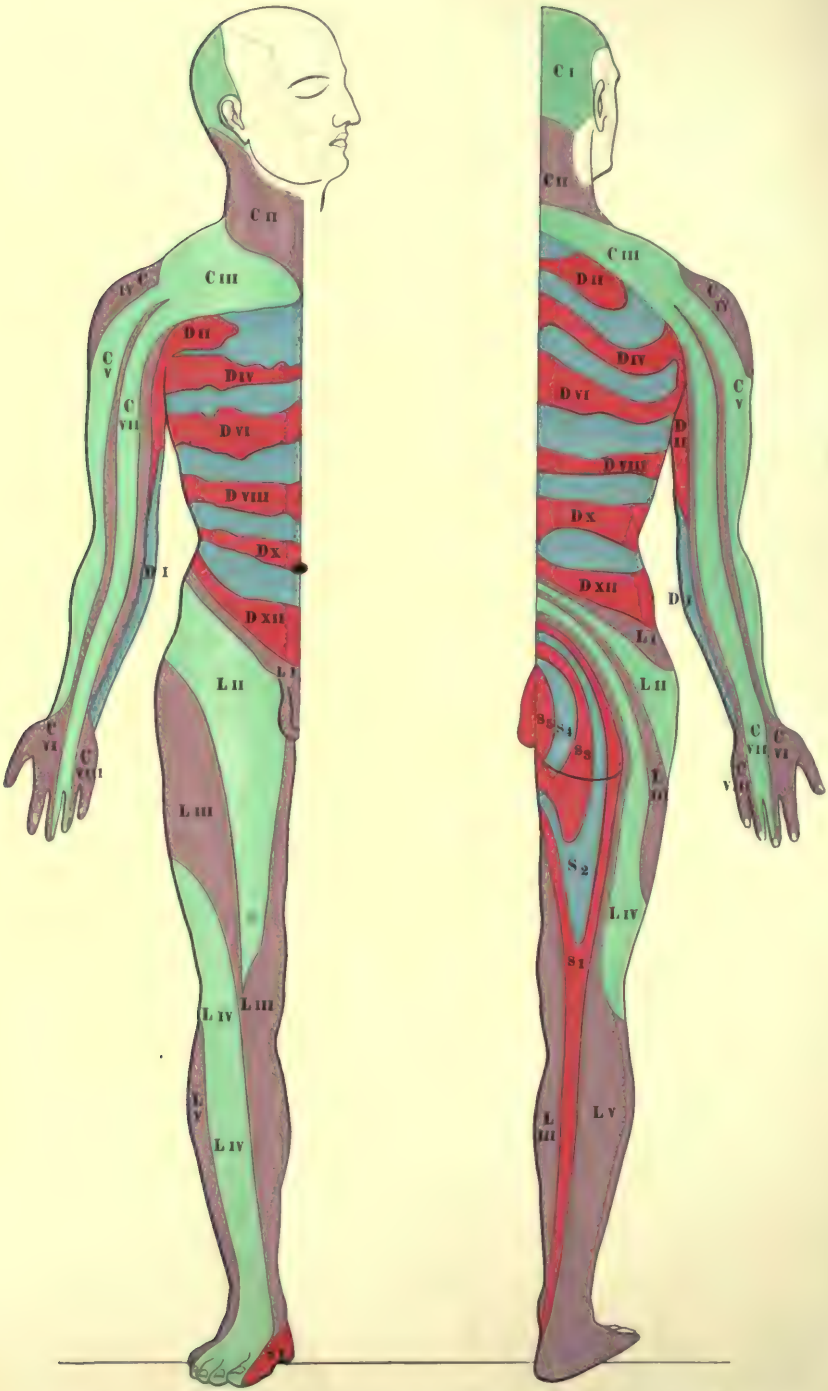
In a unilateral lesion of the cord it is usually found that there are tactile anæsthesia, analgesia, thermo-analgesia in the side of the body opposite to the lesion, together with some hyperæsthesia of all these sensations in the same side of the body as the lesion; and it is from this clinical fact that the conclusion is reached that sensory impulses pass across the cord and ascend in the columns of the opposite side.

In syringomyelia, in which the central gray matter of the cord alone is affected, there is a loss of temperature and pain-sense only. This occurs on the side of the lesion. It has been supposed until recently that these sensations were transmitted upward in the column of Gowers, but recent cases prove that this column passes to the cerebellum.

In locomotor ataxia, in which the lesion is limited to the posterior root zone, all the sensations are more or less impaired, and there is secondary degeneration ascending in the posterior columns only. In general myelitis all the sensory tracts are implicated, and here too all forms of sensation are impaired.

In transverse lesions of the cord at any segment it is evident that the impulses reaching that segment from its own pair of nerves, and the impulses passing through that segment to and from the segments below it, will be cut off; hence after transverse lesions there is a condition of total anæsthesia in the body below the segment which is de-

PLATE I.



Areas of Anæsthesia upon the Body after Lesions in the Various Segments of the Spinal Cord.

The segments of the cord are numbered: C I to VIII, D I to XII, L I to V, S 1 to 5, and these numbers are placed on the region of the skin supplied by the sensory nerves of the corresponding segment.

stroyed. If, therefore, a series of cases is brought together of lesions of the cord in every segment from the last sacral up to the upper cervical, and if the area of anæsthesia in the body in each of these cases is accurately determined, it is evident that it will be possible to ascertain the exact region of the skin related to the individual segments of the spinal cord. In Plate I. these areas are carefully laid down. It is evident, therefore, that in any case of spinal-cord disease it is necessary to test the sensations and to compare the area of anæsthesia with the diagram here given, and thus to determine the level of the spinal cord

FIG. 25.

The lesion in the posterior columns at the first dorsal region in a case of locomotor ataxia.

affected. It is to be remembered, however, that the skin of the body is plentifully supplied with sensory nerves which anastomose freely at their terminations, and the researches of Sherrington have demonstrated that each part of the skin is supplied with sensory nerves from two adjacent segments of the cord; hence a condition of anæsthesia in the skin indicates a suspension of function of two segments of the cord at least, for if one segment alone were affected, the segments above and below it would be capable of supplying the skin with sensation. This conclusion, drawn by Sherrington from physiological experience, I have confirmed in a case of spinal disease in which it became necessary to divide completely the posterior nerve roots at the sixth dorsal level. This division did not produce any anæsthesia around the body, because the fifth and seventh nerves supplied the skin of the trunk in the domain of the sixth nerve sufficiently to prevent anæsthesia. If, however, two adjacent nerves are divided, a zone of anæsthesia is produced. The overlapping, therefore, of adjacent sensory areas is not to be forgotten in determining the level of the segment affected. Thus if in a case the areas of anæsthesia on the body correspond to the section shown

in the diagram as belonging to the second, third, fourth, and fifth sacral segments of the cord, the lesion of the cord undoubtedly involves the first sacral segment also, but is certainly not any higher.

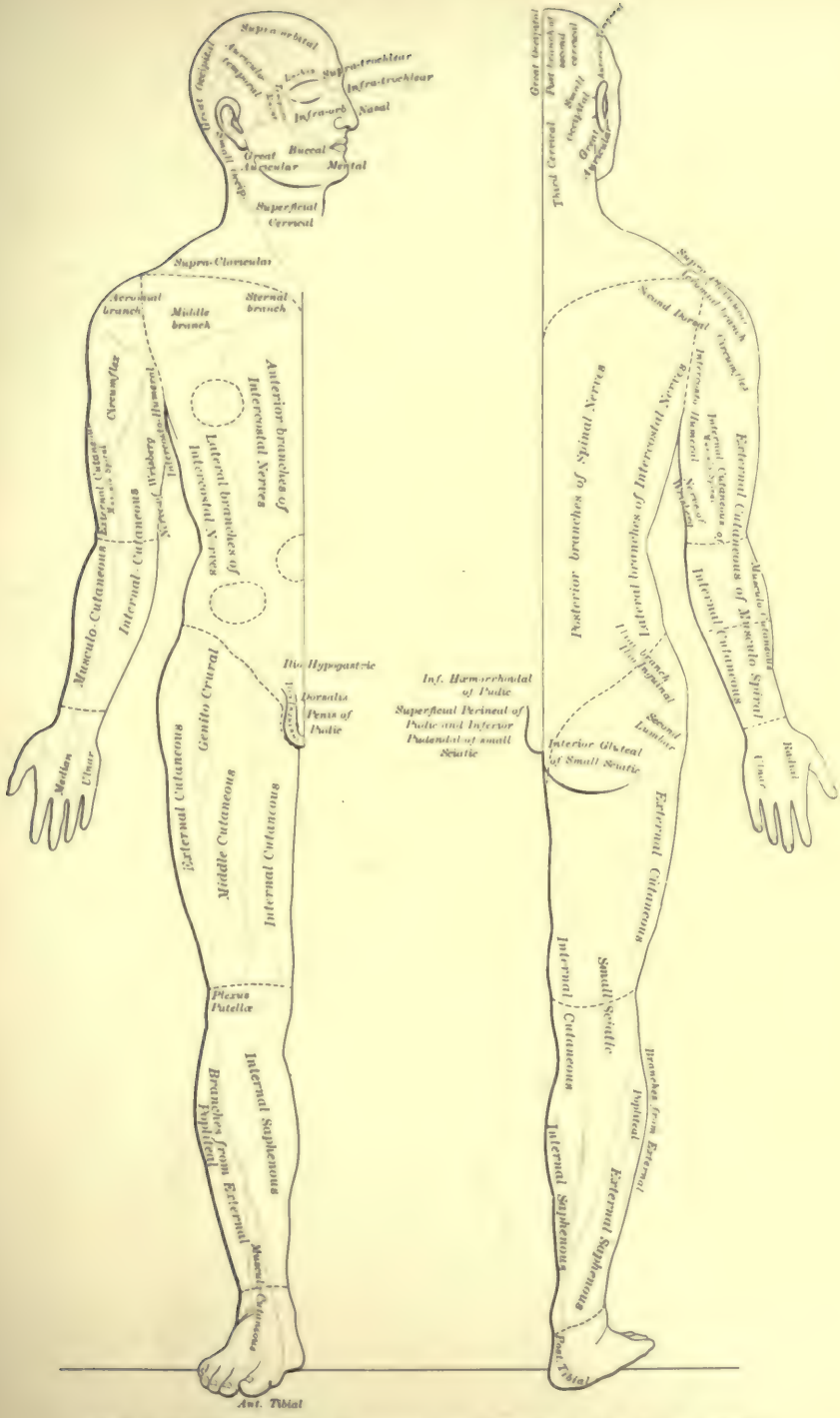
The determination of the area of anæsthesia is of particular importance in cases of compression of the cord by tumor or by dislocated or fractured vertebræ, as it is the most positive indication of the exact level of the cord which is invaded by disease. Thus in the cases in which tumors have been successfully removed from the cord the level of the anæsthesia has been the guiding symptom to the surgeon. In such cases the normal anatomical relation between the segments of the spinal cord and the vertebræ is not to be forgotten.

Small areas of anæsthesia in the body corresponding in distribution to the diagram, and due to small localized foci of disease in the spinal cord, are found chiefly in syringomyelia, in hemorrhages in the cord, in small areas of softening in the cord, or in tumor of the cord. These lesions destroy the terminal filaments of two or three sensory nerves, and do not invade the long tracts coming from the parts below the level of the lesion; hence localized anæsthesia is indicative of a small limited lesion, not of general transverse diseases. Such small limited lesions are not very common. In all cases the anæsthetic area fades gradually into the area of normal sensibility. In hysteria the line of demarkation between sensitive and insensitive skin is much sharper.

The determination of the exact area of anæsthesia in any case is of great service also in differentiating lesions of the spinal cord—first, from lesions of the nerve trunks; secondly, from lesions of the cauda equina; thirdly, from hysteria; fourthly, from multiple neuritis. In lesions of the nerve trunks the distribution of the anæsthesia is different from its distribution in the spinal-cord affections. This will be evident if the areas in Plate II., showing the distribution of the nerves of the skin, are compared with the areas in Plate I., showing the distribution of the nerves from the segments.

Fractures of the lower lumbar vertebræ or of the sacrum often produce pressure upon the cauda equina. It has been found that when such pressure is slight, it is the nerves lying innermost within the cauda which suffer most, and the greater the compression the greater the number of nerves which suffer. The distribution of the anæsthesia in caudal lesions resembles closely the distribution of anæsthesia in lesions of segments of the cord, and from a study of the anæsthesia alone it is impossible to differentiate absolutely a caudal lesion from a cord lesion. A diagnosis may, however, be made—first, from a study of the surgical indications, chiefly of the nature of deformity, the relation of the vertebræ to the segments of the cord being remembered. The spinal cord ends at the first lumbar vertebra; hence any fracture below that level necessarily compresses the cauda equina and does not destroy the spinal cord. Secondly, a diagnosis may be made from a study of the paralysis which accompanies the anæsthesia. This paralysis is very slight in lesions of the sacral segments of the cord. Thus when a lesion is at or below the third sacral segment, the paralysis is confined to the peronei muscles. When the first sacral segment is also involved, the paralysis affects the anterior and posterior tibial muscles, and it is only when the entire lumbar region of the cord is destroyed that movements of the

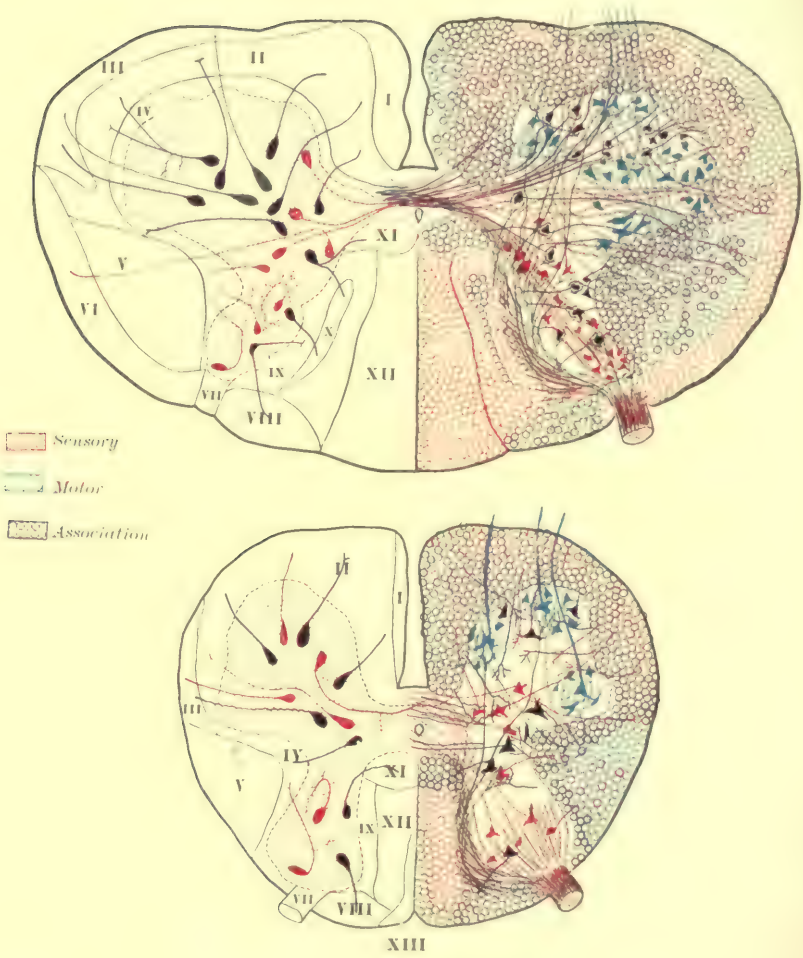
PLATE II.



The Distribution of Sensory Nerves in the Skin.
(After Flower.)

The areas of the skin supplied by the cutaneous nerves are shown in finely dotted outline. The circles on the trunk show areas occasionally anæsthetic in hysteria. The lines across the limbs at ankle, knee and thigh, wrist, elbow and shoulder show the upper limits of anæsthesia in multiple neuritis of varying degrees of severity.

PLATE III.

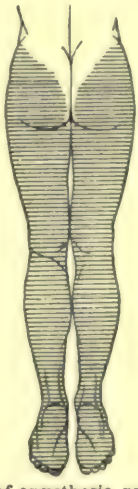


The Cervical and Sacral Enlargements of the Spinal Cord in Cross Section—Showing the Various Neurons in the Gray Matter, the Direction of their Axons, and the Varieties of Fibres in the Different Columns of the Cord. Blue, Motor-; Red, Sensory-; Purple, Association- Neurons and Axons.

hip-joint will be affected. In cauda-equina lesions, on the other hand, pressure upon the nerve roots is often sufficient to produce widespread paralysis when sensation is but slightly affected. Thus in the lesion of the spinal cord the distribution of the paralysis will correspond to the segment of the cord invaded by disease, and will correspond to the distribution of the anæsthesia produced by a lesion of that segment, while in lesions of the cauda equina the distribution of the paralysis may be much more extensive than that indicated by the distribution of the anæsthesia.

The distribution of the anæsthesia is also of much service in differentiating hysterical paralysis from organic disease of the spinal cord. Charcot pointed out the fact that in hysterical paraplegia the anæsthesia never involves the genital organs or the perineum and the anus. Paralysis of the bladder and rectum is a very rare thing, and if it occur is of the nature of retention of urine rather than of incontinence. Fig. 26 shows the area of anæsthesia commonly observed in hysterical cases, and it will be noticed that its distribution is very different in outline from that in Plate I. It is also to be remembered that in hysterical paraplegia there is no reaction of degeneration in the muscles, and there is no loss of tendon reflexes.

FIG. 26.



Area of anæsthesia present in hysterical paraplegia, anus and genitals escaping.

In many cases of traumatic neurosis or of irritation of the spine following injuries, and in the so-called spinal concussion associated with anæsthesia, it will be found that the distribution of the anæsthesia corresponds to the hysterical rather than to the organic type.

In multiple neuritis the region of anæsthesia assumes a stocking- or glove-shaped area on both limbs symmetrically, and does not extend to the trunk.

It is important to be able to locate the lesion accurately in spinal-cord disease, no matter whether the lesion thus determined corresponds to well-known forms of spinal disease or not. It has recently been shown[1] that vascular diseases of the spinal cord, hemorrhages into the cord of small extent, or long perforating hemorrhages in the cord of small lateral extent, or small areas of softening of the cord due to thrombosis of diseased spinal arteries or their branches, are more common than was supposed. The diagnosis of these conditions is only possible by an accurate study of symptoms, and such a study may indicate a local lesion suspending the function of a small area at any level.

Ataxia.—Ataxia is a symptom of spinal-cord disease due to an interference with the muscular sense impressions which pass into the cord through the posterior nerve roots and root zone (Plate III). Both the automatic and voluntary movements of the body are guided by the impressions received through muscular sense, and if those impressions are lacking, the movements become awkward. Such movements can be

[1] R. A. Williamson: *Manchester Medical Chronicle*, 1895.

guided by the eye, and hence an ataxic will always perform a motion more deftly if he watches the limb which is moved, but deprive him of vision by closing his eyes and the symptom of ataxia is readily developed. The lesions of the spinal cord which produce ataxia are those which destroy the fibres in the posterior external column or column of Burdach, through which the fibres pass which convey the impressions of muscular sense. As has been already stated, these fibres pass upward and downward in the column of Burdach, sending their collaterals into the posterior gray matter of the cord at different levels, so that the impressions coming in through a single nerve are conveyed to many segments of the cord. In addition, there are the long tracts passing to the medulla, already described in the column of Goll. When one thinks of the very numerous and complex movements which are involved in such a simple act as lifting a glass of water to the lips, involving almost the entire body, made in order to preserve its balance, one realizes that to properly guide any movement, however simple, a very extensive action of a large number of muscles is required. This action is guided automatically by the muscular sense. It is only when the muscular sense is interfered with that one realizes its extensive use and its constant function. The disease in which ataxia is most evident is posterior sclerosis, but any lesion affecting the same area of the cord will produce ataxia. Thus multiple sclerosis, or tumors of the meninges on the posterior surface, or tumors within the cord, or syringomyelia when it involves the posterior columns, or a general myelitis, are capable of producing this symptom. The ataxic gait has already been described.

Pain.—Pain is a symptom of considerable importance in spinal-cord disease. It may be felt in the spine itself; that is, in the back and deeper structures, under which circumstances, as a rule, there is a more or less extensive affection of the nerve roots or of the meninges, but not of the spinal cord. Severe pain in the back and spinal ligaments is not at all uncommon in functional affections, such as traumatic hysteria and traumatic neurasthenia; in nervous prostration; in hysteria. It is sometimes present in affections of the viscera, under which circumstance the pain is really a "referred pain," referred to the back when the actual irritation comes from the sympathetic nervous system connected with the viscus diseased. In organic affections of the bones, ligaments, and nerve roots spinal pain is also a frequent symptom.

When pain is produced by disease of the spinal cord itself, it is due to an irritation or injury of the posterior nerve roots at their entrance or to an irritation of the sensory tracts passing upward through the spinal cord. Under these circumstances the pain is not referred to the back, but is felt in the part of the body from which the irritated nerve root or sensory tract has come. Thus in locomotor ataxia the sharp shooting pains are referred to the limbs rather than to the back, and as the disease almost uniformly begins in the second and third lumbar segments of the cord, these pains are usually referred to the anterior surface of the thighs, and as the disease advances downward to the fourth and fifth lumbar segments, the pain is felt in the feet; as it advances upward into the dorsal region, the pain is felt about the body. When the process has ascended to the lower cervical region, pains begin

to be felt in the axilla, on the inner side of the arms, and in the little fingers, and finally, as the upper segments of the cervical region are invaded by the sclerotic process, the entire arms and shoulders become the seat of pain. The location of pain, therefore, in any case of disease of the cord is an indication of the level of the disease, and comparison of the distribution of the pain in any one case with the diagram (Plate I.) will enable one to locate the affection.

In syringomyelia, in which the terminal filaments of the pain-sense nerves within the gray matter are destroyed, the analgesia which finally develops is usually preceded by a stage of pain, and here too, as in locomotor ataxia, the pain is referred to the periphery corresponding to the segments of the cord involved.

Pain referred to the extremities is also a symptom in injuries of the cord, in hemorrhages within the cord, or in crushing of the cord such as follows dislocation or fracture of the spine. In these cases the pain is referred to the periphery, but is less exactly referred than in locomotor ataxia, for the injury affects all the sensory tracts coming from the parts below the site of the lesion, and these are damaged to a greater or less extent; consequently, the reference of the pain is more widespread. Thus in a hemorrhage in the lower cervical region intense pain may be felt in the entire body below the level of the arms. In these cases any movement of the body causing an increased mechanical pressure upon the cord may be attended by sharp shooting pains felt in any part of the body below the lesion. Pain is an early symptom in tumors of the cord, and in these cases it may be referred to the periphery from which the nerve root comes, which is primarily compressed at the site of the tumor. Thus in a tumor in the dorsal region the pain may be felt in the thorax, epigastrium, or abdomen. It may also be referred to the parts of the body below the tumor, because of the irritation of the sensory tracts passing through the cord at the site of the tumor.

There are some cases in which pain is referred to an anæsthetic portion of the body. Thus in caries of the spine, in localized meningitis, in injuries of the nerve roots, and in some cases of sclerosis of the cord the sensory nerves may be destroyed, so that no sensations can pass in over them, and hence the surface of the body may be anæsthetic; but at the same time the proximal ends of these destroyed nerves may be irritated by the disease, and hence painful sensations may constantly be sent inward to the cord. These, being received and transmitted upward, are referred to the anæsthetic surface, giving rise to the symptom called anæsthesia dolorosa. This condition is very rarely seen in diseases of the cord itself, but is very common in diseases of the vertebræ, especially in tumors (carcinoma, sarcoma) and in caries of the spine.

Vasomotor and Trophic Symptoms.—In the gray matter of the cord there lie certain cells which regulate the mechanism by which nutrition in the body is maintained. It seems probable that these mechanisms are set in activity by sensory impulses reaching the cord through the posterior nerves, because many posterior nerve or nerve-root lesions are attended by trophic disturbances—*e. g.* joint disease and perforating ulcer of the foot in locomotor ataxia. In locomotor ataxia the joint diseases are quite frequent, the ankle, knee, and hip being affected in

the order mentioned. In syringomyelia it is the elbow, wrist, fingers, and shoulder, in the order mentioned, which are most commonly affected. It is quite noticeable that such joint affections occur chiefly among the lower classes or in persons who are exposed to injury or to falls; hence it is concluded that their origin is traumatic, even a slight injury being sufficient to produce them. There is usually at first an effusion of fluid into the joint, subsequently a thickening of the ends of the bones. If such joints are treated by perfect rest in the early stage, the disease disappears. If, however, they are neglected, as they are very liable to be because of the absence of pain, the process goes on and increases until the joint is rendered useless. It is a very suggestive fact that these joint diseases appear only in those spinal affections in which the sense of pain is impaired, and this is another proof of the conservative influence of pain in securing rest or immobility in the diseased part.

The mechanisms started by these sensory impulses which control nutrition lie in the central gray matter of the cord, and to some extent in the anterior horn, for trophic disturbances, especially eruptions in the skin, necrosis of the fingers, atrophy of the muscles, fragility of the bone, occur in lesions limited to the gray matter of the central area or invading the anterior horn, as, for example, in syringomyelia and in anterior poliomyelitis. It is probable that these mechanisms are not the only ones which preside over nutrition, and that throughout the body and in the skin the nerve mechanism of the vessels is quite competent to regulate nufrition, provided it is not put to too great a strain. Thus bedsores and cystitis are in my opinion rarely due primarily to lesions of the spinal cord, and may usually be avoided by proper care. In the normal body continuous pressure upon any one part for a long time without a shifting of position does not occur even in sleep, and if a similar repeated change of posture is kept up in a case of spinal paralysis, bedsores will not appear. It is undoubtedly the continued pressure without change of position which produces such sores, and this is proven by the locality in which they uniformly appear—namely, over the sacrum, upon the trochanters, upon the heels, and upon spots subjected to greatest pressure when the patient lies long in one position. In cystitis it is the introduction of germs into the bladder by means of the catheter which is responsible for the disease, or else an over-distention and consequent inflammation of the bladder when the proper catheterization is neglected. While it is admitted, therefore, that the spinal cord has trophic functions in the sense that it regulates the degree of circulation and the rapidity of processes of nutrition, it is not believed that a lesion in the cord alone under proper care of the patient will result in lesions of the skin or mucous membranes.

The atrophy of the muscles which occurs in spinal paralysis appears to be too rapid to be accounted for by disuse, and the fragility of the bones which appears in syringomyelia can only be accounted for by a distinct trophic influence of the cord upon the bones.

In all conditions of destruction of the spinal cord the part of the body related to the part of the cord destroyed is found to present a bluish appearance, indicating an imperfect capillary circulation due to a lack of tone in the arteries, and consequently a cold condition due to

imperfect processes of nutrition going on in consequence of the venous stasis. This is particularly noticeable in the parts of the body below a transverse lesion of the cord, in syringomyelia and also in anterior poliomyelitis; hence it is evident that the spinal cord controls the vasomotor mechanism. Hence in any case where vasomotor or trophic symptoms appear in a patient the suspicion is aroused of a spinal-cord disease.

DISEASES OF THE MEMBRANES OF THE SPINAL CORD.

By CHRISTIAN A. HERTER, M. D.

ACUTE SPINAL MENINGITIS.

Synonyms.—Acute spinal leptomeningitis; Acute internal meningitis.

Definition.—Under Acute Meningitis are included the various acute inflammations or infections of pyogenic origin that involve the spinal pia mater or dura mater. When the inflammation involves chiefly or primarily the pia mater (acute spinal leptomeningitis), as it generally does, the process is diffuse and often extends to the internal surface of the dura. We have no reason to think that the symptoms observed when the internal surface of the dura is thus involved differ materially from the symptoms observed when the pia arachnoid is alone affected. The only case where an acute spinal meningitis is not internal is where the external surface of the dura is the seat of acute purulent inflammation (generally secondary to bone disease or injury). Here, again, the nervous symptoms alone cannot be clearly distinguished from those of acute spinal leptomeningitis, and it therefore seems unnecessary to attempt the creation of distinct clinical types for acute external spinal meningitis and acute internal spinal meningitis.

Etiology.—Acute spinal meningitis is probably always due to infection by pathogenic micro-organisms, for the most part such as are pyogenic and of the same character as those which cause acute cerebral meningitis (streptococcus pyogenes, staphylococcus aureus, pneumococcus, etc.). The condition occurs—

(1) In tuberculosis in connection with cerebral meningitis (this is the most common cause observed in general hospitals, but its symptoms are subordinate to the cerebral symptoms and are not usually very acute (see Meningeal Tuberculosis, Vol. I. p. 755).

(2) In specific cerebro-spinal meningitis (epidemic or endemic). This, again, is one of the most frequent causes of acute spinal meningitis (see Epidemic Cerebro-spinal Meningitis, Vol. I. p. 425).

(3) In the course of certain infectious diseases—pneumonia, scarlet fever, smallpox, typhoid fever, pyæmia, and septicæmia. These are rare causes of acute spinal meningitis. When the process occurs in the course of pneumonia it is associated with cerebral meningitis, and the inflammation does not often extend below the cervical region.

(4) From direct or indirect injury to the spine (wounds, fractures, etc.) which afford an avenue for the passage of infection to the meninges (as from operation on spina bifida, sacral bedsores, vertebral abscess,

etc.). In such cases the process is local at first, and then rapidly spreads.

(5) In the early period of active syphilis the pia of the cord may be the seat of an acute (more often subacute), non-suppurative inflammation. The cord is always implicated (see Syphilis of the Cord, Vol. I. p. 885). Very rarely an acute partial tubercular pachymeningitis is observed as the result of vertebral tuberculosis.

It is evident that if we exclude cases where the spinal lesion is associated with a preponderant cerebral lesion (which throws such cases into a different clinical category), and, further, exclude the cases of syphilitic origin, acute spinal meningitis is a very rare condition even in hospital practice.

Acute spinal meningitis is probably more common among men than among women, and is more frequent in the first than in the second half of life. Over-exertion, exposure to cold and wet, and traumatism appear sometimes to favor the operation of influences that cause acute spinal meningitis.

Pathology.—After a short initial stage of hyperæmia there is an exudation of fibrin and serum, and usually of pus, into the meshes of the pia and arachnoid. The membranes are rapidly covered with a semisolid or more fluid purulent exudate, which sticks them together in places and which renders the cerebro-spinal fluid turbid. When the process is tubercular the exudation is not purulent, but gelatinous, and not very abundant, and miliary tubercles can usually be seen studding the pia arachnoid and inner surface of the dura. When the process extends from the cerebral meninges it may pass only a few inches down the spinal meninges, but, as a rule, the spinal membranes are involved throughout their entire extent. In general the exudate is most abundant in the posterior circumference of the membranes, owing probably to the influence of gravitation. The exudation extends about the nerve roots and sometimes into their substance. There is usually some infiltration of inflammatory products into the periphery of the cord, and this is especially marked in syphilitic meningitis. The inflammation extends to the cord along pial arteries; hence when the arachnoid is chiefly involved and the pia but little, the cord itself escapes damage.

In acute external meningitis, which generally results from the extension of contiguous disease (especially of the vertebræ), but which is said sometimes to be primary, the space between the dura and bone is filled with pus which may extend as high as the fibrous dural attachment to the upper cervical vertebræ. The pus may be confined to the posterior aspect of the dura. It may escape between the arches of the vertebræ into the muscles of the back. In acute leptomeningitis from septicæmia the cord may be partially destroyed by offensive pus. Purulent meningitis is sometimes associated with a rapidly extending central myelitis, due to the same micro-organism (Holt and Van Gieson).

Symptoms.—In most cases of acute internal meningitis the cerebral symptoms preponderate, so that the distinctively spinal symptoms are masked.

In the rare instances where the lesion is wholly spinal the following symptoms are observed:

(1) Onset with rigors and considerable but irregular temperature;
(2) Pain in the back, exaggerated by motion;
(3) Severe radiating pains in upper and lower extremities;
(4) Rigidity of the muscles of the back, perhaps with opisthotonos;
(5) Tonic spasm of the extremities and muscles of the abdomen and chest;
(6) Occasional clonic spasm;
(7) Marked hyperæsthesia, usually general;
(8) Greatly exaggerated skin and deep reflexes, including ankle clonus;
(9) Retention of urine; and
(10) In many cases the inability to extend the legs in the sitting posture, owing to flexor spasm (Kernig's symptom).

If the patient survives the acute stage, the irritative symptoms may give way to paralytic phenomena, partial paraplegia, partial anæsthesia in places, incontinence of urine and fæces, loss of knee jerks, and sometimes irregular breathing and heart action and pupillary symptoms. Where the meningitis is purulent, irritative symptoms are sometimes singularly slight, perhaps because the nerve roots are not infiltrated. In cases of acute external meningitis the symptoms are more apt to be anomalous and unusual than in acute internal meningitis. In very severe cases death may occur in two or three days. In most cases the duration of the symptoms is from one to two weeks. In cases that recover considerable rigidity and weakness may last for months or years.

Diagnosis.—In most instances the pain in the back, the rigidity of the muscles of the neck and back, and the hyperæsthesia occurring with an acute febrile onset make the nature of the case clear. But it should be remembered that occasionally the purulent form runs an almost latent course, and that here error is sometimes unavoidable.

Prognosis.—The outlook is grave in every case, and is particularly bad in the so-called spontaneous cases. The danger to life is great in proportion to the severity and acuteness of the symptoms and the height of the fever. The prognosis is somewhat less grave in traumatic than in other cases, and in persons in middle life than in those in childhood or old age.

Treatment.—The first indication is to secure absolute rest and quiet in bed. It is better for the patient to lie on his side than on the back if he can be made comfortable. The bowels should be kept gently open. If there are much pain and restlessness, these symptoms should be kept well under control by means of ammonol, phenacetine, or even morphine. Movement may be distinctly harmful. The room should be kept dark. At the onset it is good practice to apply dry or wet cups (according to the vitality of the patient) along the spine. Then, if it does not interfere with comfort, ice-bags may be applied to the spine.

The wet pack for one or two hours daily may lessen the rigidity, and will almost certainly help to quiet and comfort the patient. It is thought by some competent observers (notably Gowers) that mercurial inunctions sometimes exert a favorable influence. In the late stage of the disease the contractures and pains should be combated by means of

hot baths or hot douches and by applications of the actual cautery to the spine. In acute external meningitis with bone disease the pus should be given free exit.

CHRONIC SPINAL MENINGITIS.

Synonyms.—Chronic internal pachymeningitis; Chronic lepto-meningitis.

Definition.—Under Chronic Spinal Meningitis will be included all inflammatory processes affecting the pia mater or the internal surface of the dura mater. Chronic inflammatory affections of the external surface of the spinal dura mater will not be considered here. It may be said of them, in passing, that they almost invariably result from adjacent bone disease, which is generally tubercular, but are occasionally secondary to syphilitic disease of the vertebræ (see Syphilis of the Spinal Cord, Vol. I. p. 885).

The great majority of all cases of chronic spinal meningitis are due to syphilis, and such cases are discussed in a separate section. An important though very rare form of chronic spinal meningitis is that known as hypertrophic pachymeningitis. This form possesses many characteristic clinical features, and is also separately discussed. Thus what is said in this section does not relate particularly to the forms just mentioned.

Etiology and Pathology.—As chronic internal meningitis is rarely fatal, our knowledge of its pathology is exceedingly limited. It was formerly inferred that chronic symptoms (especially spasm) similar to the acute symptoms known to depend on acute meningitis were due to chronic meningitis. It is now clear that these spastic symptoms are due to damage to the spinal cord itself, and there is reason to think that many cases of "chronic meningitis" were really cases of lateral sclerosis. All forms of chronic internal meningitis are most common in adult life, and affect men oftener than women. In the rare form which is general and primary it is thought that general debility, prolonged over-exertion, and exposure to cold, all favor its development. A chronic internal meningitis probably results sometimes from severe concussion of the spine. The lesion also may follow the chronic external meningitis that results from trauma, and, on the other hand, may perhaps be secondary to chronic inflammation of the substance of the spinal cord. The lesion is then usually limited in extent. In general it may be said that a local chronic meningitis may follow any chronic disease of the bones (caries, sarcoma, carcinoma) or of the membranes (tumors) or of the substance of the cord itself. Alcoholism is an important cause of general chronic inflammation both of the pia arachnoid and of the periphery of the cord. Of course chronic internal meningitis may follow acute internal meningitis from any cause.

Where the process is only slightly or moderately developed the pia arachnoid is merely opaque and slightly thickened, the internal surface of the dura is slightly granular, and the cerebro-spinal fluid is turbid

and increased in quantity. When the process is more advanced the pia arachnoid and dura are connected by the formation of an unevenly thick layer of connective or granulation tissue. The pia may largely lose its cellular structure and become a thick, homogeneous layer. The pial vessels are usually greatly thickened. The nerve roots are usually compressed, and partially or wholly atrophied where they pass through the inflamed membranes. These nerve-root changes are especially marked where the roots pass through the thickened, unyielding dura. The damage to the spinal cord varies much in different cases, but there is always some extension of fibrous tissue from the pia into the periphery of the cord, with destruction of nerve elements, and some evidences of chronic inflammation or even spots of old softening. This peripheral sclerosis is often very pronounced in a small extent of the cord, especially in cases of traumatic origin. Ascending and descending degenerations may result from it.

In the rare condition described as hemorrhagic pachymeningitis or hæmatoma of the spinal dura mater the inner surface of the dura is thickened by layers of fibrinous exudate rich in new bloodvessels, which in places have ruptured and produced extravasations which may be encysted. The lesion is similar to, and usually associated with, the commoner condition of the cerebral dura. It occurs especially in the insane, but also as the result of trauma and of alcoholism. A simple chronic pachymeningitis interna, with the formation of new connective tissue containing brain-sand, is not very rare. If the brain-sand is abundant, the condition is described as a psammoma. These changes only rarely give rise to symptoms. This is true also of the white fibroid plates occasionally found in the arachnoid.

Symptoms.—The symptoms of chronic meningitis, like those of acute meningitis, depend on three factors: irritation of the nerve roots, partial destruction of the nerve roots, and partial destruction of the periphery of the cord itself. In acute meningitis the spasm caused by irritation of the anterior nerve roots is the dominant symptom; in chronic meningitis spasm is much less prominent, and may indeed be subordinate to some other symptom, such as pain or paralysis. In different cases the symptoms differ considerably, both in nature and in distribution, according as irritation or destruction of nerve elements preponderate and according to the level and extent of the lesion. Commonly the following irritative symptoms are observed: (1) pain in the back or discomfort, increased by movement; (2) stiffness of the muscles of the back (retraction of the head if the lesion involves the cervical region); (3) pain on pressure of the vertebral spines; (4) radiating pains, sharp, darting, and paroxysmal, in the neck, arms, trunk, or legs, according to the level of the nerve-root irritation, with sometimes a girdle sensation; (5) areas of hyperæsthesia; and (6) slight tremor or twitching or (occasionally) clonic spasm in one or more extremities. In some cases only the first and second of the symptoms enumerated are observed. In most cases, after weeks or months, in which the above symptoms vary in severity, slight paralytic symptoms appear and partially or wholly replace the irritative nerve-root symptoms. In certain areas, sometimes areas that have been the seat of hyperæsthesia, loss of sensibility to pain or touch, or both, makes its appearance. The corre-

sponding muscles become weak, and may undergo moderate or even considerable wasting, with the R. D. (Reaction of Degeneration). The weakness and wasting are often very irregularly distributed, owing to the uneven damage done to the anterior nerve roots. Eventually cord symptoms make their appearance, the legs grow weak, the knee jerks are increased, inco-ordination of the legs may appear, and control over the sphincters becomes imperfect. When the lesion is advanced in the lumbar cord or in the cauda equina, the paralysis is associated with atrophy, R. D., loss of reflex action, and sphincteric paralysis. In traumatic cases of limited extent in the dorsal region the chief symptoms may be slight spastic paraplegia and local pain and rigidity. There is nothing distinctive about the symptoms in hemorrhagic pachymeningitis; there are simply the indications of a slight chronic meningitis.

Diagnosis.—The diagnosis of chronic internal meningitis is based chiefly on the gradual development of symptoms due to irritation and partial destruction of the motor and sensory nerve roots. Owing to the fact that chronic meningitis may be associated with such varied processes in the structures about or within the membranes, it is not always easy to say whether the meningitis is primary or secondary. It is well to remember that the secondary chronic meningitis is fairly common, but that the primary condition is very rare. It is rare as an isolated affection, even after injury to the spine. Tubercular and malignant disease of the spine produce many of their symptoms by causing local chronic meningitis. The absence of indications of bone disease, in combination with the meningeal symptoms, serves to make the diagnosis.

Prognosis.—Owing to the differences in different cases of chronic spinal meningitis as regards the nature, degree, and location of the lesions, it is difficult to state any prognostic rules of general application. The outlook has rather to be formulated from an accurate estimate of the pathological condition in every case individually. Where there is evidence of considerable inflammation of the cord the danger to life may be considerable, especially if the lumbar centres are involved. As a rule, the danger to life is chiefly that which arises from the impaired general health which comes with prolonged disability. In some cases the nerve-root symptoms, pain and spasm, give rise to much discomfort and inconvenience. The neuralgic pains may indeed be so severe as to be almost unbearable, but this is rare. In many cases, even without other treatment than rest, both the irritative symptoms and those that depend on compression of nerve structures wear gradually away. Aside from syphilitic cases, which are not under consideration, the prognosis is best in traumatic cases where the lesion is of small vertical extent, the outlook here being improved by the possibility of successful surgical interference. Where the general health is impaired the prospect of improvement appears to be lessened by this fact.

Treatment.—In all severe cases, and even in those of moderate severity, absolute rest in bed is the first indication. It is probably advantageous for the patient to lie in the prone position for one or two hours daily. Counter-irritation is important. The Paquelin cautery should be applied lightly every second or third day to the skin on either side of the spine through the vertical extent of the lesion. It not

merely relieves pain, but serves to aid the absorption of recent inflammatory products. Hot douches to the spine also relieve pain and may temporarily diminish rigidity. They may be used alternately with the cautery if the latter is lightly applied. General hot baths may be of service. Sedatives for the relief of pain should be avoided, but this is not always possible. Cocaine is to be preferred to morphine, but should be cautiously used except in hopeless cases. The antipyretics may be tried, but are not likely to be of much service. Mercurial inunctions and iodide of potassium are thought to have some influence over non-syphilitic as well as syphilitic cases, but in the writer's experience have done little or no good. Massage should be employed where wasting or contractions appear. Galvanism is probably useful only in exercising wasted muscles. The general health must be carefully watched. In traumatic cases of limited vertical extent, but with severe symptoms (rigidity, pain, paralysis, etc.), the separation of the dura from the pia by surgical interference or the removal of extradural newly-formed fibrous tissue may lead to marked improvement as regards rigidity, pain, and power.

HYPERTROPHIC CERVICAL PACHYMENINGITIS.

DEFINITION.—A chronic hyperplastic inflammation of the dura mater, involving especially its inner layers, and of the pia arachnoid, leading to a great thickening of the dural membrane (¼ inch or more), with consequent irritation and gradual compression of the nerve roots and of the substance of the cord in the cervical region. The term "hypertrophic" is inappropriate for the designation of an inflammatory lesion, but is sanctioned by usage.

ETIOLOGY.—This is a very rare affection. It is the belief of the writer that it is far rarer than syringomyelia, many cases of which were probably diagnosticated as hypertrophic cervical pachymeningitis in former days. It is seen oftener in men than in women, and occurs especially between the ages of forty and fifty years. A few cases are as clearly referable to syphilis as is any syphilitic affection of the cord. The majority of cases are certainly not syphilitic in origin. It seems likely that local trauma and exposure to cold facilitate the progress of the disease, and it may be that trauma can initiate it.

PATHOLOGY.—The pathology of this condition is still the subject of discussion. According to one view (that of Charcot and of Joffroy, now sustained by Oppenheim), the process affects primarily the dura, and involves the pia and cord only secondarily, the morbid process affecting the cervical region only. In the light of more extended modern investigations (especially those of Adamkiewics, of Wieting, and of Köppen) it appears probable that the disease begins in the pia arachnoid of the cervical region as a small round-cell infiltration associated with arteritis; that this infiltration extends to the cord on the one hand and to the dura on the other; and that the enormous thickening of the dura in layers of new connective tissue is preceded by cell pro-

liferation in the dural endothelial layer which lies between the arachnoid and the dura proper. The increase of small round cells is due, in part at least, to the multiplication of the fixed connective-tissue cells of the membranes, and leads here, as elsewhere, to the formation, first, of granulation tissue, later of fibrous connective tissue. It further appears from recent studies that these changes are not confined to the cervical region, but in the case of the pia arachnoid may extend the entire length of the cord, and in the case of the dural thickening may extend to the dura adjacent to the medulla and pons. According to the earlier view, the spinal cord simply suffered mechanical compression; according to the modern conception, the cord changes are the result of inflammatory cell proliferation. In some cases the arteries have been the seat of extensive hyaline degeneration. It is not yet clear what special local conditions determine the remarkable hyperplastic thickening in the dura of the cervical region. But the fact should be noted that here the dura hugs the pia arachnoid rather closely. A thorough discussion of the pathology of this disease cannot be attempted here, and the reader is referred especially to the articles of Wieting[1] and of Köppen.[2]

Symptoms.—The earliest symptoms of the disease are due to the irritation of the nerve roots—pain between the shoulders, in the back of the neck and head, slight rigidity of the muscles of the back of the neck, tenderness of the cervical vertebræ to percussion, slight disturbances of sensibility, paræsthesias, and more or less symmetrical neuralgic pains, especially in the distribution of the ulnar and median nerves. The rigidity may extend to the shoulder and arm muscles, and there may be a coarse intention tremor. After a period of weeks or months this neuralgic period is followed by a stage in which paralytic symptoms gradually preponderate, owing to compression of the nerve roots. In the region supplied by the ulnar and median nerves (that is, in the parts corresponding to the lowest cervical and first dorsal roots) partial or complete anæsthesia develops, and paralysis, with muscular atrophy and the R. D., becomes pronounced. The paralysis and wasting of the intrinsic muscles of the hand and of the flexors of the fingers leads to a rather characteristic attitude of the hand in a good many cases; the unaffected muscles cause persistent over-extension of the wrist; the phalangeal joints are flexed, while the metacarpo-phalangeal joints are in extension. In some cases the long extensors are also paralyzed (Köppen). Hand in hand with these symptoms there develops paraplegic weakness from infiltration or compression of the cord. There is sometimes slight sensory loss below as well as at the level of the lesion, the pain sense especially being reduced. There may be incontinence of urine and of fæces. Occasionally there is staccato speech, not unlike that of multiple sclerosis, due apparently to embarrassed respiration. The pupillary reaction may be sluggish and the palpebral fissure may be at first wider, later narrower, than normal. The local symptoms are apt to be approximately symmetrical in distribution. It sometimes happens that the usual initial stage characterized by pain is wholly lacking, perhaps owing in such cases to the very gradual progress of the disease. It may also happen that, owing to the lesion being placed

[1] *Ziegler's Beiträge*, vol. 13, p. 411. [2] *Archiv für Psychiatrie*, Bd. 27, p. 918.

a little higher than usual, the shoulder muscles suffer chiefly, and not the intrinsic muscles of the hands. In certain cases the muscular atrophies have been very slight, doubtless because the anterior nerve roots were very little implicated. Severe intercostal neuralgia has occurred where the lesion has implicated the upper dorsal region of the cord.

Diagnosis.—In typical cases the diagnosis presents few difficulties. From most tumors of the cord the distinction can be made by the slower course and more symmetrical disposition of the symptoms, but it is possible that confusion may be unavoidable in rare instances. Caries of the spine may lead to symptoms that are hardly to be distinguished from those under consideration. The presence of marked bone tenderness and of deformity usually serves to make the diagnosis of caries.

Prognosis.—The progress of the disease is slow and extends over many years. In any stage of the disease progress may be indefinitely arrested. In rare cases recovery is said to have occurred (Charcot, Blyn, Remak). In every case the disease threatens life when well established. It is not likely that the outlook is better in syphilitic than in other cases.

Treatment.—Treatment has little influence on the course of the disease. The bowels should be kept open and the general nutrition well maintained. Local counter-irritation by means of compound tincture of iodine, and especially by the actual cautery, often relieves pain, and may possibly do something to retard the morbid process. Mixed treatment is indicated in syphilitic cases, and may do some good in other cases. Prolonged warm baths are to be recommended. It is said that the galvanic current has been serviceable in some cases (Remak, Oppenheim).

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Synonyms.—Hæmatorrachis; Meningeal spinal apoplexy.

Definition.—Under these titles are included cases of hemorrhage external to the dura (extrameningeal) and cases of hemorrhage within the dural sac (intrameningeal); of these two forms the extrameningeal is the more common. The intrameningeal hemorrhages are either subdural, between the dura and arachnoid, or subarachnoid, between the arachnoid and pia.

Etiology.—In a very large proportion of cases the hemorrhage is the result of injury. Blows or falls on the back and falls on the feet or buttocks are common causes. There may or may not be fracture or dislocation of the vertebræ (chorea, epilepsy, eclampsia, tetanus); usually there is not. Various convulsive diseases are liable to lead to spinal meningeal hemorrhage by their interference with the circulation within the spinal canal. In many of these cases the hemorrhage occurs during the last minutes of life, and does not give rise to symptoms. A variety of other causes are occasionally operative, among them the following:

prolonged severe muscular exercise; blood states in which there is a hemorrhagic tendency (purpura, acute specific diseases, rarely typhoid fever); the rupture of an aortic aneurysm into the spinal canal (the vertebræ having been eroded); the bursting of an aneurysm of the vertebral artery. Blood sometimes extends into the spinal membranes from a cerebral hemorrhage, especially one that is meningeal. This is seen particularly in the case of newly-born children. Rarely extravasations have occurred in the course of an intense spinal meningitis. Occasionally spinal hemorrhage occurs without discoverable cause. In general this condition is seen more often in males than in females. It may occur at any time of life, but spontaneous hemorrhage is not seen in infancy. Spinal meningeal hemorrhage is a rare disease as compared with hemorrhage into the cerebral meninges, but is considerably more common than extravasation into the substance of the spinal cord.

Pathological Anatomy.—In extradural hemorrhage the extradural plexus of veins is the source of the extravasation. The hemorrhage is much more often located in the cervical region than elsewhere. The amount of extravasated blood is usually not great, and is generally most abundant on the posterior aspect of the dura, owing to the influence of gravitation and the considerable space between the bone and the membrane. As a rule, the spinal cord is not much compressed. Occasionally a hemorrhage is so extensive as to reach over the dura almost throughout its length. The blood may then pass out of the spinal canal along the nerve roots.

It is important, but not always easy, to distinguish from hemorrhage the moderate accumulation of blood outside the dura which escapes on cutting spinal veins that have been greatly distended with blood owing to the influence of post-mortem gravitation.

In subdural hemorrhage the effused blood may extend from one level to another, and in some cases fill the entire cavity. When the hemorrhage is subarachnoid the pial vessels are commonly the source of the effusion. Usually the extravasation is small and surrounds the cord for a few inches only, but occasionally the blood fills the whole cavity. Even a moderate subarachnoid effusion may cause considerable compression of the cord. Very large intrameningeal hemorrhages are generally extensions from a cerebral meningeal hemorrhage. After a few days the irritation of the effused blood may set up slight inflammatory exudation in the membranes. Occasionally central hæmatomyelia coexists with hæmatorrachis.

Symptoms.—Occasionally a small meningeal hemorrhage is found in persons who have shown no definite symptoms of it. This occurs especially in cases where there have been convulsive symptoms, and it is likely that the extravasations have occurred during the convulsions immediately preceding death.

There is little difference between the symptoms of extra- and intrameningeal hemorrhage. In both varieties the symptoms are, broadly speaking, much like those of spinal meningitis, with the important exception that the onset is sudden and not acute or gradual.

The first symptom of spinal meningeal hemorrhage is usually severe and sudden pain in the back, corresponding in a general way with the position of the lesion. This is usually accompanied with shooting pains

along the course of the nerves and with hyperæsthesia, tingling, etc., in the limbs especially. These symptoms are due mainly to irritation of the posterior nerve roots.

Muscular spasm is an early symptom, and accompanies the sensory symptoms in most cases. The rigidity involves ordinarily the vertebral muscles, causing opisthotonos, and the limbs below the level of the hemorrhage are the seat of varying spasm, which may be clonic for a time. Rarely there are general convulsive movements. These symptoms are due to irritation of the anterior nerve roots. Sometimes these various irritative symptoms, motor and sensory, are the only symptoms. As a rule, however, the effusion of blood is sufficiently great to exert some degree of pressure upon the cord. Hence partial paralysis and anæsthesia soon develop in the legs. The paralysis is rarely absolute, for the degree of compression is not usually great. In a small number of cases paraplegia develops within a few minutes of the onset, and indicates a large hemorrhage (as from bursting of an aneurysm into the spinal canal) or coincident hæmatomyelia. The precise distribution of the symptoms naturally varies much with the level and vertical extent of the hemorrhage, but it is not necessary to describe here the various combinations of symptoms that arise.

In spinal meningeal hemorrhage the knee jerks are retained unless the lesion is in or near the lumbar region. When the reflexes are lost and the lesion is above the lumbar region, the loss probably always depends on an extensive coincident crush of the cord. During the early stage of the affection there may be spasmodic retention of the urine. Consciousness is occasionally impaired for a short time at the onset, but usually the mind is unaffected.

An extensive cervical extradural hemorrhage has been known to develop gradually, without pain, producing only increasing weakness in the arms and dyspnœa (Jackson).

The height of the symptoms is reached in a few hours, rarely in a few days. Improvement in the symptoms may be checked after a few days by symptoms of secondary meningitis.

Diagnosis.—The diagnosis depends on the combination of sudden local pain with the symptoms of irritation of the nerve roots and membranes. In meningitis the same irritative symptoms are met with, but they develop gradually and are attended by fever. The symptoms of hemorrhage into the cord differ from those of meningeal hemorrhage in the following respects: (1) vertebral pain is relatively slight and limited, and may be entirely absent; (2) the symptoms of damage to the cord itself are prominent—paralysis dominates the irritative symptoms from the outset; (3) in cases that partially recover the paralytic symptoms are persistent. Occasionally both types of hemorrhage are combined, as when the extravasation of hæmatomyelia breaks into the membranes. The order of development of the combined symptoms then gives a clue to the nature of the case. A diagnosis can hardly be made where the hemorrhage is of very gradual development.

Prognosis.—In the first hours the danger to life is great in all severe cases. The prognosis is worst where the lesion involves the cervical region, owing to the respiratory embarrassment. A considerable number of cases survive the dangers both of the irritative period and

of the secondary meningitis which sometimes develops. Paralytic and spastic symptoms may endure many months, and then pass away almost completely. In this respect the outlook is distinctly better than in cases of hæmatomyelia.

TREATMENT.—Absolute rest on the face or side is necessary at first. The abstraction of six or eight ounces of blood by scarification and cupping from a region near the spine and at the level of the pain is strongly to be advised. Later an ice-bag to the spine and ergot internally (as in hæmatomyelia) are indicated. If ice to the spine does not relieve the pain, morphine should be given subcutaneously ($\frac{1}{6}$–$\frac{1}{3}$ gr.). The bowels should be kept open by means of salts, but violent purgation should be avoided.

Both in extra- and intrameningeal hemorrhage the question of surgical interference arises when life is threatened and there is evidence that the cord is considerably compressed. The canal should be opened at the level of the lesion and the blood removed. If the blood is intradural, no hesitation need be felt about opening the dura to reach it if the proper asepsis is observed.

DISEASES OF THE BLOODVESSELS OF THE SPINAL CORD.

By CHRISTIAN A. HERTER, M. D.

Pathological changes in the walls of the bloodvessels of the spinal cord stand in close relationship to various important forms of spinal-cord disease. It has not been customary to consider such disease from the standpoint of vascular lesions, in part because a knowledge of these lesions is of very recent date and is still very imperfect, in part because the classification of spinal-cord disease on such lines would be essentially a pathological classification, and would throw into the same category conditions presenting wide clinical differences. It is instructive, however, to review what little is known about the pathological conditions of the vessels of the spinal cord, for it is certain that future studies of these conditions will do much to broaden and perhaps simplify our ideas regarding the origin of some common forms of spinal-cord disease.

ANÆMIA AND HYPERÆMIA OF THE SPINAL CORD.

A short time ago it was usual for writers upon nervous diseases to formulate an elaborate symptomatology and pathology for anæmia and hyperæmia of the spinal cord. Not merely were these clinical types drawn with a clearness of outline suggestive of confidence in their accuracy, but the types themselves were further subdivided and regional hyperæmias and anæmias were described—anæmia and hyperæmia of the anterior horns, of the lateral columns, etc.

We have now come to realize that these descriptions were based on hypothesis, and on hypothesis at variance with facts. We realize that there are few or no symptoms that can be fairly attributed solely to anæmia or congestion of the vessels of the cord, and that the descriptions referred to are chiefly descriptions of symptoms that arise from hysteria, neurasthenia, dyspepsia, and slight nutritional disturbances. There is no good reason for supposing that severe mental activity ever causes anæmia of the spinal cord, nor is there any reason to think that excessive sexual excitement gives rise to chronic congestion of the vessels of the cord. We are not in a position to disprove all the statements on which the clinical types of anæmia and hyperæmia depend, and the necessarily theoretical discussion of the question involved would be out of place here. The burden of proof rests with those

who assert the existence of distinct clinical forms of disease due to quantitative variations in the vascular supply of the cord. The contention of the writer is that we have not sufficient evidence on which to create clinical types based on primary variations in the blood supply of the spinal cord, and that we are probably never justified in practice in making a diagnosis of primary anæmia or hyperæmia of the vessels of the cord. But this is not to be construed as a denial that there are conditions under which the cord or portions of the cord are anæmic or congested, and in which this congestion may play a minor rôle in causing symptoms. For instance, suddenly produced anæmia of an area of the spinal cord at once abolishes function, and, if continued, leads to necrosis of the nerve elements; but such anæmia results only from structural disease of bloodvessels. In chronic spinal meningitis the blood passing through the thickened arteries is doubtless diminished in amount, but any effects which might be due to anæmia are obscured by the structural changes that generally exist in the meninges or cord. It is possible that anæmia of the cord due to vasomotor spasm is the cause of the tetanoid spasm and tingling of the hands which in some persons come on at night. A small dose of digitalis at bedtime may prevent these symptoms (Gowers), which favors this explanation. In anæmia and pernicious anæmia there is a universal disturbance of nutrition and function, from which the spinal cord is not exempt. In such patients the legs are readily fatigued and may be the seat of paræsthesia. The muscles and peripheral nerves may be as much concerned as the cord in producing these symptoms. It should be noted that in many cases of marked general anæmia there are no nervous symptoms referable to the spinal cord. In pernicious anæmia various observers (Lichtheim, Minnich, Nonne) have noted the occurrence of degenerative changes in the spinal cord, especially changes in the posterior columns, and the possibility of an organic basis for the spinal-cord symptoms of pernicious anæmia should never be lost sight of. It is further to be remembered that in pernicious anæmia the influence of a toxæmia upon the nerve elements cannot be excluded. Occasionally a paraplegia comes on after an extensive hemorrhage (especially from the stomach or uterus). Sensory symptoms are usually absent, but hyperæsthesia may occur (Leyden). The paralysis comes on from a few hours to a few days after the hemorrhage, lasts a few days or weeks, and, as a rule, ends in recovery. It may be that the symptoms in such cases are due wholly to the effect of the general anæmia upon the nerve elements of the cord.

Mechanical congestion of the cord, due to the influence of gravitation, may possibly in some debilitated persons give rise to an aching sensation in the legs and spine when the body is recumbent. It is very doubtful if such symptoms are met with in cases of heart disease with broken compensation.

So far as active congestion of the cord is concerned, our knowledge consists in the fact that such congestion occurs as the first stage of inflammation. If symptoms of beginning acute myelitis occur and then disappear wholly in the course of a few days, we may perhaps be justified in suspecting that they were due to congestion of the vessels of the cord.

The vessels of the cord are liable to become much dilated whenever

there is prolonged and violent activity of the nerve elements of the spinal cord, as in the spasms of tetanus, hydrophobia, or strychnine-poisoning, or after violent exercise or excessive sexual excitement. In all probability very considerable variations occur in the vascularity of the spinal cord within the limits of health.

If there is reason to suspect the existence of acute hyperæmia of the cord, an icebag may be applied to the spine, but we should remember that the symptoms which suggest such congestion are almost invariably, and perhaps always, the initial symptoms of myelitis.

The TREATMENT of spinal-cord symptoms due to general anæmia is the treatment of the latter condition (see Vol. II. p. 678).

ARTERITIS AND ENDARTERITIS.

As yet there is not a sufficient number of observations upon the occurrence of inflammation of the walls of the bloodvessels of the spinal cord to enable us to form an opinion as to the precise relation of these changes to the various lesions of the spinal cord with which they may be associated. Enough is known, however, to make it extremely probable that the changes in the walls of the small arteries (arteritis, endarteritis) of the cord are a very important cause of damage to the nerve elements themselves under certain conditions. These alterations in the walls of the vessels may arise acutely or only after the lapse of a considerable period of time. In both cases they are probably due to the action of irritants in the blood (bacteria, toxins), but the proof of this is lacking. The recent studies of Hektoen on tubercular meningitis make it clear that the tubercle bacilli may penetrate the unbroken endothelial layer of a vessel and stimulate marked proliferation of the subendothelial connective tissue. Possibly syphilitic arteritis may arise in a similar way. It is likely that the effect of an intense irritant, such as may be produced in the course of almost any infectious disease, is to act upon the endothelium of the walls of the smaller vessels and capillaries in such a way as to permit the escape through these walls first of serum then of leucocytes, the latter infiltrating and surrounding the vessel.[1] Where an acute arteritis is thus produced the irritant which gives rise to it may be competent to act directly on the nerve elements themselves and to give rise to symptoms. But very often the acute inflammation of the artery and the changes in the blood lead to the occurrence of thrombosis, with effects upon the nerve elements of the cord which will be discussed elsewhere. When the irritant is less intense and continues its action through a considerable period of time, as in the case of the syphilitic and tubercular poisons, the vascular changes are apt to consist chiefly in a proliferation of the endothelium or of the subendothelial connective tissue, which may reach a high grade (endarteritis obliterans), in addition to the small round-cell infiltration about the vessels ("coat-sleeve infiltration"). In syphilis, and perhaps also in tuberculosis, the walls of the vessels may undergo hyaline or waxy degeneration.

[1] There can be no doubt that arteritis often arises by extension of inflammation to the adventitia, and thence through all the coats of the vessel.

An important effect of a chronic arteritis or endarteritis of the smaller vessels feeding the spinal cord is to diminish the blood supply to the region or regions corresponding to the vessels affected. This decrease in the volume of blood is usually very slowly produced, owing to the gradual reduction in the lumina of the vessels; and this slow starvation leads in time to an overgrowth of the connective-tissue elements of the cord and a coincident atrophy of the nerve elements, with corresponding loss of function. We cannot be certain that the sclerotic process is the result solely of the diminished blood supply, for it is not unlikely that the toxæmia (if such it be) which caused the vascular change played a part in inciting the overgrowth of connective tissue. It seems clear, however, that the degenerative process in the cord is usually confined to the areas fed by the diseased vessels. This seems to have been clearly worked out in the case of locomotor ataxia, and is probably equally true of other degenerative diseases of the cord. The arterial changes referred to are especially common and extensive as the result of the syphilitic poison, which attacks the smaller bloodvessels of the spinal cord in a relentless way. The small veins are often involved in changes similar to those seen in the arteries.

Another important way in which the chronic arterial changes in the spinal cord act is through the production of thrombosis.

THROMBOSIS.

Theoretical considerations render it probable that the arrangement of the vascular supply of the spinal cord is such as to favor the occurrence of thrombosis. The arteries entering the cord are small in calibre, and, especially in the lower part of the cord, pursue a long and tortuous course. Moreover, these vessels anastomose freely on the surface of the cord, and the blood passing through them eventually leaves the cord by the very tortuous network of veins surrounding the dura. All these conditions are conducive to low arterial pressure and a slow blood stream. If in addition to these influences there are pathological changes in the vascular walls (especially endarteritis) or changes in the composition of the blood, it is easy to see why thrombosis of the arteries of the cord should occur. According to Kadyi, the small arteries entering the cord are terminal arteries, and we should expect their obstruction to give rise to acute softening of the corresponding parts of the spinal cord. It is indeed extremely probable that a large proportion of the cases of softening of the cord which have been described as examples of acute myelitis have been in reality examples of necrotic softening from vascular obstruction, and that the evidences of inflammation have been of secondary origin. This view is in accord with the conditions observed in the brain, where acute softening is almost invariably the consequence of arterial obstruction.

Unfortunately, we have not, as yet, many reliable pathological observations by which to prove the correctness of the views just expressed. The following case, reported by Williamson, is of interest in this con-

nection: A man who had contracted syphilis two years previously developed pain in the back. One month later retention of urine came on rapidly and was succeeded by unsteadiness of gait. The following day both legs were completely paralyzed, there was complete anæsthesia up to the level of the sixth intercostal space, and the sphincters were involved. Death occurred fifteen days after the onset of the paralysis. The chief changes found in the most diseased portion of the cord (mid-dorsal region) were thrombosis and great dilatation of the vessels, inflammatory changes (increase of nuclei in and about the vessel walls), and hemorrhagic infiltration around the vessels where the disease was most marked. It is likely that thrombosis of the bloodvessels occurred before the hemorrhage, and not at the onset of it, because "(1) though the thrombosis was well marked in the mid-dorsal region at the seat of hemorrhage, yet thrombosed vessels were found for some distance above and below, and also in the meninges far away from the hemorrhage (in the cervical and lumbar regions); (2) in most of the sections in the mid-dorsal region the chief features were the thrombosed and dilated vessels, whilst the surrounding hemorrhagic infiltration was comparatively small, and in some sections absent; (3) many of the thrombi were of older date than the hemorrhage; (4) the blood-corpuscles in many of the thrombosed vessels were broken down into granules, and the clot partially organized, whilst the corpuscles in the hemorrhage around were unaltered, and evidently the hemorrhage was more recent than the thrombus; (5) there were marked syphilitic vascular changes which alone would account for the thrombosis."

EMBOLISM.

At present there is no positive evidence that embolism of the vessels of the spinal cord is a cause of spinal-cord disease, but there are facts which strongly suggest that embolism in very rare instances gives rise to acute softening of the spinal cord. Thus Weiss records a case in which sudden and complete paraplegia, followed by bedsores, etc., developed in a boy aged sixteen with chronic mitral disease. Four months later he died, and the lumbar enlargement was found completely softened. The arteries of the spinal cord contained old coagula, there were infarctions of the spleen and kidney, and the cerebral cortex on either side was the seat of small foci of softening. Practically, all we can say of such cases is that we are justified in suspecting the existence of embolism of the vessels of the cord when a sudden cord lesion occurs in a patient with endocarditis (especially malignant endocarditis), who presents evidences of embolism elsewhere (brain, kidney, spleen). When we consider the anatomical conditions of the circulation in the spinal cord, it is not singular that embolism should be rare here as compared with embolism of the brain. The spinal arteries are so small and the anastomosis on the surface of the cord is so free that an extensive area of softening could probaby be produced only by multiple embolism. The largest arteries of the cord (anterior and posterior spinal) are

derived from the vertebrals, which, judging from cerebral embolism, only rarely convey emboli. The remaining spinal arteries, coming from the intercostal and lumbar arteries, are small twigs which an embolus is not likely to reach. From the recent experiments of Lamy it is evident that the obstruction of the small arteries of the spinal cord by means of inert powders gives rise to foci of hemorrhagic softening, which occur first in the gray substance.

It is of interest to note the relation of the topography of certain lesions to blood supply of the cord. Thus it has been pointed out by Williamson that the area affected in bilateral poliomyelitis corresponds to the distribution of the anterior median artery. In hereditary ataxia (Friedreich's disease) the degenerated area corresponds roughly to the part supplied by the peripheral arteries of the cord (the vasa corona). In combined posterior and lateral sclerosis the sclerotic region corresponds in a general way with the area supplied by the posterior arterial system of the cord (Marie).

PARALYSIS FROM LESSENED ATMOSPHERIC PRESSURE.

SYNONYMS.—Caisson disease; Diver's paralysis.

DEFINITION.—A form of nervous disease characterized usually by paralysis (paraplegia) and by a variety of nervous symptoms due to rapidly lessened atmospheric pressure following exposure to greatly increased pressure.

ETIOLOGY.—The immediate cause of the nervous symptoms is the rapid reduction of atmospheric pressure which occurs on returning from a caisson or diver's apparatus to the outer air. That the suddenness of the change is an important element is shown by the fact that if the lowering of pressure is very gradual, the nervous symptoms rarely or never occur.

The increase in pressure is usually more than two atmospheres; if it is less, symptoms seldom occur.

From the nature of the exposure the disease is naturally limited to males.

A variety of conditions appear to predispose to the affection. Whatever lowers general vitality increases the susceptibility. Alcoholism, myocarditis, chronic nephritis, old age, seem to operate in this way. There is some reason to think that obesity and fulness of habit predispose to the disease. When the Brooklyn Bridge was in process of construction a strikingly large proportion of cases was noted among heavily built and corpulent men (Andrew H. Smith). Novices are subjected to far greater risk than those who have become accustomed to work at gradually increasing depths. It is stated that during the building of the St. Louis Bridge nearly all the deaths were among inexperienced workmen whose first watch was under considerable pressure. The duration of the exposure is also a factor of importance, the risk increasing rapidly after the first hour. Excessive muscular exertion and ex-

posure to draughts after leaving the lock probably predispose to the condition. There seems to be no doubt that the risk is greatly increased by entering the caisson in a fasting condition (Jaminet, Andrew H. Smith).

Pathological Anatomy.—The facts regarding the pathology of the caisson disease are so meagre that it can hardly be profitably discussed. There is some reason to think that the symptoms of caisson disease are due to the escape of gases from the blood which have been dissolved in considerable excess during the exposure, and which make their escape on the return to the normal atmospheric pressure. It is thought that if the excess of gas in the blood is not extreme, the lungs are competent to rid the blood of it, but that if the excess is great, small bubbles of gas are liberated in the body and damage the structure and functions of the spinal cord. In a few cases appearances have been found in the human spinal cord which suggested that there had been a sudden escape of gas into the lateral columns, and it is claimed that bubbles of gas were seen in the vessels and nervous tissues of the spinal cords of dogs which had been let down to various depths in a diver's apparatus (Catsaris).

In many patients who have died from caisson disease the cerebrospinal pia is found to be congested, and the substance of the brain and cord may have the appearance of being congested. It is held by some writers that the paralysis depends on congestion of the cord followed by stasis of blood (which would be equivalent to anæmia). That the symptoms do not depend on congestion itself is indicated by the fact that they do not come on at the time when we must assume the congestion to be greatest—namely, during the exposure to pressure. But the congestion is probably followed by a flow of blood from the cord on the return to the normal pressure, and this anæmia of the cord may be the cause of the symptoms. We know that in rabbits in which the lumbar cord is rendered anæmic by shutting off the blood supply by pressure on the aorta, paralysis comes on suddenly, and is often accompanied by signs of severe pain. If the anæmia lasts more than an hour, the paralysis is permanent, and a true anæmic necrosis occurs, which is followed by an adjacent reactionary myelitis. A myelitis occurs in many subjects of caisson disease if the patients live long enough, and perhaps arises as the consequence of an anæmic necrosis, but the facts are not sufficient to warrant us in assuming this view to be correct.

Symptoms.—The symptoms usually come on rapidly within a few minutes after the return to the surface, but may be delayed for half an hour or, rarely, an hour. Dyspnœa, tinnitus aurium, nausea, vomiting, and severe pains, especially in the joints, are usually among the earliest symptoms. The pains are so common as to be characteristic. In severe cases a condition of collapse rapidly supervenes, and death may occur in the course of a few hours. In mild cases the chief symptoms are pain and tingling in the extremities, which wear away in the course of a day or two. In such cases there is no paralysis. In other cases paralysis is a more or less prominent feature. It is usually paraplegic, involving chiefly the legs; rarely it is hemiplegic. If the paralysis is partial, there may be complete recovery in a few days; if complete, it usually lasts for weeks and may be permanent. In severe cases anæs-

thesia is present, and may be of wide extent and complete in degree. The sphincters are implicated in some cases. In many cases the skin is the seat of large hyperæmic areas. Sometimes many small petechial spots appear. In short, in such cases the clinical history is that of an acute myelitis, with marked vaso-motor disturbances of general distribution. There also occur fatal cases in which paralysis is slight or absent, and in which death does not come on for several days or a week. Here the prominent symptoms are sharp neuralgic pain, hyperæsthesia, exaggerated reflexes, dyspnœa, and cyanosis. In some cases which have entirely or partially recovered from paraplegia, tremor and inco-ordination are developed in the course of a few months. With these symptoms there may be exaggerated reflexes, hyperæsthesia, and headache. Tinnitus and hysterical symptoms may make their appearance.

Diagnosis.—The relation of the symptoms in the early stage to their cause is so obvious that an error in diagnosis cannot occur. When the secondary symptoms occur their origin may readily be overlooked if the patient does not volunteer the information that he once suffered from caisson disease.

Prognosis.—The prognosis as regards life is good in the majority of cases of caisson paralysis; probably less than 10 per cent. prove fatal. When death occurs it is apt to come early, either within a few hours, in collapse, or after a few days, from respiratory embarrassment. The cases in which cyanosis is marked and persistent should be regarded with suspicion even though paralysis is slight. Death may occur late from the effects of the secondary myelitis. Some patients live for years with partial paralysis and a spastic gait.

Treatment.—Prophylactic treatment consists in causing workmen in caissons and divers to accustom themselves gradually to the increased pressure, in limiting the exposure to one hour at a time, in reducing the pressure very gradually (five minutes for each atmosphere) on the way to the surface, and in avoiding exposure when the stomach is without food. When the symptoms have developed recompression usually relieves them if practised early. After relief by recompression the pressure should be very gradually reduced. When the disease is established morphine may be employed continuously, at first for the relief of pain. Bandaging the painful limbs tightly may give much relief (Snell). The fluid extract of ergot should be given in large doses (℥j–q. 4 hrs.), as it certainly exerts control over the irritative symptoms in some cases. In cases which recover from the paralysis, but in which there are tremor, hyperæsthesia, headache, etc., ergot in large doses sometimes reduces the symptoms markedly while the drug is being given. When the paralysis persists the treatment is that indicated in Myelitis (see page 156).

HEMORRHAGE INTO THE SPINAL CORD.

Synonyms.—Hæmatomyelia; Spinal apoplexy.

Definition.—Hemorrhage into the substance of the spinal cord as distinguished from hemorrhage into or between the membranes. The hemorrhage in the cases under consideration is primary, and the in-

stances of hemorrhage into a previously diseased spinal cord (hemorrhage secondary to myelitis, to acute poliomyelitis, or to tumors and cavities in the cord), and the ante-mortem punctate hemorrhages that occur in convulsive diseases (tetanus, hydrophobia, etc.), are not here included. Nor is hemorrhage secondary to crushing of the cord referred to here.

Etiology.—Probably more than nine tenths of all the cases of hæmatomyelia follow an injury, such as a blow on the back, a fall in the sitting posture against the shoulder, or a forced movement of the head. In dislocations and fractures of the vertebræ hemorrhage into the cord is common, but in the cases under consideration there is no appreciable damage to the spine. Both violent muscular exertion and repeated coitus have been known to occasion hæmatomyelia. Whether in these cases the vessels of the cord are necessarily the seat of pre-existing disease cannot be stated, but it is probable that arterial disease in the cord is more common than has been suspected. In some cases the hemorrhage has occurred in persons with hæmophilia. It is said to have followed the suppression of the menses and of hemorrhoidal bleeding (Oppenheim). Occasionally it has occurred without known cause. The condition is much more common in males than in females, for obvious reasons. It has been observed at all periods of life, including infancy, but is oftenest observed between twenty and forty. Primary hæmatomyelia is a rare condition, perhaps even more rare than tumor in the substance of the cord. Its occurrence aside from trauma was once denied (Hayem), but without sufficient reason.

Pathological Anatomy.—The gray substance of the cord is the usual seat of primary hemorrhage, doubtless owing to its rich blood supply and the feeble support which it affords the vessels. The gray substance of the enlargements is especially liable to hemorrhage, the cervical more so than the lumbar. The extravasation displaces the cord substance and replaces it with a clot which is sometimes fusiform, sometimes spheroidal or irregular in form, and of varying length. Usually the vertical extent of the hemorrhage is from half an inch to a few inches, but occasionally it extends the entire length of the cord, sometimes in long narrow lines in the gray or white matter, sometimes in the central canal. The cord may be markedly enlarged at the level of the hemorrhage. The blood may break its way into the membranes, but this is not common. A certain amount of necrotic softening usually occurs in the immediate vicinity of the extravasation.

It should be noted that the seat of predilection for hemorrhage in the cord is much the same as that of the gliosis that leads to syringomyelia. The bearing of this fact will be seen in reviewing the symptoms. Occasionally a considerable hemorrhage occurs in the central disintegrating region of a gliomatous tumor, and it has been suggested that gliosis may develop in the structures immediately about a primary hemorrhage; but there is little evidence in favor of this view. In a very few cases miliary aneurysms are said to have been found on vessels contiguous to a hemorrhage (Bloeq). The striking contrast between the frequency of cerebral hemorrhage and the rarity of hæmatomyelia has often been commented on, but never fully explained. It is reasonable to suppose that the following facts are partly responsible for the differ-

ence: (1) The long and tortuous arteries which supply the cord are not subjected to high pressure; (2) miliary aneurysms are rare; and (3) there is less supporting connective tissue in the brain than in the cord.

Symptoms.—The onset of the symptoms is usually sudden or very rapid. In rare cases it has been associated with loss of consciousness of short duration, but this has in many instances been due to associated concussion. It is possible that in some cases a considerable hemorrhage in the upper part of the cord is, *per se*, the cause of temporary loss of consciousness.

The onset may be ushered in with spinal pain or sharp pain radiating along the nerves involved, but this may not be a conspicuous feature. Motor paralysis is the cardinal symptom. The degree depends on the extent of the hemorrhage, its position on that of the lesion. The presence or absence of muscular atrophy depends on the locality of the lesion. Rapid wasting is common. As a rule, the motor loss is paraplegic in distribution. Sensibility may be unimpaired, but usually there is some anæsthesia of irregular distribution. In short, the symptoms are much like those of a partial transverse myelitis. In a certain number of cases the symptoms have been those of a unilateral lesion, a Brown-Séquard paralysis,[1] more or less typical, being developed. In a not inconsiderable number of cases paralysis and wasting are found with disassociated anæsthesia; that is, with preservation of tactile sensibility and loss of pain and temperature senses. The pain and temperature senses are usually not completely lost, but only much impaired. The distribution of this sensory disturbance is usually irregular, but sometimes is strictly hemiplegic or is confined to a segment of a limb. As a rule, it is more marked on one side than on the other. This peculiar disturbance, which was formerly regarded as almost pathognomonic of syringomyelia, is thought to depend in these cases on a hæmatomyelia occupying the gray matter immediately about the central canal, especially on one side. The writer has observed cases of hemorrhage into the cord in which an upward extension of the symptoms was referable to the ascent of hemorrhage in the central canal.

Sequelæ.—The chief sequelæ are spastic paralysis, contractures, and trophic change in the skin, including bedsores.

Diagnosis.—The sudden onset of the symptoms with local pain are the features on which the diagnosis is based. The suddenly developed paralysis and the absence of root symptoms (severe radiating pains, spasm) distinguish hæmatomyelia from hæmatorrachis. The sudden onset without fever are features of distinction from acute myelitis, but it must be remembered that sudden hemorrhage may occur in the course of such a myelitis. When the patient is seen for the first time in a late stage of the affection the condition may be indistinguishable from gliosis with cavity formation (syringomyelia) unless the history of sudden onset or of marked retrogression of the symptoms can be obtained.

Prognosis.—In cases where the symptoms are of wide extent and great intensity death may occur in a few minutes or hours, and a relatively small lesion above the cervical enlargement may be quickly fatal. In a large majority of cases the symptoms undergo retrogression, the pain and paralysis growing less marked after a few days. If atrophy

[1] See article on Localization in the Spinal Cord, p. 71.

occurs rapidly, if the sphincters are paralyzed more than a week, and the sensory symptoms show no improvement, the prognosis should be exceedingly guarded, for recovery is apt to be slow and very limited in degree. The implication of either of the enlargements makes the probabilities of recovery less than in a lesion of the same size occupying the dorsal region. Whatever wasting and paralysis remain at the end of three months will lessen little or not at all. A considerable number of cases die in the course of a few months from cystitis or bedsores or exhaustion. Complete recovery is exceedingly rare. The cases that recover permanently usually present some degree of motor and sensory loss and some wasting, usually with contractures.

TREATMENT.—During the early period of the affection absolute rest in bed should be insisted upon. Probably it is better practice to have the patient lie upon the side or chest than upon the back, at least during the first days of the trouble. Sneezing, coughing, and straining should be carefully avoided by the patient. For the relief of the pain an ice-bag to the spine should be employed. It is likely that the use of large doses of ergot exerts some influence in controlling the hemorrhage and in preventing its immediate recurrence. The fluid extract should be given in drachm doses every hour during the first half day. If it cannot be borne by the stomach, it should be given by rectum diluted with six volumes of hot water. The bowels should be kept freely open for several days by means of salts, but active and continued purgation should be avoided. There is no indication for the use of electricity during this stage. The after-treatment is essentially that which is appropriate for myelitis.

ANTERIOR POLIOMYELITIS.

By M. ALLEN STARR, M. D.

Synonyms.—Infantile spinal paralysis: Acute atrophic paralysis; Atrophic spinal paralysis; Regressive paralysis.

Definition.—This is an acute disease, chiefly observed among children, but occasionally among adults, characterized by sudden complete loss of power in one or more limbs, usually in the legs, followed by rapid atrophy of the paralyzed muscles and by an imperfect growth of the limb affected, but not attended by any permanent sensory disorders.

Etiology.—The disease occurs in both sexes with about equal frequency. There is no history of its being inherited. The following table demonstrates that the age of maximum liability is between the first and fourth years, but children at all ages are liable to the disease, and it occurs in adult life:

Table I.—*Age of Onset.*

	1st Year.	2d.	3d.	4th.	5th.	6th.	7th.	8th.	9th.	10th.
Seeligmiller . . .	20	25	18	1	1	2				
Galbraith	17	38	15	4	1					
Sinkler	44	92	55	29	9	2	3	6	0	3
Gowers	21	21	25	9	17	4	2	6	4	
Starr	16	38	27	9	10	4	2	2	4	3
Total No. of cases	118	214	140	52	38	12	7	14	8	6

The youngest case on record is mentioned by Duchenne in a child twelve days old, and Sinkler has seen a case develop in a child at the age of six weeks. The youngest patient in my own records was five months old. It has been noticed by all authors since the time of Barlow (1878) that infantile paralysis develops most commonly during the warm season. This is especially true in England and in America, as is shown by Table II., which demonstrates the month of the year in which cases developed, as noted by Barlow, Gowers, Sinkler, and myself.

The disease has occurred in epidemic form in a number of different localities in every case during the summer. Colmer[1] first recorded the occurrence of the disease in epidemic form, for he mentions that in a village where he saw 1 case 10 other cases had developed during the preceding few weeks. Cordier[2] published an account of an epidemic occurring in Lyons, France, in 1885. He saw 13 cases developing during the months of June and July in a small town of 1500 people

[1] *Am. Journ. Med. Sci.*, 1843.

[2] *Lyon médical*, 1887.

TABLE II.—*Month of Onset.*

	Barlow.	Gowers.	Sinkler.	Starr.	Total.
January	1	1	4	2	8
February	0	1	3	1	5
March	4	1	9	6	20
April	2	1	4	2	9
May	4	1	10	3	18
June	5	11	27	6	49
July	16	13	52	16	97
August	11	13	65	27	116
September	4	15	29	17	65
October	3	6	25	8	42
November	1	2	4	4	11
December	2	5	3	2	12
	53	70	235	94	452

where in other years the disease had been extremely rare. Medin of Stockholm described an epidemic of the disease occurring in the months of August, September, and October, 1889, 44 cases having been observed by him during that time; and Rissler, who examined 3 of these cases post-mortem, demonstrated that it was a true anterior poliomyelitis. Medin mentions that a small epidemic had occurred in 1881 in the town of Umea in Sweden. Leegard observed a small epidemic in Mundal in Norway in 1890. The most extensive epidemic of the disease on record was described by Caverly[1] of Rutland, Vt. It occurred between the 20th of July and the 20th of September, 1895, in the Otter Creek Valley, within a radius of twelve miles of the city of Rutland, and during the summer an unusual number of isolated cases of the disease were observed through the State of Vermont. Caverly reports 144 cases of various grades of severity developing both in children and adults, children below the age of six being the chief victims. Pieraccini[2] observed a small epidemic near Florence, Italy, in the same year (1895) in July and August; and Medin[3] of Stockholm has recently reported a second epidemic in Stockholm in the year 1895. The occurrence of the disease in epidemic form is exceedingly suggestive of its infectious origin. In confirmation of this hypothesis it is to be noted that the disease has been frequently observed in connection with other infectious diseases. Thus it is an occasional sequel of diphtheria, meningitis, measles, pneumonia, scarlet fever, and acute malarial poisoning.

Exposure to cold or to a sudden check of perspiration has been supposed to be an exciting cause in certain cases. I have seen several children who were attacked with infantile spinal paralysis subsequent to long-continued bathing in cold water during the summer. Traumatism is frequently assigned as a cause by parents, and a few cases are on record, which I can confirm by my own observations, in which the disease has developed immediately after a severe fall or blow on the back. In some cases no cause can be discovered.

The frequency with which the disease appears in children who are learning to walk, together with the fact that the symptoms are located much more commonly in the legs than in the arms, has led to the sup-

[1] Caverly: *N. Y. Med. Record*, Dec. 1, 1895.
[2] Pieraccini: *Lo Sperimentali*, xlix. No. 27, Sept., 1895.
[3] Medin: *Nord. Med. Ark.*, 1896, No. 1.

position that a functional hyperæmia of the cord in its lumbar region, due to over-exertion, going on to a pathological congestion, and hemorrhage may be an etiological factor.

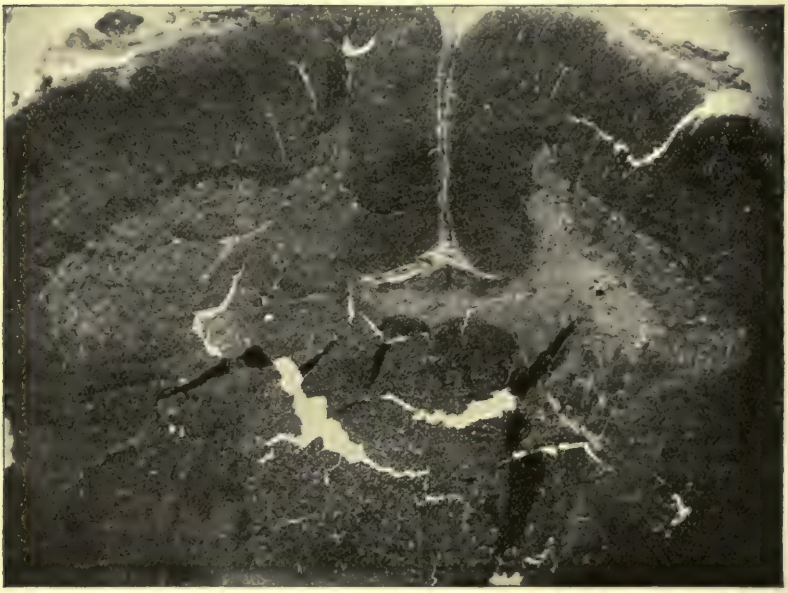
Pathological Anatomy.—It is only within the past few years that the exact pathological changes occurring in infantile spinal paralysis have been accurately described, autopsies having been recently obtained within a few days of the onset of the disease and at longer intervals in different cases up to the state of chronic permanent change in the cord. The earlier description of Charcot was based upon cases examined only in the chronic stage of the disease, and this fact explains the discrepancy between his observations and those of modern pathologists.

In the early stage of the disease there is active congestion of the spinal meninges and of the gray matter of the spinal cord supplied by the branches of the anterior spinal artery. The bloodvessels are distended, and some of the capillaries are ruptured, allowing extravasations of blood cells; the perivascular spaces and the gray matter of the cord are filled with emigrating leucocytes; and there is a considerable exudation of serum. The serum fills the lymph spaces about the vessels and about the nerve cells; the leucocytes infiltrate the tissues everywhere, cluster about the cells, and make their way into the cells. There is a great increase of small cells and nuclei throughout the neuroglia, which may be due to a proliferation of the neuroglia cells or of the endothelial elements, or may be due to an emigration from the bloodvessels. This infiltration of the tissues with leucocytes and nuclei may be so intense as to obscure all other elements. It is thus evident that the bloodvessels and the neuroglia, as well as the ganglion cells in the gray matter of the cord, share in the pathological process. The changes in the motor neurons (ganglion cells) are very marked. All varieties of degenerative changes may be seen. The cell may have a cloudy appearance and be slightly swollen, staining more deeply by reagents, the chromophile granules appearing to be larger than in the normal cells, as seen by the Nissl stain, and the nucleus appearing granular. A further stage of degeneration is shown by the fact that the protoplasm no longer absorbs stains; the cell is swollen, has lost its sharp outline, has a homogeneous appearance, and the nucleus is faint, as are also the outlines of some of the dendrites. It is probable that in both these stages of degeneration an arrest of the process is possible, and a gradual regeneration and a return to the condition of health with resumption of the function of the cell. If the process of degeneration proceeds beyond this point, however, no repair is possible and the function of the cell is for ever lost. When degeneration has gone on beyond the stage last described the cell appears to be changed into a swollen irregular or spherical mass of material; its protoplasm becomes cloudy, and its nucleus is not visible, if stained at all; the chromophile granules have lost their regular arrangement in concentric rings about the nucleus or in radiating lines toward a neuraxon, and the homogeneous mass is seen to be permeated with vacuoles, the dendrites having dropped off. In the last stage the protoplasm shrinks, the cell body being reduced in size very materially, having lost its polygonal shape and being now no larger than its original nucleus. It stains deeply and has a granular appearance. During

the later stages of this degeneration leucocytes may be observed penetrating into the pericellular space and encroaching upon the cell body. Both in the dendrites and in the neuraxon similar degenerative processes may be observed in progress, and they are destroyed before the cell undergoes its final degeneration.

While in the majority of cases there is a parallel degree of change in the interstitial tissues and in the ganglion cells, so that there is a shrinkage and progressive destruction of the neuroglia as well as of the nerve elements, there are a few cases in which the cellular degeneration is attended by few changes in the interstitial tissues. The degree to which various groups of cells are affected varies greatly at different levels of the cord, and in some groups a larger number of cells may be affected

FIG. 27.



Spinal cord at sixth cervical level, from a case of infantile paralysis. The atrophy of the right anterior horn, the existence of sclerotic scar tissue in the horn, as well as the absence of groups of cells, are shown. The left anterior horn is normal.

than in others. The extent of the paralysis and the degree of the paralysis in any one muscle will necessarily depend upon the number of groups of cells affected and upon the number of cells destroyed in any one group.

The result of the atrophy of cells and of the neuroglia is a gradual shrinkage of the entire size of the anterior horn, leading secondarily to a collapse inward of the white columns surrounding the gray matter and of the nerve fibres issuing from the horn through these white columns into the anterior nerve roots. There is also a degenerative atrophy of fibres in the anterior nerves. Many of the cells in the anterior horns of the cord send their neuraxons to the antero-lateral column, where they turn upward and downward to pass to other levels, and terminate in the anterior horn, thus serving to associate the action of

different cells lying at different levels of the cord. These association cells suffer from degeneration as well as the motor cells, and hence there is a secondary degenerative process in their neuraxons, leading to a shrinkage and slight sclerosis in the antero-lateral column of the cord for a varying distance above and below the site of the lesion. In various cases the extent of the lesion in the gray matter varies. In some cases the pathological change is strictly limited to the anterior horn, being particularly severe in its peripheral region. In other cases the central gray matter which lies between the anterior and posterior horns is also affected. It is in these latter cases that the symptom of pain is marked in the early stage of the disease, and the growth of the limbs is interfered with in the chronic stage, the central gray matter of the cord having a closer relation to the growth of the tissues than other parts.

The majority of recent pathologists believe that in anterior poliomyelitis there is an acute inflammatory process limited to the domain of the anterior spinal arteries, involving both the neuroglia and the ganglion cells, and resulting in degeneration and atrophy both of the interstitial tissue and of the ganglion cells. A few observers, among whom Von Kahliden[1] may be cited, still believe that Charcot was right in supposing that the degeneration was limited exclusively to the cells, and was not accompanied by any general inflammatory process in the interstitial tissues. In a number of cases the explanation of the origin of the process is found in a thrombosis of one of the branches of the spinal artery or in a hemorrhage into the anterior horn.

It is probable that in the cases which are due to infection the lesion is one of acute inflammation, such as we find in other organs in acute inflammatory infectious diseases—*e. g.* the lung in pneumonia, the joints in rheumatism. It is probable that in the cases in which there is an onset without fever or evidence of an infectious process the lesion is primary degeneration of the ganglion cells or is due to hemorrhage or thrombosis in a spinal vessel.

Symptoms.—The disease usually begins, like an acute infectious disease, with fever, sometimes attended by convulsions and delirium, especially in infants, sometimes by considerable pain in the back, body, and limbs, sometimes by digestive disturbances, vomiting, and diarrhœa, sometimes merely by general malaise. The temperature rises rapidly to 102° or 103° F., and the patient may have a chill followed by sweating. The temperature remains about 101° or 102° for several days, with slight morning remission, then gradually sinks to normal, the entire febrile movement rarely lasting more than a week. Within a day or two of the onset paralysis sets in, usually in both legs or in both arms or in one limb alone or in all four extremities. If the child is young and is confined to bed by the fever, the paralysis may not be noticed until the second or third day. In older children and adults the paralysis is usually well developed within twenty-four hours of the onset. It is observed that children cry a good deal during the period of onset, and some of those who are able to complain say that they suffer from pain in the back and in the affected limbs. This pain may remain for some weeks. Occasionally there is some rigidity of the spine or neck suggestive of meningitis, but this soon subsides. There is usually

[1] *Centralblatt. f. Allgem. Path. u Path. Anat.*, 1894, vol. v. p. 729.

no disturbance of the bladder or rectum, though in a few cases retention of urine has been noticed for a few days. There is no tendency to the development of bedsores or of trophic changes in the skin. There is no complaint of numbness or of paræsthesia, and there is never any loss of sensation, but the limbs are sometimes painful upon any movement, especially in the joints.

After the fever with its attendant malaise and digestive disturbances has subsided, and the general health has been restored, there remains a paralysis more or less extensive. This paralysis is usually more extensive at the onset than it is destined to be permanently. Thus the child may at first be completely helpless, and later recover power in all but one limb, or the trunk may be paralyzed at the onset, but not permanently affected. Both legs are commonly affected together, but the final paralysis is usually found in one limb only. Occasionally the neck muscles are distinctly weak and there may be difficulty in swallowing. This is seen in cases in which the arms are paralyzed, and yet the final paralysis may affect but one arm. The face has been paralyzed with the arms, and the ocular muscles also, but this is a rare occurrence. In a number of cases in which the final paralysis has been limited to two or three muscles the original paralysis was widespread, involving all the limbs. This fact should be remembered in giving a prognosis in the early stage. Sometimes the onset of the paralysis is not sudden, but there is a gradual increase during a week or ten days, then a stationary period, and then a regression. The subsidence of the paralysis begins from a week to two months after the onset, and then goes on steadily, but it is not until after three months that it is quite possible to determine what muscles will eventually recover. There is always a certain amount of paralysis which is permanent.

The muscles which are paralyzed undergo atrophy, which is more rapid and complete in those that are to be permanently paralyzed; and there is a change in the size of the limbs which is well marked within a month. The paralyzed muscles are relaxed, never rigid, and show a reaction of degeneration to the electrical tests. The reaction of degeneration consists of a loss of the response of both muscle and nerve to faradic stimulus, and a loss of response in the nerve to galvanic stimulus. The galvanic reaction of the muscle remains, but in such a muscle there is found an alteration of its normal contractility to galvanic currents. For the first few months the muscle responds too strongly to galvanism, and contracts under the positive pole more quickly than under the negative pole when the current is sent through it. Later the contractility to galvanism is progressively decreased, until in a totally paralyzed muscle it is lost. It may be stated as a prognostic sign that the muscles in which the faradic reaction is preserved will recover, though paralyzed for a time at the onset. Such muscles also preserve their tone, so that they contract when percussed sharply with a hammer.

The circulation in the affected limb is considerably impaired, and it is cold, blue, and flabby, but not œdematous. In some cases the bone is subsequently hampered in its growth, so that the limb is shorter and more slender than its fellow in after life.

While the description just given of an acute onset with fever applies to about three quarters of the cases of anterior poliomyelitis, there

remains one quarter in which there is no febrile onset.[1] In these cases the child while in a state of perfect health is suddenly paralyzed in one

FIG. 28.

Infantile paralysis, with atrophy and impaired growth of the right leg, with drop-foot; four years after the onset.

or more limbs. It gives no sign of pain, it does not appear to be ill,

[1] Of 100 consecutive cases in my clinic, 69 began with fever and 31 began without fever. Sinkler reports 178 with fever, 40 without fever.

and the paralysis surprises the mother by its sudden onset. In these cases the paralysis is soon followed by atrophy and by vasomotor paralysis. It is not attended by pain or tenderness on motion, and usually decreases to some extent, leaving the limb, however, in part permanently paralyzed.

These two types of onset of the disease are evidently quite distinct from one another, and their pathological basis is probably different, as has been already stated.

After the onset is over there is a slow progressive improvement up

FIG. 29.

Infantile paralysis and atrophy of the left arm two years after onset: the partial luxation of the humerus is evident and also the *main en griffe*.

to a certain point, and then the permanent condition of paralysis is found to vary greatly in different cases.

The location of the paralysis is usually in the legs, and here two types of the disease may be recognized—the leg type and the thigh type. In the leg type the peronei alone or with the anterior tibial muscles are most commonly affected, although the posterior tibial group may share in the paralysis or may even be as fully paralyzed as the others. As the paralysis remains, deformities of the ankle and foot will appear, the form of talipes developed depending upon the muscles chiefly paralyzed. In the thigh type the ileo-psoas and iliacus muscles and the glutei and

muscles about the thigh are the ones chiefly affected, the muscles on the inner side of the thigh and the muscles below the knee often escaping. In these cases the leg hangs like a flail from the body, and cannot support the weight at all. In some cases nearly all of the muscles of the lower extremity are paralyzed, and the atrophy is uniform throughout the limb. In these severe cases it is not uncommon for the muscles of the back and abdomen to share in the paralysis and atrophy.

When the arms are attacked two types of paralysis have been described, the upper-arm type and the lower-arm type. In the upper-arm type the muscles about the scapula and the deltoid, the biceps, and supinator longus are paralyzed and atrophic, and consequently the motions of the shoulder- and elbow-joints are seriously hampered. In these cases the shoulder-joint is unduly movable, and the head of the humerus falls out of the socket. In the lower-arm type the muscles below the elbow are invaded; the flexors or extensors of the wrist and fingers, or both together, are affected, the supinator longus escaping. In other cases the interossei muscles of the hand and the thenar and hypothenar muscles are paralyzed, while the long flexors and extensors escape. Occasionally a combination of upper- and lower-arm types occurs, in which case the entire extremity is useless. The upper part of the trunk is occasionally involved in the paralysis, together with the arms. Rarely the muscles of the back and trunk are the only ones permanently paralyzed.

In a very few cases the entire muscular system of the body appears to be affected by this disease; both legs, the trunk, and both arms are more or less paralyzed; but even in these cases a careful examination will show that the degree of the paralysis and atrophy is not the same in all the muscles. The relative frequency of paralysis in different parts of the body is shown in the following table (Table III.). The paralysis is very rarely, if ever, exactly symmetrical when both legs or both arms are involved.

TABLE III.—*Of Distribution of Permanent Paralysis.*

	Duchenne.[1]	Seeligmuller.[2]	Sinkler.[3]	Starr.	Total.
Both legs	9	14	107	40	170
Right leg	25	15	63	20	123
Left leg	7	27	62	27	123
Right arm	5	9	5	7	26
Left arm	5	4	8	4	21
Both arms	2	1	1	2	6
All extremities	5	2	35	5	47
Arm and leg same side	11	2	26	4	33
Arm and leg opposite sides	2	1	1	4	8
Trunk	1	. .	22	3	26
Three extremities	. .	. .	10	2	12

In addition to the paralysis and atrophy, there is a loss of reflex action at the level of the lesion in every case. The skin reflexes usually return after a time, but the deep reflexes are absent for a long period, even when a partial recovery of the muscle involved has taken place. Thus the knee jerk is uniformly absent when the thigh muscles are paralyzed, and the elbow and wrist jerks when the arms are affected.

[1] *Archives gén. de Méd.*, 1864, 38. [2] *Gerhardt's Handbuch d. Kinderkrankheiten.*
[3] *Keating's Cyclop. of Children's Diseases.*

Sensation is preserved in almost every case, but I have so frequently observed a permanent hypersensitive condition to painful impressions in the paralyzed limb that I cannot but believe that the lesion in the gray matter affects the pain-sense tracts in their passage through the cord at the level of their entrance, and has a relation to this symptom.

FIG. 30.

Infantile paralysis with atrophy of the right leg. The curvature of the spine is secondary to the shortening of the leg.

There is a marked vasomotor paralysis and lack of vasomotor response in the limb to applications of heat and cold.

Deformities of the joints are a common sequel in infantile spinal paralysis. The approximation of articular surfaces is secured in part by the tense action of the muscles, especially at the shoulder, hip, and knee; and hence paralysis of the muscles controlling these joints is

attended by relaxation and a greater degree of mobility than normal. Thus the head of the humerus falls from its socket when the deltoid is paralyzed, and abnormal extension of the knee is often seen in the upper-leg type of paralysis. After some months of paralysis the muscles which are the natural opponents of the paralyzed muscles are apt to become permanently contractured, and this also occasions deformities. The action of gravitation on a flaccid part of the limb combines with the contracture in the case of the foot to increase the deformity there, and hence all forms of talipes may ensue on infantile paralysis. Similar deformities of the wrist are also observed, but these are not common. Curvature of the spine from paralysis of the muscles of the back is frequently seen, all varieties having been described. Its most common cause is the shortness of one leg, due to arrest of its growth. Such curvatures differ from those due to bone disease in the fact that they do not persist during suspension of the body by the head and arms. It is one of the most important points in treatment to prevent the development of these deformities.

The progress of the disease in any case may be divided into stages. After an acute onset there is a stage of maximum intensity lasting from one to six weeks, and followed by a period of steady improvement which may extend from six months to a year. Then follows the permanent chronic condition, in which the normal growth of the child may lead to a slow development of the limb, but not to any change in its power of use. It is very rare for a complete recovery to take place after an attack of infantile paralysis. Even in the lightest cases there are usually some weakness, slight atrophy, and coldness left, and one or two muscles will be particularly feeble. In the majority of cases considerable permanent paralysis remains, requiring the use of apparatus to assist the use of the limb and to prevent deformities. Death has occasionally occurred during the acute onset, but is very rare, and once this stage is passed there is nothing in the disease to threaten life.

It is the chief characteristic of the atrophic paralysis in this disease that it selects certain muscles to the exclusion of others. This selection bears no relation to the arrangement of muscles in the limb or to the conjoint action of muscles in producing any definite movement. It is wholly dependent upon the arrangement of the groups of cells controlling the muscles in the anterior horns of the spinal cord. The exact localization of the disease can be determined by referring the symptoms in any case to the table given on p. 80 in the section on the Diagnosis of Spinal-cord Diseases.

Diagnosis.—There is no difficulty in recognizing the disease, and it is hardly likely to be mistaken for anything else. Occasionally a child will be attacked with acute articular rheumatism, and on account of the pain in the joints will be unwilling to move the limbs, and thus may be thought to be paralyzed. A careful examination will soon demonstrate the real condition, for acute rheumatism never causes any atrophy or paralysis, and the local tenderness in the joints, the sweating, and the lack of coldness of the limbs may also aid in the diagnosis. Rachitis, sometimes caused among children living in healthy and comfortable surroundings by the use of artificial patent foods containing considerable sugar, may lead to a sudden febrile onset, with much pain and tender-

ness in the limbs and unwillingness to move. But the child is not really paralyzed, and the state of its bones, the general condition, and the sweating, as well as the lack of limitation of the pain and immobility to one or two limbs, will prevent this disease from being mistaken for infantile paralysis. In some cases of anterior poliomyelitis there is considerable pain felt in the limbs, and some tenderness of the surface and of the muscles.[1] In painful cases it has been suggested that a neuritis accompanies the poliomyelitis. The existence of pain alone is not sufficient to warrant this diagnosis, inasmuch as the newer pathology indicates that in the early stages there is a congestion of the gray matter of the cord which might be sufficient to explain the pain. If, however, the pain continues and tenderness develops in the muscles and nerves, it is probable that a neuritis due to the same infectious agent has developed. It is to be remembered that polyneuritis is usually a disease affecting the extremities symmetrically, and causing drop-wrist and drop-foot; that the distal parts are more severely paralyzed than the proximal parts of the extremities; that there is no such selection of muscles paralyzed as in poliomyelitis; and that there are usually sensory disturbances of a permanent nature, anæsthesia and analgesia or ataxia, in addition to the pain and tenderness along the nerves; hence in the acute stage of onset a polyneuritis should not be confounded with a poliomyelitis. When polyneuritis accompanies poliomyelitis the clinical picture will be made up of a combination of these symptoms.

A localized injury of the brachial plexus (Erb's paralysis), causing paralysis of the deltoid, biceps, coraco-brachialis, and supinator longus, is not uncommon in infants, and might be mistaken for infantile palsy. The history of trauma during delivery and the local anæsthesia in the distribution of the circumflex nerve will, however, correct the mistake. Such cases usually recover.

Prognosis.—The prognosis in anterior poliomyelitis is always grave. Patients do not often die of the affection, but they rarely escape a permanent paralysis in some part of the body. It is true that in the majority of cases the original paralysis subsides, so that there is an apparent improvement of a considerable degree. Thus a patient who has originally been paralyzed in both legs may recover entirely the power in one leg, and may be left with a condition of paralysis in the peronei or in the anterior tibial group of the other leg, so that the terminal condition is very much less severe than that of the onset. As a rule, the limb that is affected never entirely regains its power, and usually shows some atrophy and shortening, for the growth of the limbs is hampered by the existence of the disease, and hence in a growing child the unaffected limb outgrows the other. It is thought that an electrical examination after the onset of the disease will afford some ground for a prognosis. It is believed that the muscles which respond to the faradic

[1] The existence of pain during the first two days of the disease occasionally leads to mistakes in diagnosis. Thus Marsh (*Lancet*, Jan. 16, 1897) records a case of a child five years of age who was suddenly attacked with pain in the left leg extending down the thigh to the knee. The limb was flexed, abducted, and rotated outward, and any motion was painful; hence the case was recorded as acute hip disease, but closer examination showed the hip-joint to be quite freely movable, and after two days, when the pain had passed away, the case was found to be one of infantile paralysis. The fever and general constitutional disturbances present at the onset had rendered the diagnosis obscure.

current three weeks after the onset of the disease will eventually recover, while those that fail to respond to this current at that time will always be somewhat impaired in power. The loss of faradic reaction, however, is not an indication that these muscles will be totally paralyzed, since the faradic reaction has been known to return in a muscle a year after it has been lost; yet such a muscle never recovers completely its size or power. The prognosis is much better in the cases which begin with fever than in those which do not.

TREATMENT.—The treatment of infantile spinal paralysis in the acute stage consists in keeping the child quiet in bed and applying a mild form of counter-irritation along the spine, which is best done by a paste of mustard 1 part and flour 3 parts, applied in a poultice along the back and removed as soon as the skin is reddened, and then renewed after three hours, so that for at least a week there shall be continual counter-irritation without the discomfort of a blister. The frequent application of dry cups along the spine may be used to produce the same effect. Frequent sponging with alcohol and cool water is indicated in the cases in which the temperature is above 101° F., but phenacetine or antipyrine is not to be used unless the temperature reaches 103° F. There is some advantage to be gained from the internal use of ergot. The dose of ergot is 10 minims of the fluid extract for a child below the age of two years every four hours, and 2 minims more for each additional year. Iodide of potassium may be given in 1-grain dose in the early stage, and moderate doses of salicylate of sodium (2 gr.) or of quinine ($\frac{1}{2}$ gr.) may be used for a child of two years. If the child is in much pain or has convulsions, bromide of sodium (5 gr.), with or without codeine ($\frac{1}{10}$ gr.), may be employed as a symptomatic remedy. The general treatment of febrile conditions, a light diet and laxatives, are not to be neglected. The best laxative is castor oil ʒj, glycerine ʒj, cinnamon-water ♏ x, given with an equal amount of lemon juice sweetened. Rest in a prone position in bed is better than constant lying upon the back.

When the acute stage is passed there is little to be done during the first week excepting to nourish the child well and keep the paralyzed limb warm. Iodide of potassium in 1- to 3-gr. doses may be administered three times a day.

When the paralysis begins to subside spontaneously it is well to administer strychnine in full dose, $\frac{1}{30}$ gr., three times a day for a child of three years of age. This remedy is best given at intervals, and not continuously, and it is my rule to use it one week and then intermit for three days. The condition of mechanical irritability in unparalyzed muscles, as determined by percussion with a hammer, is a good indication of the degree of effect being produced by the strychnine, and the strychnine may be increased up to the point of a distinct increase in this irritation. It is to be remembered, however, that twitching of the limbs or stiffness of the back, usually indicative of strychnine effect, is not to be relied upon in infantile paralysis when the muscles are paralyzed. Whether general tonics, such as cod-liver oil, hypophosphites, or arsenic, have any effect of a favorable kind may be left to the judgment of the physician in each individual case.

The most important indication during the stage of regression is to preserve the nutrition and function of the paralyzed muscles, and this

is to be attained by skilful massage, by hydrotherapy, or by the use of electricity. Massage is of the utmost importance in these cases, and should be given once or twice a day with care, combined with such attempts at active movement as the child is able to make. Among the poorer classes it is well to instruct the mother how to give this, so that it should be given with persistence. The massage should not be of the hardest kind, and yet should be sufficient to stimulate the circulation in the limbs and promote the lymphatic and venous flow. Next to massage, mechanical devices which shall induce the child to make use of the weakened limb are to be employed. A household gymnasium can easily be devised by the physician adapted to each individual case, and if such exercises are made of the nature of play to the child's imagination, much good will be derived from his own efforts.

Hydrotherapy is also an important aid in treatment. The general circulation in the cold and flabby limb is aided by warm baths, and it is my habit to have these children play in warm water, temperature 99° F., for a half hour twice daily. This warm bath may be followed by a cooler sponging and brisk rubbing, but cold water should not be employed in the bathing of these children, as the temperature of the paralyzed limb is always below that of health, and the vasomotor paralysis prevents the quick reaction which is so beneficial in other conditions. Proper protection of these limbs by extra flannel clothing is always advisable.

Electricity is a valuable agent in the treatment of infantile paralysis, but a clear statement of its use should be made by the physician to the family. Electricity has no influence whatever upon the course of the disease. It does not affect the lesion in the spinal cord either to decrease the hyperæmia or to increase the nutrition of the nerve centre. Applications, therefore, of galvanism to the spine are absolutely useless, but applications to the muscles may be of distinct service in two different ways—first, by causing their contraction, and thus exercising them when voluntary exercise is impossible, and secondly, by promoting the chemical changes in the muscles which are essential to growth and nutrition.

Examination in any case will show a certain number of muscles in the paralyzed limb which respond to faradism. These muscles will eventually recover entirely, yet the tone of the muscles and their strength can be kept up during the period of improvement by means of exercise with either the faradic or galvanic current. It is quite well proven that just as exercise of a healthy arm will markedly increase the size of the biceps muscle, so applications of faradism regularly to a muscle which it will contract will increase the size of this muscle; hence to the weakened muscles which still respond to faradism an application of the faradic current for about ten minutes once or twice a day will be of service. The majority of the paralyzed muscles do not respond, however, to faradism, and it is time wasted to apply the faradic current to these muscles. They do respond, as a rule, to galvanic interrupted currents, the positive pole being placed over the muscle and the negative upon the limb at a short distance above. The interruptions should be made by an electrode held in the hand and provided with a finger-key, and each muscle should be treated for about three minutes daily, fifty

to sixty interruptions being made per minute by the finger. The strength used should be the least which will secure contraction in the muscle. When interruptions of the current do not produce a prompt response alternation of the current may be employed by reversing the current rapidly by means of the pole-changer on the battery. It is to be remembered that in this disease the application of electricity is more painful than in health. It is also to be remembered in applying electricity to children that their confidence must be gained, and that if they are frightened at the first application, subsequent treatment will result in a continual struggle. It is my custom, therefore, to begin a course of electrical treatment to a child by several applications of the sponges and electrodes while no current is passing, thus accustoming the child to the apparatus and gaining its confidence. After two or three such applications it will be possible to use a weak current, and then day by day to increase its strength until by the end of ten days the necessary strength is being used. In this way a daily struggle, with the result of unsatisfactory and probably useless applications, can be avoided, and the parents' consent obtained to a course of treatment which they would eventually object to if every application resulted in a struggle. Any intelligent mother or nurse can be taught to give the galvanism or faradism to a child in this manner; and it is best to interest the attendant in the treatment from the beginning, and instruct her carefully, so that within a week the treatment can be left entirely in her hands. Such an application of electricity is to be made daily or twice a day for two or three years. Spontaneous recovery will have been reached at the end of the first year, but even after this time these muscles may be brought into a condition of hypertrophy by means of continued exercise. When, however, a child is quite able to move voluntarily with some force any paralyzed muscle, it is far better to rely upon voluntary exercises than upon electrical applications. If no effect is obtained from massage, bathing, and electricity in a muscle at the end of a year, there is no use in continuing the treatment of that muscle, as it will never recover, its nerve cells being entirely destroyed.

The use of apparatus plays a great part in the treatment of infantile paralysis in the chronic stage. It is to be remembered that many weak muscles can do their work only when the limb is placed in an advantageous position or when they are assisted in their action. Many of the muscles have as part of their function to keep the joints in place, and this part can be supplied by properly adjusted braces; hence an apparatus may enable the child to use a muscle or move a joint which it could not do if the joint were unsupported. Again, the result of paralysis of one group of muscles is to allow the joint to be bent by its opponent or to yield to the influence of gravitation, and hence the paralysis is often followed by deformity if a brace is not applied early to correct this tendency. There is no disease in which orthopædic apparatus is of more service than in infantile paralysis, and it cannot be applied too early, as it may prevent the development of contractures and of deformities. There is no stage in which it is too late to fit a brace, for even if these deformities have occurred, tenotomy may be employed to straighten and adjust a joint, and then the limb can be fixed by the brace in a proper position. But every case has to be

treated skilfully in accordance with its own condition, and the ready-made braces of the shops are often worse than none. Hence for each case a special apparatus must be fitted under the direction of an orthopædic surgeon; and it is to be remembered that in a growing child such apparatus must be constantly readjusted, its length and size being changed from month to month in accordance with the development of the limb.

In many cases of deformity where there is a strong contracture of a fairly healthy muscle overcoming the weak paralyzed muscle the question of tenotomy will arise. Such tenotomy will of course result in a replacement of the deformed limb to its natural position temporarily, but unless the joint can be held by a brace in a proper position, tenotomy alone will be of no permanent service. Hence tenotomy is only to be regarded as a preliminary in some cases to the proper application of apparatus. Apparatus has also been devised (especially in the treatment of infantile paralysis of the hands) by means of which weakened muscles may be reinforced by elastic bands so applied as to take the place of the paralyzed muscle. Thus a dropped wrist or a paralysis of the extensors of one side of the wrist can be somewhat relieved by a series of elastic bands attached to the finger-tips or to rings, and to the elbow, and running through a bracelet at the wrist. Dropped foot may also be similarly remedied. Such devices, however, are usually discarded after a time, as they are more cumbersome than useful. Apparatus is especially applicable to spinal curvature of the paralytic type, and in any case in which the body or back muscles are involved at the onset it is well for the child to wear a brace in order to prevent the development of some form of curvature. A thick cork sole will prevent the curvature due to a short leg.

It has been proposed to divide longitudinally tendons of certain healthy muscles, and attach one half to the severed tendons of paralyzed muscles about the ankle, knee, wrist, and elbow, in order that the healthy muscle may be made to do the work of the muscle which is paralyzed; and a few successful attempts in this direction have been reported. I have seen one case of paralysis of the peronei, in which a part of the posterior tibial tendon was attached to the cut peroneus longus tendon, permanently benefited in a remarkable degree by this method of treatment.

ACUTE AND CHRONIC MYELITIS; LANDRY'S PARALYSIS.

By EDWARD D. FISHER, M. D.

Definition.—Myelitis is an inflammation of the spinal cord. By this term is generally meant a lesion which involves the whole transverse area of the cord. The symptoms must, therefore, be such as show interference with the spinal cord as a whole; that is, we observe sensory, motor, reflex, trophic, and vasomotor disturbances. Leyden lays emphasis on the fact that clinically we cannot distinguish between softening of the cord—*i. e.* myelo-malacia—and inflammation. It is indeed possible to have inflammation without softening, but in the severe forms the intensity of the inflammation marks itself out by the degree of softening. On the other hand, there are various forms of softening of the cord, as in compression, hemorrhage, anæmia, etc., which are unaccompanied by inflammation. The various forms of myelitis have been classified as transverse, diffuse, disseminated, and central. Myelitis may be acute, subacute, or chronic: the subdivision subacute, however, is of very little value from a clinical standpoint.

ACUTE MYELITIS.

Definition.—Acute myelitis is an inflammation of the cord involving usually its whole area transversely, and extending longitudinally, as a rule, so as to involve one or two segments of the cord. Any lesion, therefore, destructive in character, as hemorrhage into the substance of the cord, laceration from traumatism or from fracture of the spine, or compression, as in caries of the spine, will give the same class of symptoms as we observe in inflammation of the spinal cord; in other words, in the study of the symptoms of myelitis one of the points of greatest importance to consider is the extent of the lesion involving the cord, as whatever the cause may be the results are the same.

Leyden gives an interesting subdivision of the forms of acute myelitis from three points of view: (1) From the extent and region of the cord involved; (2) from the etiological, and (3) from the clinical, standpoint, as follows:

I. Forms of acute myelitis according to the distribution of the lesion:
   (1) Transverse myelitis:
      (*a*) Dorsal myelitis;
      (*b*) Lumbar myelitis;
      (*c*) Cervical myelitis;
      (*d*) Bulbar myelitis;
      (*e*) Myelitis ascendens.
   (2) Multiple myelitis (disseminated).

II. Forms of myelitis according to the etiology:
   (*a*) Traumatic myelitis;
   (*b*) Compression myelitis;
   (*c*) Genuine myelitis—(1, due to infection, as in pregnancy and the puerperal period; 2, through intoxications and poisons; and 3, spontaneous myelitis—*i. e.* those without known cause).

III. Forms of myelitis according to the clinical aspect:
   (*a*) Myelitis apoplectica;
   (*b*) Myelitis acuta;
   (*c*) Myelitis subacuta.

Acute myelitis may, however, be diffuse; that is, involve the cord transversely to a considerable extent of its length, manifesting then symptoms similar to those of the limited area of the cord usually involved, but more general, and giving the picture of acute ascending, or, more rarely, descending, paralysis. This condition is rare, and as the distribution of the paralysis, and not its character, is the only difference from that of ordinary acute transverse myelitis, its description will be included in that of the latter.

Central myelitis may be limited to a few segments of the cord, or it may be diffuse, involving many segments. It differs in the symptomatology from acute transverse myelitis only in its location around the central canal. In most cases it affects principally the posterior horns; more rarely it affects the anterior horns. Being similar in its etiology and pathology, it will also be included in this description. As we might expect from its location, the symptoms will refer often to sensory disturbances similar to those observed in syringomyelia, with, at times, muscular atrophy. Another reason for considering it under this description is the tendency to extension of the inflammatory process. Thus, under this condition it is impossible to make the differential diagnosis from acute transverse myelitis, into which, in fact, it has passed.

Disseminated myelitis will require separate consideration, as, although the pathological changes are similar, the localization is so different that the symptoms present a clinical picture distinct from that of myelitis in general; they resemble rather multiple or disseminated sclerosis. It differs from the latter essentially in the acuteness of the onset of the disease, and at times in the paralysis and atrophy that are present. This relationship to myelitis is shown clinically by certain cases in which the disseminated lesions become united, probably by extension of the inflammatory process; and we have presented to us

the symptoms of diffuse or, more rarely, of transverse myelitis. A number of such cases have come under my personal observation during the past year in which for a time there was great difficulty in the diagnosis. I shall follow Leyden in his description of this disease, as he has taken it up as a special form of myelitis.

Etiology.—Exposure may be considered as a prominent factor, and, while we are unable to explain the process which leads to the vascular changes at the basis of inflammation, experience shows that such occupations as necessarily expose those thus employed either to extreme cold or to variations in temperature are particularly liable to result in myelitis. Thus, engineers, stokers, bakers, truckmen, etc., with constant exposure to heat and cold, are frequent subjects of this disease. Alcohol is without doubt a predisposing agent in all these cases. Myelitis not infrequently also accompanies or follows the various contagious and infectious diseases, such as measles, typhoid fever, smallpox, etc. The disease is at times an endemic or, more rarely, an epidemic acute infectious process. It has been observed in the course of influenza and malaria. Gout and rheumatism are occasional etiological factors, and it may follow neuritis from alcohol, arsenic, lead, mercury, diphtheria, etc. These latter conditions are comparatively rare, however, and, with the exception of diphtheria, are more apt to assume the chronic rather than the acute form of myelitis. Syphilis may cause typical acute transverse myelitis, although here, again, we generally observe the chronic form of the disease.

Acute myelitis may be secondary to other diseases of an inflammatory character, and may result from extension, as in meningitis, abscess, caries, or cancer of the spine.

This brings us to a class of causes which produce destructive lesions of the cord, resulting in symptoms precisely similar to those found in myelitis, which, however, if we are to strictly adhere to our definition of myelitis as an inflammation, may be questioned as coming under this head. I shall include them here, however, as they have the same symptomatology, although we may not be able at present to describe them under the same pathological division.

Traumatism is one of the commonest causes in this relation, and may produce laceration of the substance of the cord or hemorrhage into its substance, or, indeed, meningitis, causing compression of the cord. So-called concussion of the cord resulting in paralysis, while perhaps revealing no structural changes post-mortem, is probably explained by the presence of minute disturbances in the capillary circulation, for it is scarcely possible to conceive of such serious symptoms without a distinct lesion.

The myelitis present in caisson disease is probably due to the same cause—indeed, at times distinct capillary hemorrhage, leading to softening, is found in these cases. Hemorrhage into the cord, however, may take place independently of injury, as from exposure or muscular overexertion. Dislocations or fractures of the spinal column usually result in laceration of the cord or hemorrhage into its substance or destruction by definite compression. Compression myelitis is frequently present in caries of the spine, as in Pott's disease, and, although this is usually slow and chronic in its onset and course, it may at times be character-

ized by the sudden appearance of paralytic symptoms. Tumors of the meninges and cord may also be mentioned at this time, although, as with caries of the spine, the form of myelitis is chronic rather than acute. Exudations compressing the cord, as in pachymeningitis cervicalis, are not uncommon causes. They produce secondary symptoms of myelitis—in fact, in Pott's disease itself it is usually the exudation which causes the spinal-cord compression, and not the vertebræ themselves, even in cases where the kyphosis is extremely marked. These cases are more truly meningo-myelitis, and are therefore characterized by great pain, referred to the meninges at the site of the lesion, and extending thence into the parts supplied by the nerves involved in the meningeal inflammation.

Pain is rarely present, or is certainly never extreme in myelitis alone —in fact, if the lesion is complete and transverse, there is loss of all sensation below the lesion. Therefore, although the symptoms of myelitis may be well defined, if associated with it we have pain we can feel assured that there is some meningitis also present, and must therefore look for the etiological factor, probably of the myelitis, either in a meningitis pure and simple, which has extended into the cord substance, or for some compression lesion, as we have said, of the nature of bone disease, fracture, or a new growth.

Syphilis, gonorrhœa, tuberculosis, pregnancy, and septic abdominal inflammation, especially during the puerperal period, are not infrequent causes. I have observed myelitis following abdominal operations. I believe the usual cause in the cases is of a septic character. These cases may run a very rapid course, of a typical transverse myelitis, which it is impossible to believe is reflex, although it may be difficult to trace the course of the infection. These are the cases of metastatic myelitis, such as may follow purulent cerebro-spinal meningitis, abscess of the lungs, etc., in which we have abscess of the spinal cord (Rückenmark's abscess). Clinically they present the usual symptoms of myelitis.

Pathological Anatomy.—On inspection the gross changes observed in the cord are softening of its substance, even to complete diffluence, so that all form and structure are lost, and there is no distinction between the white and gray matter. The bloodvessels are dilated, and there is always more or less extravasation of blood from the capillaries, giving a reddish appearance to the cord, called "red softening." However, these terms, red, yellow, and white softening, are misleading, as they would seem to indicate a different inflammatory process, when the real cause of the difference in appearance is dependent alone on the amount of blood extravasated or the stage of the inflammation. In the so-called yellow or white softening, for example, the vascular changes are less marked or resorption is taking place. Besides these vascular changes, the gray matter shows most markedly the destructive character of the process: the cells are destroyed, and there may be considerable loss of substance, leaving spaces in the anterior horns. The white fibres, when the lesion is not a destructive one, are swollen in appearance, so that there seems to be an actual enlargement of the cord at the site of the lesion. In more marked cases, however, none of the fibres can be recognized as such, and the whole substance of the cord appears creamy and pultaceous. Microscopically, we observe various ele-

ments of the cord which have undergone degeneration. The cells are at first swollen and enlarged and the seat of granular degeneration. The processes are often lost and the cell itself vacuolated. The neuroglia tissue is the seat of inflammatory changes. We observe numerous spider cells or Deiter cells. They are also largely distributed throughout the white substance of the cord. The nerve fibres are also swollen, and there may be complete destruction of the myelin sheath, the axis cylinder alone remaining, which also later may disappear. In the swollen sheath we find fat globules of myelin, blood cells, leucocytes, amorphous material, various round cells of doubtful origin, and corpora amylacea. There is evidence of increase of neuroglia-tissue cells. The bloodvessels, especially the capillaries, are very much dilated, and their walls are lined with nuclei. There is often, as seen microscopically, considerable extravasation of blood. The perivascular spaces are filled with round cells, blood-corpuscles, and pigment masses. When the inflammation is mild in character there is simply a hyperæmia of the cord, with little if any extravasation of blood, and the consistence of the cord is very little changed. These changes are not symmetrical nor of equal intensity, certain parts of the cord remaining intact and lying in the midst of softened areas. In transverse myelitis not completely involving the cord the periphery usually escapes; or, again, one side of the cord may also be involved through many segments, as in diffuse myelitis, or only to a limited extent, as in transverse myelitis; or, again, the lesion may be a disseminated one, as indicated in the various forms of myelitis.

The later changes, after the inflammatory process has ceased, show an increase of connective tissue, so that as the acute process ends and the disease passes into the form of chronic myelitis we find many shrunken, atrophied cells without processes, ascending degeneration of the sensory tracts of the cord, and a descending degeneration of the motor tracts of the cord, with a descending and ascending degeneration of the association tracts. The bloodvessels are few and show thickening of their walls. These later changes are always present where the disease lasts six weeks or longer—in fact, the secondary changes have been estimated to occur as early as the second or third week. While there is considerable difference of opinion as to whether these changes are due to an inflammatory process or result from acute softening from plugging of capillaries, I believe the picture found corresponds more closely with the inflammatory theory than with the latter. In cases where the disease seems almost epidemic we can probably trace the cause to some infection of a microbic character. The origin is essentially vascular; in some cases the lesion is limited to the anterior horns, giving us poliomyelitis,[1] and in others, as we see, involving the whole transverse area of the cord, as in myelitis. The changes are similar in both conditions in the areas of the cord affected.

The same can be said of disseminated central and diffuse myelitis. In destructive lesion of the cord following traumatism, or in fracture of the spine or hemorrhage into the cord, the picture of the changes is not dissimilar to that described under the head of acute myelitis, especially in the later stages. The changes in the early stages of the

[1] See "Poliomyelitis," p. 129.

traumatic myelitis seem especially characterized by excessive hyperæmia of the cord, the arteries, capillaries, and veins being dilated and surrounded by masses of round cells and red blood-corpuscles. This infiltration may press the nerve fibres apart, destroying them, or they may become enlarged and swollen. Even at this early stage the nerve cells may appear shrunken and have lost their processes. The second stage of the inflammatory process shows a marked decrease in the hyperæmia, and evidence of fatty degeneration which gives the appearance of yellow or white softening. The nerve fibres now appear smaller and the seat of fatty degeneration involving both the sheath and the axis cylinder. The nerve cells are in many parts shrunken and destroyed, the walls of the vessels are thickened, and there is marked increase of the neuroglia connective tissue, compressing the nerve fibres.

In compression myelitis, as in Pott's disease, tumors of the cord, etc., the changes are identical with those of the secondary changes referred to in acute myelitis—increase of connective tissue, thickening of the walls of the vessels, and ascending and descending degeneration in the sensory and motor tracts.

Symptoms.—The characteristic symptoms in complete acute transverse myelitis are—paralysis of motion, occurring in a few hours or days, with complete loss of sensation below the site of the lesion, accompanied by paralysis of the bladder and rectum. Within a week the appearance of bedsores usually occurs over the sacrum, the hips, and the heels, with more or less atrophy of the muscles, making a picture hardly possible of confusion with any other disease. These disturbances of spinal function may be accompanied by some temperature changes and even convulsions.

We shall now go more into detail in the description of the various individual symptoms. The motor symptoms consist of paralysis of the parts below the line of lesion. The disease is usually situated at the lower dorsal segment.[1] The paralysis would, therefore, in this case involve the lower extremities, including also paralysis of the bladder and rectum and loss of sensation below the lesion. The legs are absolutely powerless. The patient is able to move the trunk muscles, and by the aid of his arms turn himself upon his side. The muscles are rigid and show some wasting, even in the muscles not supplied by the segment involved in the lesion. However, the atrophy is extreme only in those muscles which are supplied by those spinal nerves involved in the special portion of the spinal cord which is diseased. It is well here to refer also to the electrical reaction of these muscles, as it gives us the means of accurately localizing the extent and situation of the spinal lesion. All the muscles cut off from their source of nutrition—the large multipolar cells lying in the anterior cornua—undergo marked wasting within a few days (ten) of the onset of the lesion, and there is complete loss of faradic response and the presence of the reaction of degeneration. In the early stages of the disease, indeed, there is increased response to galvanism, with, however, the qualitative changes referred to. The muscles below the lesion, not supplied by the diseased segment of the cord, are also paralyzed, as already stated, but this is due to the fact that the impulses from the brain can no longer reach them, being inter-

[1] See "Localization of Spinal-cord Disease," p. 71.

rupted by the lesion in the spinal cord, which lies between the brain and the spinal segment of these muscles. There is not, therefore, in these muscles any marked wasting; the latter, indeed, if present, is the result of disuse. Nor is there the same degree of flaccidity referred to in the muscles whose nutritional centres have been destroyed.

It is usual after a few weeks to find some rigidity present in these muscles, and spasmodic jerkings on slight peripheral irritation; or, again, contractions may take place involuntarily. The electrical response, as would be expected from what has been said, is not affected qualitatively to either the faradic or the galvanic current. Therefore the reaction of degeneration is never present, although a slight decrease in the amount of electrical response is usually found—in other words, a slight quantitative reduction. These conditions are all well defined in the later stages of myelitis when it is passing into the chronic form, but it is of great importance to recognize the condition at the onset of the disease. The reflexes are exaggerated, giving an exaggerated patellar reflex and ankle clonus.

When the lumbar segment is involved, the paralyzed muscles are flaccid and the reflexes, both superficial and deep, are lost. The atrophy of the muscles is rapid and the reaction of degeneration is well defined. The marked difference between this condition and that of the dorsal lesion is flaccidity of the muscles and their loss of tonus. The paralysis is also more complete, involving the muscles of the foot and ankle and giving thus a pronounced "foot-drop," leading to deformities in position. The bladder is paralyzed, but in this case the centre of the sphincter itself is involved, and there is incontinence from the beginning.

When the lesion involves the cervical region of the cord, the paralysis is situated in all parts below the diseased segment, but the wasting and atrophy affect only the arms; and here, perhaps, we see more clearly defined the distinction which we have spoken of in regard to the condition of the muscles at the site of the lesion in the cord and those supplied by the segments further below, and therefore not included in the inflammatory disease. The loss of faradic response and the reaction of degeneration are thus only present in the muscles of the upper extremities, which are also flaccid; the lower extremities, while of course paralyzed, are not usually flaccid, but often rigid, and show slight changes only of electrical response. The bladder and rectum are involved in the same manner as in the lesion in the dorsal region; that is, we have incontinence from overflow of the bladder. The spinal centre of the bladder and rectum, being situated in the sacral segment, is not involved in either of these lesions of the cord. However, the connection of the bladder with the brain being cut off by the disease, the bladder fills without the brain being informed of it—in other words, the patient is unconscious of it. The brain is unable on this account to send any impulses to inhibit the sphincter; hence the bladder becomes overfilled, and finally overcomes the sphincter and the urine escapes. This is quite different from the form of incontinence which occurs when the lesion involves the spinal centre for the bladder. Here, it is true, there is an incontinence, but it is due to another cause—*i. e.* the paralysis of the centre for the sphincter—the result being continuous passing of the urine from the bladder as soon as it reaches that organ from the

ureters. This would occur whether the influence of the brain over the cord remained intact or not, as in this case the reflex spinal centre is destroyed.

We may also, in this lesion, have disturbance of respiration from paralysis of the chest and abdominal muscles, or, when the lesion is still higher, paralysis of the diaphragm. When the lesion involves the eighth cervical or the first dorsal we find pupillary changes, in which the pupil is contracted. There is some narrowing of the palpebral fissure and the eyes appear retracted.

One word requires to be said in regard to the deep spinal reflexes: In the majority of cases of myelitis we find the patellar reflexes exaggerated when the lesion is above its spinal centre, but in not a few we may find them lost when the lesion is situated in the cervical and dorsal regions. It has been observed in injuries of the spinal cord of the cervical region, which have resulted in complete destruction of the cord, that the patellar reflexes, contrary to what we would suppose from a physiological standpoint, are absent; and this has been regarded clinically as a diagnostic point, as proving that the cord had been completely destroyed at that situation. I think from analogy, therefore, we can conclude, in cases of inflammatory origin when this symptom is present, that we are dealing with a very severe lesion which has destroyed the cord, and we must regard the prognosis as especially unfavorable.

Disturbances of Sensation.—In complete transverse myelitis there is observed loss of sensation in all the parts below the lesion. This applies to all forms of sensation—*i. e.* touch, temperature, pain, muscular sense, and general sensibility. The patient suffers absolutely no pain in this disease in the parts affected. This applies in all destructive lesions, and is of importance to remember as a diagnostic feature of the disease, and also in regard to treatment, for otherwise the application of heat to the parts, not being perceived by the patient, and therefore not complained of, may lead to extensive destruction of the skin, causing sloughing of the soft parts.

At the upper border of the lesion in the cord there is always more or less hyperæsthesia, the patient being susceptible to all forms of sensation, even excessive pain being occasioned by moderate irritation, as from the examination with an æsthesiometer. The explanation of this probably lies in the fact that the sensory nerves entering the spinal cord at this site are subject to inflammatory changes. There is often also considerable pain in the back in this region—a "girdle pain," involving the whole body like a band or belt. This is probably due to some involvement of the spinal membranes, as lesions strictly confined to the cord substance rarely cause pain. It is similar to the pain in pneumonia caused by affection of the pleura, which is present to a greater or less degree in all cases. Pain, however, is not, as a rule, of long duration in myelitis, nor is it as severe as in spinal meningitis. A point of interest in regard to the line of demarkation of the sensory disturbance is that it is rarely a straight line across the body, but is usually oblique, which indicates that the lesion in the cord itself extends somewhat higher on one side than on the other. This is indeed confirmed by the pathological conditions found post-mortem.

Trophic Symptoms.—Besides the muscular atrophy we observe disturbances in the nutrition of the skin of a well-marked character, adding another very distinctive diagnostic point in myelitis. Bedsores almost invariably form within a week or earlier from the onset of the paralysis. They form at any point where there is the slightest pressure, and therefore are especially apt to be found over the sacrum and coccyx, over the trochanters and the heels, and not infrequently on the inner side of the knees when they have been allowed to press against each other. When the lesion is in the cervical region of the cord, we find them over the elbows and shoulders. They may even be found on the penis at the meatus when a catheter has been used and left in the urethra. The cause, however, is not the pressure, except in the sense of acting as a predisposing agent, for bedsores will form independently of pressure, as I have had occasion to observe in several cases, and are the direct result of the destruction of the nutritive centre of the skin in the cord. This centre is situated in the posterior horns, somewhat centrally and posteriorly. The character of the bedsore differs also from that observed from pressure in the various exhausting diseases, such as phthisis, typhoid fever, etc.: it is far more extensive, and is often irresponsive to all treatment, progressing steadily as long as the disease in the cord is still active and progressive. There is often a necrosis of all the tissues extending through the skin, tendinous sheaths, and muscles, and down to the bone itself. We have, therefore, the various stages of necrosis of tissues—a virtual gangrene of the parts. First, a slight redness or flushing of the skin takes place, which in a few hours passes on frequently to marked sloughing of the part, and finally the whole area is the seat of a black mass. This is associated with the odor characteristic of necrosis. With recovery these blackened sloughs finally come away, and under them we observe healthy granulations. In severe cases the sloughing of the tissues may be so extensive that a probe can be passed in all directions from the sacrum to the trochanters. The exhaustion accompanying this extensive destruction of tissue, associated as it is with a temperature ranging from 101° to 103° F., is one of the direct causes of a fatal issue.

Vasomotor Symptoms.—There is usually at first some elevation of the temperature of the paralyzed limbs; later this changes, and the limbs have a lower temperature than normal, and there is often present a cold, clammy sweat. Associated with this there is usually considerable œdema. In the chronic stages the skin becomes dry and harsh.

In cervical lesions there may be pupillary changes probably due to disease of the cervical sympathetic. Optic neuritis has also been observed in some cases. The intelligence remains conserved throughout the disease, and it is certainly a peculiar and characteristic picture to observe the comparative comfort of these patients, lying absolutely paralyzed, with incontinence of urine and extensive bedsores, the only functionally active parts being the centre of respiration and the cerebrum.

Complications and Sequelæ.—Under this head we shall speak of such complications as are really a part of the disease itself, but which are not always present in the same marked degree. Among the most common is cystitis. This is largely due to retention of some of the urine in the bladder. Despite the greatest care, the urine becomes

alkaline and undergoes decomposition. There is, at times, some disturbance of nutrition in the bladder wall itself, owing to disease of the spinal centre. A common cause of cystitis is infection due to the introduction of the catheter. The greatest caution should be observed in this regard, the catheter being thoroughly boiled after each operation. We may have extensive ulceration of the bladder itself, and extension of the inflammatory process to the kidneys or the peritoneum, or an abscess which may open into the peritoneal cavity, causing peritonitis (Gowers). Extensive bedsores, with the accompanying fever, lead to exhaustion and, at times, to secondary infection of the lungs, and there may be secondary renal complications, as amyloid degeneration of the kidneys.

The inflammatory process may be confined to the segment of the cord first involved, and reach its height in ten days or two weeks, or it may extend downward or upward. In the former case we recognize its progress by the increased atrophy of the muscles, which were at first only paralyzed; also by the extension of the anæsthesia and the loss of the reflexes. The same may be observed if it extends upward, so that if the respiratory centres are involved, death results.

Diagnosis.—Acute transverse myelitis may be confounded with several other diseases associated with disturbances of motion and sensation, such as multiple neuritis. This disease, indeed, until recent times was usually classed as a myelitis. We have in both paralysis of the extremities, associated with wasting, and coming on often more or less acutely. In neuritis, however, there is rarely any paralysis of the bladder and rectum, nor are bedsores present—certainly never extensive necrosis of the tissues, as observed in myelitis. The paralysis in neuritis almost invariably appears in the lower extremities, giving the characteristic foot-drop, and then extends to the upper extremities, involving the extensors of the wrist, with marked wrist-drop. We have, therefore, a different set of muscles involved, the paralysis being almost entirely confined to the extensors. As we have said, the paralysis in myelitis in the majority of cases is confined to the lower extremities, so that the clinical picture of paralysis of both upper and lower extremities should always suggest neuritis. The most marked diagnostic symptom is the presence of pain in the muscles when they are grasped, and along the course of the nerves when pressure is made. This may at times be exquisite in character, and is never present in myelitis. The absence of all mental symptoms in myelitis and their very common occurrence in alcoholic neuritis—the form we commonly have to deal with—is another diagnostic point. In the latter there are not infrequently delusions of sight and hearing and loss of knowledge of time and place. The tendency to recovery in neuritis, the contraction of the unparalyzed muscles, and the flaccid condition of the paralyzed muscles are important conditions to remember. The anæsthesia in neuritis is never complete, as in myelitis; there may be delayed sensation or paræsthesia, with areas of hyperæsthesia and anæsthesia, but never a well-defined line below which all sensation is lost.

Poliomyelitis distinguishes itself essentially by paralysis and wasting, and while, at times, both lower and upper extremities are simultaneously involved, it more often consists in a paralysis affecting

one lower extremity or one upper, or, again, one upper and the opposite lower extremity. The difference—especially important, however—is the absence of all sensory disturbances and of bedsores, or paralysis of the bladder and rectum. It is commonly also a disease of early life, although it may occur in adults.

Hysterical paralysis, when affecting the lower extremities, may be mistaken for myelitis. There is rarely, if ever, any wasting, nor do we find any change in the electrical reactions. As a rule, there is no distinct line of anæsthesia through any segment of the cord; that is, if anæsthesia is present, it is irregular in its distribution, and may involve a much larger area than that of the paralysis, or even be in the form of a hemianæsthesia. There is rarely paralysis of the bladder, although the mental condition may be such as to lead to loss of attention to the wants of nature, and consequently to involuntary micturition or to retention. There is an absence of bedsores. We must look for the special stigmata of hysteria, which have usually preceded the paralysis. In some cases of hysteria there may be extreme general wasting of the body, but in these cases there is often no actual paralysis, the weakness being due to the interference with nutrition.

Disseminated and central myelitis differentiate themselves from transverse myelitis by their localized symptoms. In the former we have focal symptoms, as paralysis of one arm and the opposite leg, or a hemiplegia of the Brown-Séquard type; that is, loss of motion and, perhaps, exaggerated reflexes on one side of the body and anæsthesia on the opposite side. A case of that character I have now under observation which, during the past two years, has terminated in the chronic form of disseminated myelitis, with secondary contractures and rigidity common to myelitis in general.

In central myelitis the symptoms are especially characterized by trophic changes, associated with atrophy of the muscles. Morton Prince reports two cases of central myelitis with dissociation of sensory symptoms, such as are observed in syringomyelia. The extent of the paralysis would depend on the extent of gray matter involved in the anterior horns, and the sensory disturbances on that of the posterior horns.

Hemorrhage into the cord would be characterized by the suddenness of the onset of the symptoms. There is often difficulty in diagnosis in these cases, for in myelitis, as we have seen, there may be extensive capillary extravasation. The initial symptoms in both instances run a similar course. They must necessarily be the same if the same areas of the cord are involved; in hemorrhage, however, there is not usually extension of the symptoms to any great degree, except from softening of the cord in the immediate neighborhood of the lesion. A history of traumatism followed by paralysis would render the diagnosis of hemorrhage almost certain where dislocation or fracture of the spinal vertebræ could be excluded.

Spinal meningitis differentiates itself almost absolutely from myelitis by the presence of pain in the back of a severe character, which is increased by any motion of the limbs which causes traction on the meninges. The pains also shoot down into the limbs in a radiating manner, beginning from the site of the lesion. The patient lies in

a position of flexion in order to relieve tension on the spine, and the muscles are in a state of rigid contraction. Unless paralysis results later from extension of the inflammation to the cord or from compression, there is no vesical or rectal paralysis, bedsores do not form, and there is no change in the electrical response of the muscles. The two conditions are especially characterized by excessive pain, with little paralysis, in meningitis, and by the absence of pain and the presence of paralysis in myelitis.

Prognosis.—Complete recovery in transverse myelitis is unusual; partial recovery is not rare, but more or less paraplegia remains. We usually find in these cases the evidence of the secondary changes which have occurred in the spinal cord—that is, the ascending degeneration of the posterior columns or sensory tracts—and therefore more or less ataxia of the parts above the seat of the lesion. More marked than the preceding are the signs of descending degeneration in the lateral tracts of the cord, or secondary lateral sclerosis, with the accompanying rigidity or spasticity of the muscles and exaggeration of the reflexes; in other words, the patient recovers with more or less well-defined spastic paraplegia. If the inflammatory process continues and there is no improvement for two or three months, especially if the vesical paralysis and the condition of the bedsores are extreme, the prognosis is unfavorable. An important symptom of the ending of the progress of the disease is the subsidence of the further extension of the bedsores and the beginning of healthy granulation. An early indication of improvement also is the partial return of sensation. There may be considerable improvement even after a year, although this is unusual. The recovery of course depends upon the actual amount of destruction of the structure of the cord. If this has been complete, there can be at no time a restoration. A fatal ending may take place, as has been said, as a result of the exhaustion from the bedsore or the presence of cystitis, with the ever-existing danger of its extension to the kidney. When long continued, as the temperature remains from 100° to 103° F., the prognosis must remain a doubtful one; necessarily also, if the lesion is in the cervical region, the prognosis is proportionately unfavorable, as extension may take place so as to involve the respiratory centres and cause death. The prognosis depends, therefore, upon the severity of the lesion. Slight inflammatory conditions, although symptomatically showing marked, or even absolute, paralysis and anæsthesia—even vesical paralysis and bedsores—may clear up rapidly and result in complete recovery. A characteristic instance of that form due to syphilis has recently been under my care, in which, after five months, complete return to power took place, with normal electrical reaction of the muscles, the only trace of the preceding disease being a slight increase of the reflexes. In cases which pass on to the chronic form of spastic paraplegia I think, with time, there is often a decrease of the rigidity and of the exaggeration of the deep reflexes.

Treatment.—At the onset of the disease, especially if it has been due to exposure, a hot bath, dry or wet cupping, or counter-irritation by means of blisters or the application of cold, may be of use, but after the acute stage has passed these agents act rather unfavorably. Great care should be taken not to interfere with the nutrition of the skin by

these means. Absolute rest should be enjoined, therefore, and as little disturbance of the patient by the above-mentioned measures should be allowed as possible. Ergot has been recommended at this time—1 or 2 drachms three times a day, or ergotin, 2 to 5 grains three times a day —and, although of doubtful effect, it may be given on general principles. Strychnine at this early stage is contraindicated. To maintain the nutrition of the muscles after the acute symptoms have passed away systematic massage and electricity are beneficial. When the paralysis is extreme and evidence of a destructive lesion of the cord exists, it is doubtful whether any form of counter-irritation can stay the progress of the inflammation, and it is advisable not to attempt it. As the disease progresses attention should be paid to maintenance of the general nutrition. The bladder should not be allowed to become distended, but should be periodically emptied by the catheter. In cystitis the bladder should be washed out with antiseptic solutions. The bedsores should be kept as free as possible from contact with the urine, which often, without great care, saturates the bed-clothing. Oakum serves the purpose of absorbing the discharge. Local stimulation in the early stages may prevent the extension of the sloughing. The best application is alcohol and water; later, iodoform with Peruvian balsam makes an effectual application, and the odor is somewhat controlled. As soon as improvement manifests itself or further progress of the disease has ceased, it is advisable to have the patient put in a reclining chair, thus giving change of position and relieving pressure from certain parts which must occur when lying in bed. At this period counter-irritation, such as the Paquelin cautery, blisters, and electricity, may be beneficial and hasten recovery. I do not believe electricity acts in any other way than to maintain the nutrition of the tissues by improving the circulation and maintaining the tonus of the muscles by bringing the individual muscles into action. When the rigidity is very marked I have found the galvanic current preferable to the faradic, and, as a rule, much more pleasant to the patient. The electricity should not be used except for the purpose of diagnosis so long as the inflammation is progressive—usually, therefore, not until the third or fourth week of the disease. Massage is very beneficial at this period also, both in its general and local effects on the paralyzed muscles. It is wise to combine with this, if possible, treatment at some one of the hot springs. Even cases of long-standing contracture may be benefited by this treatment, and greater freedom of motion attained, thus leading the patient to make greater effort on his own part to carry out some regular, though perhaps limited, course of exercises. All over-fatigue should be carefully avoided. Removal to a warm climate is also of advantage, as cold is apt to increase the rigidity. Even months after the onset of the disease I have seen marked improvement take place. While, of course, we recognize that the extent of recovery depends largely upon the degree of inflammatory destruction of the cord, nevertheless I have observed that in all cases which have been subjected to more or less manipulation and exercise the improvement has been decidedly greater than among those in which little effort has been made at systematized movement of the limbs conjoined with massage and hydrotherapy. The general nutrition, as has been remarked, should be carefully maintained

in the early stage of the disease. The bowels should be moved from time to time by enemata, as, owing to the paralysis of the sphincters, although the ordinary laxatives, such as aloin and cascara, are beneficial, there is a tendency to incomplete evacuation of the lower bowel.

In syphilitic cases I have used mercurial inunctions and iodide of potassium in large doses. When there is much restlessness and evidence of irritation of the posterior nerve roots, with spasmodic twitchings of the limbs and pain radiating into the extremities, I find that 20 grains of bromide of potassium and 10 grains of antipyrine give relief. Half a grain of codeine, combined with 5 grains of quinine, will control the temperature and produce a sense of rest and quiet.

DISSEMINATED MYELITIS (MULTIPLE MYELITIS).

Definition.—An acute condition which has been described by Eisemann, Leyden and Westphal, and others as not infrequently following the specific fevers—smallpox, typhus, etc.—but also occurring independently of these diseases. The principal diagnostic points are the acuteness of the onset of the disease, with symptoms resembling multiple sclerosis. Ebstein, in the post-mortem examination in a case of eight years' standing, found disseminated degeneration in the medulla and spinal cord. The characteristic symptoms of the disease (Westphal) are—(1) A peculiar disturbance of the speech of a slow scanning character; (2) ataxia of the extremities, with but slight motor paralysis; (3) absence of sensory disturbance; and (4) psychical disturbances, as shown by loss of memory, irritability, and dementia.

Etiology.—While, as has been said, the disease has often been observed following infectious fevers, it may occur independently without any known cause, or from syphilis and alcohol or exposure to cold.

Pathological Anatomy.—We find inflammatory processes scattered through the nervous system, involving, as in multiple sclerosis, the hemispheres, the pons, medulla, and spinal cord. They may involve the gray substance, but not as frequently as the white. There may exist the same condition as in transverse myelitis—the so-called red softening—dependent upon the degree of vascular disturbance. The bloodvessels are enlarged and filled with blood; the nerve fibres are swollen or they may have disappeared. The nerve cells are swollen, and later lose their processes and undergo degeneration. The condition is one, in fact, of a localized inflammation, and, unless the areas involved are large, the secondary degeneration is not marked. I have seen cases, however, in which the spinal lesion was more marked than the cerebral, or that of the medulla and pons, in which the secondary degeneration in the lateral tracts must have been considerable, judging from the spasticity of the muscles and the exaggeration of the reflexes. There is usually an increase of the neuroglia tissue. The final condition is that of multiple sclerosis (Leyden).

Symptoms.—The onset of the disease is acute; that is, within a day or a week there are marked tremor and ataxia of the extremities, which

usually involve all four extremities, but may be limited to one side. The head is also affected, and there are marked tremor of the tongue and, not infrequently, nystagmus. The speech interference is very marked; at times it is hardly intelligible, being slurring in character, or, again, explosive. There is often associated with these symptoms mental weakness, giving a foolish and somewhat expressionless appearance to the face. The patient is also easily excited emotionally, seeming to have lost the power of self-control and also of concentration. The deep reflexes may be much exaggerated, and later there may be marked spasticity of the muscles, with a tendency to contracture. There is usually also considerable muscular weakness; however, this never manifests itself so extremely as the inco-ordination and tremor. The bladder and rectum are not, as a rule, involved. Sensory disturbance is not prominently present. There may be some involvement of the cranial nerves, showing itself in deafness or optic neuritis. These symptoms may all disappear in a few weeks, but in most of the cases under my observation they have established themselves in a chronic form, resembling multiple sclerosis. I have observed also that the mental symptoms in the acute cases differ from multiple sclerosis in being more marked and passing somewhat rapidly into dementia.

A colored jockey who had received a severe fall came under my observation with symptoms of marked tremor, ataxia, nystagmus, stammering speech, and mental disturbance. He was unable to remember the day of the week. He was also very emotional, being easily excited to tears or laughter. He improved considerably in his spinal symptoms, and was finally discharged. Some few months afterward, however, I heard that he had been arrested as intoxicated, and later had been transferred to the insane asylum, where I saw him, and found him in a condition of complete dementia, although, unless excited, there was very little tremor or ataxia. These motor symptoms, in fact, were all increased on voluntary action, the patient lying quiet without tremor unless called upon to perform some act. The tremor is therefore of the intention variety.

Many cases present a somewhat different course from the above. There may be a paralysis of one extremity associated with some atrophy, which may undergo more or less complete recovery, to be followed by paralysis in another limb. Later there may be a more extensive lesion, involving both lower extremities and associated with exaggeration of the reflexes and contractures and rigidity of the muscles. Without a previous history of the manner of the onset of the disease—that is, of the paralysis of first one limb, more or less complete, with involvement later of either another limb on the opposite side or the same side—it would be impossible to distinguish it from a diffuse myelitis or a multiple sclerosis.

While in these cases the course is a protracted one and becomes chronic, the onset of each new development of the cord is acute in character.

COMPLICATIONS.—Under this head may be included the later mental development of dementia in many cases, or, again, the appearance of optic neuritis, or the gradual progression toward multiple sclerosis. Death does not directly result from the disease itself. In some cases,

which have more of the type of transverse myelitis, the bladder and rectum may be involved, thus adding to the complications.

Diagnosis.—Little need be added further on this subject to that which has already been stated. The most suggestive diagnosis on first examination of such a patient is multiple sclerosis. The great mental irritability and weakness, however, differ very much from that disease; the presence also of more paresis than is present in sclerosis is a marked differential point. Above all, the acuteness of the onset of the symptoms would seem the greatest point of difference. Leyden and Westphal have observed it also most frequently following the infectious diseases, and in such cases, therefore, at least we can feel more positive of the diagnosis. In the cases under my observation no such previous history was present, one apparently following an injury and another giving no apparent cause.

Prognosis.—In some cases we have a complete recession of all symptoms within a few weeks, ending, therefore, in recovery. It is possible, however, that this may not be as complete as would appear on first consideration, as it is probable that many of the cases of multiple sclerosis occurring in adults have been preceded by a multiple myelitis. The prognosis in general, therefore, is unfavorable, as, from what we have said, the usual tendency of the disease in its later stages is evidently toward multiple sclerosis. There is no immediate danger to life during the acute or chronic stages from the disease itself; the fatal result occurs, as a rule, through some intercurrent disease.

Treatment.—The same principles should be here observed as in acute myelitis—rest in bed for several weeks, and during the acute stage the use of ergot internally. No direct counter-irritation of any character is advisable. In the later stages massage, electricity, hydrotherapy, and the exhibition of the iodide of potassium and strychnine constitute the chief measures; the latter drug, however, should not be employed if the rigidity is marked.

CHRONIC MYELITIS.

Definition.—Chronic myelitis is a sclerosis of the spinal cord, usually the result of a preceding acute or subacute myelitis; that is, of a preceding inflammation, differing from the so-called systemic diseases of the cord, as tabes and lateral sclerosis, in which a previous inflammatory condition has not been present, but in which we have rather a degenerative process (Leyden). We shall speak, therefore, especially in this relation, of a disease of the cord which is usually a later stage of what we have already described as acute myelitis the result of the secondary degeneration. It may be primary.

Etiology.—From our preceding remarks it is evident that the usual cause of a chronic myelitis is an acute myelitis. In some cases we observe similar symptoms from a traumatism which has resulted in more or less destruction of the cord, and from hemorrhage, tumor, or caries. These conditions pathologically fall more closely under

the head of softening of the cord, and, according to Leyden, many years after the appearance of pressure symptoms from caries of the spine the evidence of softening of the substance of the cord is present, without, however, any true inflammatory changes. The disease may be caused also, as in acute myelitis, from extension of the lesion from surrounding tissues, as in meningitis, or primarily from cold, syphilis, etc.

Pathological Anatomy.—We find a more or less marked disappearance of both the nerve cells in the gray matter and of the nerve fibres, with an increase of the interstitial tissue and thickening of the walls of the vessels, with narrowing of their diameters. The membranes may also be involved, causing thickening of the pia, and the cord itself may appear to be much decreased in its diameter. Sclerosis, which is the characteristic pathological lesion of chronic myelitis, is present in both the ascending and descending tracts. This sclerosis may extend over months before its completion, following an acute myelitis; Leyden even ascribes a year before the final stages have been reached. On examination of the cord we find the posterior columns, extending from the site of the lesion, degenerating upward to the medulla, both posterior tracts being first involved at higher levels, the degeneration confining itself more nearly to the column of Goll, until, where the lesion has been situated in the lumbar region, we find the cervical degeneration limited to the posterior parts only of that tract—*i. e.* Goll's. The cerebellar tract and the column of Gowers show a like ascending degeneration (see Starr's plates, pp. 91, 92). The descending degeneration is confined to the lateral tracts of the cord, and extends throughout their whole lower length below the lesion.

Symptoms.—The symptoms are of a similar character to those already described in acute myelitis, but are less acute in their onset, and frequently less defined. At first the patient complains of some weakness, as a rule, in the lower extremities, accompanied usually by some rigidity and a loss of control, more or less absolute, over the bladder and rectum. It may, however, be months before a well-marked paralysis has developed. Loss of sensation is rarely complete. Various degrees of paræsthesia are present, with numbness and some tingling. As with the motor symptoms, this is often slow in development, and, although it may be extreme, as in acute myelitis, it is rarely so. The degree of atrophy of the muscles depends upon the extension of the lesion into the anterior horns, and is, as a rule, not a distinctive symptom. The reflexes become more exaggerated with the progress of the disease, and the spasticity and rigidity increase. With the extension of the secondary degenerations in the posterior columns we find a tendency to some ataxia, especially marked in the upper extremities. Bedsores may show themselves at a late stage of the disease or may appear suddenly in its course, as a result, probably, of some acute exacerbation of the inflammatory symptoms. There is rarely, if ever, any qualitative change in the electrical reaction, although there may be some loss in the quantitative response. However, at the site of the lesion we may have, as in acute myelitis, atrophy of the muscles, with the accompanying reaction of degeneration, or, again, a disturbance of sensibility, marked by hyperæsthesia at that point. The spasticity and

the contractures in some cases become extreme, the position of the patient being, as a rule, that of extreme flexion. These, however, can be usually overcome until the later stages of the disease by forcible traction. There may be more or less pain, associated with voluntary contraction of the muscles, cramp-like in character, which is the result of the accompanying meningeal inflammation.

Complications and Sequelæ.—The complications are really simply the later developments of the disease, and consist principally in cystitis as a result of the paralysis of the bladder; bedsores, often excited by the position in which the patient is necessarily confined in his bed, and not the direct result of the destruction of the trophic centre in the spinal cord, as is so frequently seen in the acute form of the disease. We may find an extension of the disease upward into the medulla, with resultant complications—in fact, the cranial nerves may be involved later, and in some cases optic neuritis and nystagmus may appear, giving thus at this period the appearance of a case of multiple sclerosis.

Diagnosis.—Chronic myelitis is most often confounded with a lateral sclerosis due to compression myelitis, as from a tumor, from an injury to the spine, or from caries. It would be rarely confounded with a chronic poliomyelitis, as in the latter there is the absence of rigidity of the muscles and exaggeration of the reflexes, as well as the absence of sensory or trophic disturbances.

The wasting in progressive muscular atrophy differs usually in its distribution from that of myelitis, and could only be confounded with that disease when the lesion is situated in the cervical segment of the cord. The absence also of spasticity of the muscles below and of exaggeration of the reflexes should make the diagnosis sufficiently plain. In amyotrophic lateral sclerosis, however, it might at times, in the beginning of the disease, be more difficult to make a diagnosis. The later course of the disease, however, with its distinct distribution of atrophy, primarily in the hands and extending to the arm, with the absence of all sensory disturbance, should aid us.

Meningitis, tumors, or caries should differentiate themselves, especially by the accompanying pain and rigidity of the back, from myelitis, the latter, indeed, usually running a course marked with little or no pain.

From multiple neuritis, running a chronic course, the differential diagnosis can be usually made by the absence of rigidity in the latter and the presence of decrease or loss of the reflexes; also by the absence of vesical or rectal paralysis. This disease, in fact, might more often be taken for tabes dorsalis than myelitis. The contractures which occur differ also from those of myelitis, being the result of shortening of the unparalyzed muscles in their unopposed action to the muscles affected, which, as a rule, have been those of the extensor groups.

Prognosis.—After the disease has been long established there is little that can be done. If the nerve fibres and cells have become degenerated, their restoration is impossible. With time, especially in cases which show little or no further progress, there seems to be a tendency to some slight relaxation in the degree of spasticity. The usual course extends over many years, with but slight tendency

toward improvement. The danger to life, however, is small except from exhaustion following cystitis or the bedsores. Extension of the disease upward may cause involvement of the respiratory centres, and thus cause death. A fatal issue is usually due to some intercurrent disease.

TREATMENT.—Rest in bed is to be recommended, as it often leads to a marked improvement, which usually goes on only to a certain degree. Later, hot baths, massage, electricity—anything, in other words, which improves the circulation of the limbs—is of advantage. Over-exertion at all times is to be avoided. Treatment at hot springs, conjoined with a warm climate, is often of great advantage. Where pain is present counter-irritation in the form of electricity, blisters, or the cautery is beneficial, but it is doubtful if this exerts any curative effect. Internally, the use of iodide of potassium is recommended, but little can be expected where the definite changes referred to in the lateral tracts have actually taken place. Where there is a specific history a thorough antisyphilitic course should be followed out, conjoined with treatment at hot springs.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS).

DEFINITION.—This is a disease characterized by an acute ascending paralysis, commencing in the lower extremities, extending to the trunk and upper extremities, and finally involving the muscles of respiration and of the heart. There is no disturbance of sensation nor atrophy of the paralyzed muscles. The electrical reactions remain normal and the sphincters are unimpaired. There may be loss of the deep and superficial reflexes. We have, therefore, if we accept this definition, to do with a disease differing in every respect from any other form of paralysis—with, indeed, an entity. In our study of its etiology and pathology we must hold this in mind, otherwise various other diseases, some atypical in their course, may be confounded with it. Thus, probably cases of multiple neuritis have been reported as Landry's paralysis.

ETIOLOGY.—Little can definitely be said in regard to its causation. The rapid onset of the disease and its course would seem to indicate that in the majority of cases there is some toxic agent present: what it is, however, we have not as yet discovered, nor can we positively state it as a fact. The disease follows, at times, as a result of exposure to cold, and has been observed in the infectious diseases, as typhoid, smallpox, diphtheria, etc. It has been confounded with acute cases of alcoholic neuritis. I should say that in my experience alcoholic cases rarely, if ever, are as sudden in their onset or as rapid in their course; probably alcoholism may act as a predisposing factor to Landry's paralysis, or indeed, in some cases, we may have the two conditions running a parallel course. It is a disease of adult life, occurring, it is generally reported, between the ages of twenty and fifty.

PATHOLOGY.—The lesions peculiar to this disease are not as yet

definitely known. Various writers have reported cases in which the changes seem to be essentially central; that is, confined to the substance of the spinal cord and the medulla. In these instances there has been usually observed much vascular disturbance, with some softening of the cord and extravasation of the blood from the capillaries, the gray substance being especially affected, resembling very closely the changes observed in poliomyelitis. Others, again, have recorded changes especially involving the peripheral nerves or nerve roots, with all the evidence of parenchymatous disease. Again, various bacilli have been observed. We have probably to do, therefore, with some poison which may involve both the central and the peripheral portions of the nervous system, affecting most profoundly the spinal cord if we may judge from its clinical course, and were the duration of the disease more protracted there would probably be more definite lesions present.

Symptoms.—The essential characteristic symptom is absolute paralysis, with usually flaccidity and loss of reflexes—with, however, an absence of all other symptoms; that is, of atrophy, anæsthesia, or hyperæsthesia, vesical or rectal paralysis, or electrical changes. The whole course of the disease may comprise but two or three days, death ensuing by rapidly ascending involvement of the centres of respiration, deglutition, etc. in the medulla oblongata. Its usual duration is at least a week, and it may end fatally after several weeks. Recoveries have also been reported.

The onset may have been preceded by some feelings of numbness and tingling in the lower extremities, accompanied by a sense of malaise. There is rarely any fever present. As stated, the paralysis usually commences in the legs, at times involving one before the other or both simultaneously. It extends then to the muscles of the trunk and thorax, and later to the upper extremities, finally affecting the tongue, lips, and palate, adding to the clinical picture dysphagia and disturbance of speech. In rare cases the muscles of the eyes are involved. While this is the typical course of the disease and the clinical picture which we must bear in mind in our diagnosis, there are at times certain variations to a limited degree, explained probably by the fact that in some cases the peripheral nerves have been also affected, or, again, that the lesion has especially involved the anterior horns in others. Thus we find, at times, some anæsthesia, or, again, there may be present loss of faradic response and the presence of the reaction of degeneration. We must always in these cases, unless the subsequent course of the disease corresponds with what has been previously said, remain somewhat in doubt as to the diagnosis.

Complications.—There is nothing to be said in regard to this subject further than what has been stated in describing the symptoms.

Diagnosis.—The disease might be confounded with diffuse myelitis, especially where some sensory disturbance is present. The absence of vesical paralysis, of bedsores, of wasting or atrophy of the muscles, and the rapidity of the progress of all the symptoms should, in most cases, enable us to differentiate it from that disease. It has more often been confounded with multiple neuritis, but here, as a rule, the wasting of the muscles, the sensory disturbances, as shown in the excessive pain along the course of the nerves and in the muscles

on pressure, with complete loss of reflexes, and even, in the most acute cases, the much slower progress of the disease, should be remembered. The later extension of the lesion in multiple neuritis, where it involves the muscles of respiration and of the heart, might render it difficult, from the similarity to the final stages of the disease in Landry's paralysis, to differentiate the two diseases.

TREATMENT.—Attention should be given from the beginning to stimulation, especially in sustaining the respiration, and to watching against heart failure. The special drugs, therefore, which are indicated are strychnine and digitalis. Nutrition should be kept at as high a point as possible. Any manipulation of the muscles by massage or electricity should be avoided.

POSTERIOR, PRIMARY LATERAL, AND POSTERO-LATERAL SPINAL SCLEROSIS.

By D. D. STEWART, M. D.

POSTERIOR SPINAL SCLEROSIS; LOCOMOTOR ATAXIA.

Synonyms.—Tabes dorsalis; Gray degeneration of the posterior columns of the spinal cord (Leyden); Duchenne's disease (Trousseau); Posterior leucomyelitis (Vulpian); Progressive spinal paralysis (Wunderlich); Progressive locomotor asynergia (Trousseau); Neuro-spinal tabes.

The term "tabes dorsalis" was common with Hippocratic authors several centuries before the Christian era. It was employed by them to represent a wide diversity of ailments, the chief characteristic of which was a general debility, most often dependent upon sexual excesses. In the early part of the present century, chiefly through the dissertation of Hahn,[1] the term began to be restricted to an affection of the spinal cord. Then for a number of years tabes was regarded to be dependent upon an atrophy of this structure, and to comprehend the various recognized forms of paraplegia, whether spinal or neural.

In Germany, Jacoby (1842) pointed out the probable localization of the lesions of locomotor ataxia in the posterior columns and nerve roots, and Steinhall (1844) published a well-marked case of the disease in which motor inco-ordination had been observed, and in which a necropsy revealed the lesion in the posterior columns of the cord. But to Todd of England unquestionably belongs the credit, still scantily accorded him, of having first recognized the true significance of the chief objective symptom of the disease, the inco-ordination, separating it from the pseudo-ataxy of motor impairment, and of inferring its association with disease of the posterior columns of the spinal cord. As a result of careful reasoning Todd was unwilling to accept the current view that the function of the posterior columns was sensory. He regarded the function of these as rather commissural between different cord segments, and as also subservient to the function of the cerebellum in regulating and co-ordinating voluntary movements. He noted that in the reported cases of disease of the posterior columns symptoms indicative of inco-ordination were present. In accordance with his view, he predicted in two cases of evident tabes that these portions of the cord would be found diseased, which prediction the necropsy verified.

Todd's view, however ingenious and leading as it did to the actual discovery of tabes, is not now accepted: the inco-ordination so conspicuous a feature of the disease is rather regarded as dependent upon affec-

[1] *De Tabe Dorsualis Prælusio.*

tion of the sensory muscle nerves or of the nerve roots in the cord. In 1851, Romberg, in the second edition of his *Lehrbuch der Neurenkrankheiten*, gave a quite accurate description of the disease, though failing, despite the published work of Todd, to distinguish between conditions in which ataxia and motor weakness were present. Romberg appropriated for the affection the term "tabes dorsalis," and describes intelligently the symptom now known as "Romberg's sign." Romberg noted also the frequent occurrence of pupillary changes and the occasional incidence of amblyopia, and quite accurately described the principal macroscopic lesions of the disease.

In 1858–59, Duchenne, in a series of able papers,[1] the fullest then published, gave a most accurate description of the disease, which to him, as to all of his countrymen, was still new. Aided by Trousseau in his famous *Clinical Lectures*, wide notice was taken of Duchenne's description, and the affection became known in France less by the title which the latter had designated than by that of his name—Duchenne's disease.

Definition.—Tabes dorsalis is a systemic disease of the spinal cord, and is of all affections of the cord the most frequent. It is a peculiar degenerative disease, having its seat in the sensory nervous system, affecting usually both cerebral (chiefly spinal) and peripheral portions, and causing, in a fully developed case, such symptoms as radiating pains, defective tactile, pain, and temperature sense; inco-ordination of voluntary movement; loss of muscle reflex action, notably the patellar tendon reflex; loss of the light reflex; and certain visual disorders. The earliest symptoms of the disease are usually radiating pains, loss of the patellar tendon reflex, and the presence of the Argyll-Robertson condition of the pupil.

Pathology and Pathological Anatomy.—There is no disease concerning which there is still so much conjecture and uncertainty as surrounds the pathology of locomotor ataxia. Concerning this question, Marie in his lectures quaintly remarks: "Fortunate are those who believe in the evidences of medical questions which are completely understood, and that tabes is one of them." In the earlier history of the disease, when the association of posterior sclerosis with ataxia was noted, it was supposed that the observed post-mortem changes in the spinal cord amply accounted for all of the symptoms of the disease. Subsequently, indications of involvement of peripheral sensory structures were often evident. This, with the fact that impairment of these alone, as in the case of multiple peripheral neuritis, with inco-ordination inseparable in character from that occurring in tabes, indicated that implication of other portions of the afferent tract than that included in the spinal cord could account for many of the symptoms of the disease.

Although the essential lesion of true tabes seems to be a sclerosis of the posterior columns of the cord, the one great fact of importance concerning its pathology is that so strongly emphasized by Gowers, that it is a neural degeneration of the sensory nervous system, both peripheral and central, the incidence of the disease being quite exclusively on afferent nerve structures. In tabes, in addition to sclerosis of the posterior columns of the cord and the nerve roots, and to degeneration of

[1] *De l'Ataxie Locomotrice Progressive.*

the peripheral sensory nerves, there occurs less often involvement of central cerebral structures, such as degeneration of the optic nerve and of the roots of the fifth nerve. Certain changes have also been noted in a few cases of tabes which may have been overlooked in many others, occurring in the cerebrum itself, probably but part and parcel of the same widespread tendency to degeneration due to a common cause.

Changes in the Spinal Cord.—*Macroscopic Appearances.*—In a well-marked case microscopic evidence is unnecessary to demonstrate gross changes. The spinal cord, especially in the lumbar and lower dorsal region, often presents a shrunken and thickened appearance in the vicinity of the posterior columns, with opacity and denseness of the membranes evident in the same region. The posterior nerve roots are usually visibly much wasted, being in marked contrast to their companions, the unaffected anterior root bundles.

On removal of the membranes the posterior part of the cord, chiefly in the lumbar region, exhibits a grayish, translucent hue. This may be evident the entire length of cord, and is due to the connective-tissue increase with destruction of nerve fibres. On section of the cord in advanced cases the grayish coloration will usually be found to extend into and occupy the greater portion of the region of the posterior columns. This grayish coloration is frequently much more apparent in the properly hardened specimen than in the fresh cord. In the former it may be noticed to extend from the lumbar region, in which it is usually most marked, occupying the whole of the posterior columns upward as a gradually decreasing tract until, in the upper dorsal and cervical region, it represents only the narrow column adjacent to the posterior median fissure. In less advanced cases the naked-eye changes are less evident and the microscope is essential for noting departure from the normal.

Microscopic Examination.—In hardened sections the sclerosed portions stain readily with carmine, and but slightly with osmic acid or by Weigert's method, the reverse of what normally occurs. Microscopically, with a low-power lens, this alteration in tinctorial power is noted to be due to the disappearance of nerve fibres in the affected area and the imbibition of the carmine by the increased connective tissue. Distinct zones of sclerosis may now be made out. In early or in slight cases, which of course come rarely under observation, with but moderate sclerosis in the area chiefly affected, involvement of the posterior external and posterior median columns may be apparent, separated by areas of healthy tissue. Then the sclerosis of the posterior external column (that of Burdach) as a rule occupies only the root zone.

At first but a slight thickening of the trabeculæ and their branches is evident. But in advanced cases the trabeculæ are abundant, thick, with fibrillation and nucleation of the connective tissue. Indications of increase in connective tissue are everywhere evident in the area most affected. The nerve fibres have largely disappeared when the process is advanced, although here and there may be seen isolated fibres narrowed and compressed with remains of axis cylinders. The blood-vessels of the affected parts usually are likewise sclerosed. The thickening affects sometimes the tunica media, but chiefly the outer coat and adventitial sheath, and leads to narrowing of the lumen of the vessel,

with extension of the connective-tissue increase into contiguous parts of the cord. At the very outset the portions adjacent to either side of the posterior horn, especially in the middle and posterior thirds (the external bandlets), are chiefly affected. This is always apparent in the lumbar region, and may be seen also in the dorsal, and subsequently in the cervical. The zone of Lissauer (extending between the apex of the posterior horn and the surface of the cord and a part of the posterior root zone) usually also early shows degeneration. Its sclerosis is commonly very marked in old cases. All the nerve fibres may then have disappeared. This becomes very apparent with the employment of Weigert's hæmotoxylin stain. The posterior median column (that of Goll) as a rule also shows early alteration, although only to a slight degree. In advanced cases of tabes, such as usually come to the post-mortem table, this portion of the cord is commonly affected throughout the lumbar, dorsal, and cervical parts of the cord. In the lumbar and lower dorsal region the whole width of the posterior columns extending from each posterior horn to the posterior commissure is apt to be completely sclerosed, with disappearance of the posterior median fissure. In some cases, however, as pointed out by Strümpell, although advanced, there remains unsclerosed even in the lumbar region a small fusiform tract. This, which is the so-called middle zone of Flechsig, is situated at the junction of the posterior with the median third of the posterior fissure. Another portion also of the posterior external column not often affected early, is the anterior part of the posterior column adjacent to the posterior commissure and to the anterior part of the base of the posterior horn. In the upper dorsal region and above, the sclerosis of the posterior external columns is commonly less evident, the degeneration continuing upward in the columns of Goll only, and presenting the usual appearances of a secondary ascending degeneration. Rarely, in very pronounced and long-continued cases, the degeneration may occupy the whole extent of the posterior columns of the entire length of the cord. The antero-lateral columns are usually unaffected, even in pronounced cases. Sometimes, however, there is connective-tissue increase here also, with involvement of the antero-lateral ascending tract (of Gowers) and of the direct cerebellar tract.[1] Involvement of these is far more common than is involvement of the pyramidal tract. In most cases, even early in the disease, there is probably at least some slight involvement of the gray matter of the posterior horns and commissure of the cord in the sclerotic process, although often not distinguishable. In advanced cases structural change is more noticeable. Degeneration is then often evident in the vesicular columns of Clarke, with atrophy or vacuolation of the nerve cells in the posterior commissure. The degeneration of the gray matter not infrequently extends into the region between the cornua. Thickening of the meninges, especially of the pia, is not uncommon in tabes. This may be limited to the posterior columns or may have extended entirely about the cord. Atrophy of

[1] These tracts transmit afferent impulses, That of Gowers is concerned in the conduction of pain sense. The exact function of the direct cerebellar tract is yet problematical. The trophic centres of this last tract exist in the nerve cells of Clarke's vesicular column. Involvement of the columns of Goll, occurring in tabes, is of the nature of a secondary degeneration, taking place probably only when the vesicular columns of Clarke are affected in the sclerotic process.

the nerve roots, appreciable even to the naked eye, almost invariably occurs in advanced cases, most apparent in the roots of the cauda equina and lumbar enlargement. These appear gray, thin, and shrunken, and microscopically show abundant evidences of degenerative change. It is an interesting and suggestive fact that this atrophy extends only to the ganglia on the posterior roots, the mixed nerve beyond showing, as a rule, no evidences of degeneration. As is well known, the nutrition of the nerve fibres in the posterior columns is controlled by the nerve cells in the posterior root ganglia, so that destruction of these ganglia or of the nerve root between the ganglia and the spinal cord promptly originates a secondary ascending degeneration, having a path through the posterior external columns of the segment connected with the affected nerve root, thence upward from the seat of the lesion in the postero-internal column (of Goll).

Changes in the Peripheral Nerves.—Degeneration of the peripheral spinal nerves is now regarded as of frequent occurrence in tabes. It is most common in the sensory branches, and affects either the peripheral nerve trunk or more usually the terminal filaments. The histologic change in the nerve is probably similar to that which affects the fibre in the posterior external column of the cord. The nerve fibre primarily wastes, with disappearance of axis cylinder and subsequent increase in interstitial tissue and nuclei. Opinions differ as to the part taken in the degenerative process of the motor nerves. It seems settled, however, that the changes are by far more common in the sensory branches only. The sensory fibres of the skin, joints, and muscles are all liable to be affected, but it is especially the fine fibres distributed to the muscles that are most involved. The implication of these last, which are especially concerned in the transmission of co-ordinating impulses from the muscles, is supposed to account for the ataxia.

Certain cerebral changes have been found in a number of cases of tabes, as before remarked. On these has been noted degeneration of nerve fibres of the convolutions, especially of the posterior lobes. They are similar to those encountered in paretic dementia, save in site of occurrence. The most probable explanation concerning their existence seems to be that they owe it to a common degenerative cause—that of syphilis.[1]

More common than alterations in the convolutions seems gray atrophy of the optic nerve with interstitial connective-tissue increase. Degeneration of the roots of the fifth nerve also is not uncommon, as are similar changes in the roots and nuclei of other cranial nerves, especially the third and sixth, and, more rarely, the olfactory and auditory. Degeneration of the trunk and nucleus of the vagus and of the glosso-pharyngeal has also occasionally been noted. Involvement of these is believed to explain the occurrence of the laryngeal and certain of the visceral crises.

Concerning the true nature of tabes much is still conjecture, and several divergent views of its pathology are strongly held. All the facts seem to point to the disease being primarily a sclerosis of the

[1] Jendrassik, who first recognized and describes these changes in the cerebrum, looks upon them as the most important lesion of the disease, to which the changes occurring in the cord are secondary. He is, however, quite alone in this view.

posterior columns. The only other view worthy of mention is that of Marie, who looks upon the disease as of the nature of an ascending degeneration, having origin in the cells of the ganglia of the posterior root and peripheral parts, with subsequent involvement of the posterior root fibres and the posterior columns. Marie holds that the nerve fibres contained in the posterior roots come from two distinct trophic centres —one constituted by the cells of the corresponding spinal ganglion, the other by a peripheral ganglion cell. This view, elaborated in Marie's work, is very plausible, but has less to recommend it than the more generally accepted one of primary degeneration in the sensory nerve fibres of the cord and peripheral nerves. Regarding the disease as primarily a sclerosis of the posterior columns, opinions differ whether the process starts as an interstitial inflammation, primarily in the neural or in the vascular connective tissue, with subsequent involvement and destruction of the nerve tubules, or whether the degenerative process primarily commences in the nerve elements themselves. While there is much to be said in favor of the first view,[1] the preponderance of evidence seems to be that in typical cases of posterior sclerosis the process almost invariably has origin in the nerve elements, the changes occurring in the connective tissue being secondary. As is remarked by Strümpell, the disease apparently always starts as a degenerative atrophy of the nerve fibres, and its distribution stands in closest relation to the physiological property of the nerve fibres. Hence certain systems always become easily and completely diseased, while others are more rarely, or if ever, but slightly affected. The order in which fibres are attacked varies somewhat, as does not infrequently its point of origin. As urged by Gowers, the fact that the accompanying degeneration found in the peripheral nerves generally starts in the nerve elements themselves, and that, even in the early stage, tabes shows characteristics of a systemic disease, affecting structures of common function in nerve and cord, strongly indicates the neural origin of the disease. This leaves little doubt, when the additional evidence pointed out by Gowers is considered, that there is every gradation between cases in which the perivascular disease is decided and those in which it is so minute as in no way to suggest primary interstitial inflammation. In all probability the disease is primarily of the nature of an irritative degeneration which is progressive and destructive in character, involving at first the nerve fibres of the cord and the peripheral sensory nerves, and is, as observed by Dana, not a simple wasting of nerve fibres, since it is accompanied by evidences of irritation, such as swelling of axis cylinders, and secondarily by proliferation of connective tissue and congestion.

Etiology.—There seems little doubt that we may regard tabes in the vast majority of cases as a degenerative sequel of syphilis. As is common in other diseases of the nervous system dependent upon syphilis, it is apparently rather the mild form of syphilis that precedes tabes than the severe forms. The initial lesion may have been so slight as almost to escape notice, and the secondary and usual tertiary symptoms, although

[1] Spitzka and a few others favor both opinions, believing that from the standpoint of origin two forms of posterior sclerosis occur, the one primarily parenchymatous, the other interstitial.

entirely without treatment, unapparent. This fact has been more than once remarked by competent observers, and is most significant.

This occurrence of tabes in subjects of past syphilis was first noted by Duchenne, Lancereaux, and Schultze, but the actual dependence of tabes on lues was first strongly brought forward by Fournier.[1]

Fournier some two decades ago asserted that he had not encountered a case in which the possibilities of a previous syphilitic infection could be excluded. As Fournier's field of observation lay quite exclusively among luetic subjects, his evidence was generally regarded as too *ex parte* to be of special clinical value; but continued observations to the present by the most competent neurologists, including, among a large number, Gowers, Erb, Strümpell, Rumpf, Althaus, and Sachs, leave little doubt as to the dependence of tabes on previous syphilis in so large a percentage that it must be accepted that this causative influence, however obscurely exerted, outranks far and away all others.

Fournier places the percentages of previous syphilis in tabetics observed by him as 98. Gowers, who views the ascertainable fact of previous syphilis in tabetics as quite below the real, can trace a causal proportion of 75 per cent. Erb[2] especially points out, in reply to the objections of Leyden, that there exists statistical proof showing that 80 to 90 per cent. of tabetic patients have previously had syphilis, whereas the percentage of tabes in the non-syphilitic is but 10 to 20: thus a relationship must exist between the two diseases. Erb noted antecedent syphilis in about 90 per cent. of his cases, stating that it is demonstrated that in the vast majority of cases tabes is a sequel of syphilis, and syphilis is by far the most important and the most common and the most potent etiological factor. Sachs's figures are still higher than Erb's. Strümpell[3] asserts he has yet to see a case in which the possibilities of a previous syphilitic infection could positively be excluded.

Notwithstanding, however, this apparent causal relation, true tabes is in no sense a syphilitic disease, but is probably, as pointed out by Strümpell, a degenerative sequel of lues, the result of a previous specific intoxication. Strümpell remarks that tabes is thus analogous to the palsies seen after diphtheria, which are the result of widespread nerve degeneration, having nothing histologically in common with the primary diphtheritic changes. He further states that nerve lesions in diphtheria are simply degenerative conditions due to the action of the chemical poisons developed in the body. Strümpell[4] believes that tabes arises similarly, and, as in the cases of other toxic degenerations of like character, there exists an intimate relation between the nature of the poison and the nerve tissue affected, each poison being a poison for only certain regions of nerve tissue and harmless for others. Thus it is understood why tabes must be a systemic affection.

Apart from the probably specific toxic influence of acquired syphilis on the development of tabes, we know little of other direct operating causes. Yet, although previous syphilitic infection is a probably causa-

[1] Fournier was the first to claim that tabes occurs almost exclusively in the subjects of past syphilis.

[2] "Zur Etiologie der Tabes," *Berl. klin. Woch.*, No. 29, 713, 1891; *Ibid.*, June 6, 1892.

[3] *Münch. med. Woch.*, 1890, No. 39.

[4] *Ibid.*, 1890, No. 39, p. 667.

tive factor of tabes, it is likewise established that in a small percentage of tabetics this may be excluded. Excluding a neurotic inheritance, which in certain instances, through transmitted vulnerability, seems to favor the development of the disease, the causative factors operative other than as a predisposition are uncertain. As concerns hereditary influences, the disease does not seem in any cases to be directly transmitted.[1]

As is well recognized in ailments other than tabes, an inherited predisposition to disease of the nervous system is very common, and this holds in locomotor ataxia, in which instances of a degenerative affection, such as hysteria, epilepsy, or insanity, in the immediate ancestry is very frequent. True tabes occurring in children of a tabetic seems always to be due to inherited syphilis. Charcot showed that diabetes is also, curiously, a frequent hereditary antecedent of hereditary tabes. A neurotic inheritance, however, can scarcely furnish more than a mere predisposition—such, for instance, as might be self-developed in post-natal life by various exhausting excesses. Fournier fancied that inherited syphilis might indeed be the prime etiological factor of the disease. This for various reasons seems improbable, chiefly from the age at which tabes is most frequent—one remote from that at which the latest lesions traceable to inherited syphilis usually assert themselves.

In the minority of cases not traceable to syphilis, tabes has been thought to be dependent (especially in a subject predisposed through constitutional weakness or neurotic heredity) on physical or mental abuse, but chiefly sexual excesses, or to be due to continued exposure to wet and cold, associated, perhaps, with privation or other debilitating influences. It has been thought occasionally due to direct spinal injury, such as might be produced by a fall from a horse. Gowers and others cite instances of direct injury being soon followed by symptoms of tabes. Marié, who is skeptical of all causes save lues, has noted cases attributable directly to injury in which symptoms were undoubtedly present prior to the supposed accident. What influences these so-called causes may have other than determining the development of the latent disease is yet problematical.

It seems possible, in the light of recent knowledge, that a toxic blood condition leading to the development of a disease allied to tabes clinically and pathologically may be originated other than by syphilis. Changes in the posterior columns of the cord have recently not infrequently been found in connection with pernicious anæmia and with diabetes, and, more rarely, similar changes in the cord have been recorded occurring in pulmonary tuberculosis, in Addison's disease, and in carcinoma of the stomach. The symptoms of cord involvement in such of these cases that are regarded as surely secondary to the existing non-nervous ailment are sometimes those of pure tabes,[2] the situation of the lesions found may be quite those of a systemic disease, such as tabes. Concerning histological character the post-mortem appearances have rarely been found similar in pernicious anæmia and in diabetes to those of tabes.[3]

[1] Excluding the special form of Friedreich's ataxia.

[2] Occasionally a differentiation may be almost impossible for a time (see Gruber's case: "Tabes or Diabetes Mellitus," *Neural. Centralblatt.*, Jan. 1, 1895).

[3] "The Spinal-cord Lesions and Symptoms of Pernicious Anæmia," C. W. Burr,

The comparative frequency of the occurrence of cord sclerosis in pernicious anæmia is most interesting. The occurrence also of a group of cases of cord sclerosis in non-luetic subjects poisoned by ergot, lead, and arsenic goes to show that a blood condition may be present independent of syphilis, which leads to sclerotic changes in the cord. It must be acknowledged, however, that except in rare instances, in which perhaps a luetic influence may have been overlooked, neither clinically nor pathologically are these cases identical with pure tabes. They are much more likely to fall into other groups, and especially to represent the mixed types of sclerosis.

Tabes usually develops in the middle period of life, between thirty and fifty, half the cases occurring between thirty and forty-five, one fourth between forty and fifty, and a somewhat less number between twenty and thirty. It rarely happens after fifty-five or under twenty. Cases have, however, been recorded occurring in young children. These seem always to be due to inherited syphilis, and are not instances of inherited tabes, the occurrence of which, apart from the special form of family ataxia, is regarded as problematical.

It seems not improbable that the age at which tabes in most cases appears is determined by the date of the preceding syphilis. This fact was noted by Erb, who pointed out that tabes may occur upward of twenty years succeeding the initial lesion of lues, but is most frequent between the sixth and fifteenth years. Thus it may be understood why tabes is most frequent between the ages of thirty and forty-five, since it is usually between the ages of twenty and thirty that syphilis is commonly contracted. Erb remarks also that with other than the usual date of infection the time of development of tabes also seems to vary directly. In 2 who contracted syphilis at a mature age tabes occurred in later life, and in 2 who became syphilitic at fifty-four and fifty-seven respectively tabes developed at the ages of fifty-nine and sixty-six years.

Tabes is more common in men than in women in the proportion of about 10 to 1. Gowers points out a similar proportion in general paresis of the insane, a disease related to tabes from more than one standpoint. As may be supposed, syphilis is apparently a similar factor in woman as in man. Of Erb's cases, lues was probable in about 90 per cent. of tabetic women. It is interesting that the disease is relatively more common in women in France than in England or in America. The cause of the comparative immunity of woman is undetermined. Probably it is due to the fact that women less frequently acquire syphilis, and, as a rule, are less subjected than the male sex to exposure and to debilitating physical and moral vices and to various excesses, such as venereal and alcoholic, that lower resistance and thus impart vulnerability. It is interesting in this particular, as Bramwell points out, that sexual excess acts more injuriously on the man than on the woman, and upon the lower than upon the upper part of the spinal mechanism.

Univ. Med. Mag., Apr., 1895; "Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anæmia," James Taylor, *Brit. Med. Journ.*, Mar. 30, 1895. Taylor states that the spinal-cord lesions in pernicious anæmia may be remarkably symmetrical. Williamson, *Brit. Med. Journ.*, Feb. 24, 1894: "Changes in the Posterior Columns of the Spinal Cord in Diabetes Mellitus." In a certain number of cases in these diseases the sclerotic changes were dependent upon small hemorrhages, such as occur in the retina in this disease.

Concerning the influence of race, it is worthy of note that tabes is rare among Hebrews, especially among the Russian Jews, a people who are regarded by certain authorities to be extremely liable to suffer from various other forms of nervous affections. It is believed that immunity in these is due solely to the slight prevalence of syphilis among them.

General Symptoms.—A typical case of tabes presents symptoms referable to derangement of the motor, sensory, and vasomotor nervous systems. Although not invariably, it tends to pursue a progressive course, unobtrusively at first, with but a few symptoms early evident, perhaps only lancinating pains and the loss of the knee jerk or the Argyll-Robertson pupil: one or all of these are commonly manifest when the case presents itself, with also, probably, slight ataxia. These ordinary indications of the disease may be preceded by such rare symptoms as optic atrophy and by various visceral crises, or by trophic changes in the skin, bones, and joints. These are more commonly followed—but perhaps not for years, during which only lancinating pains, loss of the knee jerk and of the pupil reflex to light are present—by disturbance of the genito-urinary functions and by ataxia, which last becomes more and more pronounced until it terminates after many years, should the patient survive, in the final or so-called paralytic stage.

Since the disease tends to pursue a more or less regular and progressive course, a typical case admits of division into stages. For accuracy and descriptive convenience an account of the disease will be so given here, a separation being made into the *preataxic*, the *ataxic*, and the final or so-called *paralytic* stage, in the last of which the incoordination has become so extensive that progression even with assistance is almost or quite impossible.

Preataxic Stage.—In the first or preataxic stage there is no distinct affection of gait, although there may be, and often is, present the so-called static ataxia—inability to stand with feet together and eyes closed. Symptoms in this stage are apt to be encountered referable to derangement in the sensory, motor, vasomotor, and trophic nervous system and in the special senses.

Sensory Symptoms.—Pains, usually severe and darting, lancinating or lightning-like in character, often form the earliest symptoms of tabes. These are present in the majority of cases, and may occasionally precede for years all other symptoms, even pupillary or patellar tendon reflex loss; but once evoked they usually remain throughout the course of the disease, although they may sometimes cease with the onset of marked ataxia. The pains in their incipiency may not be very obtrusive, and may at first be mistaken by the patient for rheumatism, especially if occurring, as is sometimes the case, in damp weather.[1] The pains are apt to be of spontaneous origin and of paroxysmal character, occurring occasionally at intervals regularly or irregularly and of variable duration. At first momentary twinges flashing through the part occur, separated perhaps by a long interval of freedom. Subsequently, with the progress of the disease, one dart succeeds another, until a paroxysm is made up of quickly occurring, innumerable lancinating pains of the most intense severity, continuing for hours, days, or

[1] Sometimes paroxysms seem induced by the approach of wet weather or through mental or physical over-tire.

perhaps weeks, with, again, an interval of entire or comparative freedom for days, weeks, or, less usually, months. These pains are often of the most agonizing severity, although their intensity has no relation to the gravity or duration of the disease. They are of remarkably sudden onset and disappearance, comparable in character of pain and duration to the sensation of a thrust of a knife or of a red-hot wire through the part affected. They are usually deep seated in the tissues or bones, having no relation to particular nerves, but may be felt also in limited areas of the skin, or corresponding with particular nerve trunks, such, for instance, as the sciatic or crural nerves. These last described pains, affecting the territory of particular nerves, are regarded as separable from the true lancinating pain and are of neuralgic character.

Pains are felt only in the parts enervated by the affected cord area, which corresponds usually at first to the lower portion of the trunk and the inferior extremities; subsequently, with the progress of the disease, they may occur in the upper extremities or head. In the rare cases in which the disease has its origin in the cervico-dorsal regions the pains occur, of course, first in the arms. They are often felt in the trunk and in the region of the bladder and the rectum, and may, when pronounced, be accompanied by a painful sense of constriction—the so-called girdle sensation or pain—felt about the lower dorsal or in the abdominal region in a limited or more widely diffused area. This same sense of constriction may be felt about one or more of the joints or may be noted in areas in the extremity apart from the joints. Pains in tabes may occur along the spine, in the loins, or, more rarely at first, in the shoulders. Here it is usually dull and aching in character, although perhaps more or less constantly present, but of no great severity unless the disease be accompanied by a spinal meningitis. Dull pains also occur in the lower extremities, and often long precede the development of the lancinating pains. Paroxysms of visceral pain are also sometimes present, as in certain of the so-called crises which will be referred to later.

The pains occurring in the skin are more apt to be of a scalding or burning than a lancinating character, and are frequently associated with marked hyperæsthesia (or more properly hyperalgesia) to touch in circumscribed areas. Paroxysms of lightning-like pain in the deeper structures are, however, often accompanied by superadjacent areas of cutaneous hyperæsthesia, in which a mere contact of the exquisitely tender affected area of skin by finger or by clothing induces an attack.

The lancinating and neuralgic pains of tabes are in all probability due to disease of the posterior root fibres in their passage through the posterior external columns of the cord, and perhaps also to disease of the peripheral nerve fibres. The pains are referred to the area of distribution of the nerves in accordance with the physiological law of eccentric projection.

Alteration in nutrition of the skin areas affected with hyperalgesia or with lancinating pain is not uncommon, leading to vascular disturbances, interference with the growth of hair, the appearance of herpetic eruptions, and the like.

In addition to the burning pain in the skin mentioned, the occurrence of small areas of hyperæsthesia in the lower extremities in the

first stage of tabes is not uncommon.[1] These, when not determined by the situation of the lancinating pains, are felt in the arch of the foot, about the malleoli and the knee. More common than the hyperæsthesia, and often succeeding it in areas previously over-sensitive, and frequently, too, so existing in adjacent skin areas, is anæsthesia. This, which affects not only the cutaneous structures, but the deeper parts, and which is often accompanied by a similar insensibility to pain, is not apt to be decided in the early stages of tabes, so that a full description is left until the consideration of the second or ataxic stage of the disease. Anæsthesia of the plantar surfaces of the foot, toes, and heel may be early encountered, with paræsthetic sensations, described by the patient in such words as "pins and needles," "creeping," "crawling," and "tingling" in the legs. Sensations of extreme heat or cold in the feet are also common.

Motor Derangements.—Perhaps the first symptom attracting the patient's attention to his ailment is the condition known as Romberg's sign, or static ataxia, manifested chiefly by inability to stand well when the eyes are closed, with the inner borders of the feet in juxtaposition. The appearance of this symptom has often been first noticed by the tabetic in washing his face over a basin, the eyes being covered momentarily with hands or towel; or unsteadiness has been felt in rising suddenly or in turning or in walking in the dark. Swaying then occurring may be thought by the patient to be due to vertigo, also a not uncommon symptom of early tabes, dependent perhaps upon diplopia.

A consideration of static ataxia will be found with the description of the second or ataxic stage of the disease.

Transitory paresis of muscle groups occasionally occurs in the first stage of tabes. It is difficult of explanation pathologically.

Loss of the patellar tendon reflex, or the knee jerk, as was first noted by Westphal, is usually the earliest and most important symptom of the disease. When actually absent, with coincident loss of the pupil light reflex or with the presence of lancinating pains, the certainty of tabes is beyond question. The knee jerk rarely persists in an undoubted case with the presence of the one or the other of these symptoms.[2] Occasionally, however, this reflex still exists on one or the other or both sides, but greatly enfeebled, or it may be only evoked, and then but to slight degree, by the greatest nicety of technique, even by methods of reinforcement which usually are most productive. Commonly in such cases the knee jerk subsequently disappears with the progress of the disease.

It is important to observe that very exceptionally the knee jerk may be absent in perfect health. This fact, which I have seen nowhere emphasized, is in my own experience undoubted. One instance is that of a robust young man who seventeen years ago, while a student of medicine, discovered its absence in himself. This loss was confirmed by certain of his teachers whom he consulted. Seven years subsequently I examined him on many occasions, but always with the same result. He has remained in good health, without indications of any ailment

[1] When pronounced—*i. e.* of general distribution—hyperalgesia, as pointed out by Erb, indicates an accompanying meningitic complication.

[2] This of course refers to tabes with the lesion occurring in the common situation, in which the special afferent path of reflex irritability is susceptible of extinction.

which might affect a part of the reflex arc. It is interesting in this case, knowing the great significance attached to this symptom, that the gentleman, although he had not had syphilis, developed a morbid dread of falling a victim to tabes, and for several years early in our acquaintance, in consequence of occasional slight rheumatic pains appearing, fell into a deplorable mental condition, becoming so melancholic as to require a long sojourn abroad. I have encountered several other instances of persistent absence of the knee jerk in the healthy. Of the cases carefully investigated, one is a physician. In making a study of the knee jerk some years since at the Jefferson Medical College, and systematically examining a large body of students, I found, among several with sluggish patellar tendon reflex, but one in which it was totally absent even to reinforcement. This man was in perfect health. He afterward informed me that a similar condition existed in at least two members of his family, the only near kinspeople he was able to examine. I was unable personally to confirm this statement, as these cases lived in another city. These cases, of which, as remarked, I have met other instances, clearly indicate that the knee jerk may be occasionally absent in health, but they likewise as clearly show so extreme a rarity of this absence, representing as they do so minute a fraction of loss, as to be quite insignificant and not to materially detract from the value of the sign.

As the mode of eliciting the knee jerk is of the utmost importance, and inattention to technique has often caused it to be thought absent when otherwise it might readily have been obtained, a few words concerning this are pertinent. The one important point to have in mind is that when the tendon is percussed both the extensor and flexor muscles of the leg tested should be completely relaxed, or at least as free from voluntary contraction as possible, without which even in other than stout persons it will be impossible to obtain result. In the usual method of examination the patient sits on a chair with the leg of the patellar tendon to be percussed thrown across its fellow, which latter is bent at a right angle. If the patient is stout, relaxation of leg-muscle tension is sometimes impossible in the above-mentioned position: then the method may be tried of supporting the leg to be tested by the arm of the observer passed beneath the thigh just above the knee and resting the hand on the companion knee. Another plan is to place the subject on an elevated seat, as upon a table, so that the feet are above support and the hollow of the knee is in juxtaposition to the seat's edge. This method is especially applicable in children. Whichever of these postures is chosen, the patellar-tendon should be sharply struck,[1] but in such a manner that no jarring of the leg tested is produced by the blow itself, which otherwise might be supposed by the tyro to be a slight reflex contraction. The blow, which is effected either, as is commonly done, by the ulna border of the right hand, or preferably by a percussion hammer (of which the best is that designed by J. M. Taylor of Philadelphia), should strike the middle part of the tendon. Should the reflex arc be free from disease and no mechanical difficulty interfere, usually an active contraction of the quadriceps is so produced, leading

[1] If the reflex is sluggish, a sharp blow, with induced reinforcement, may alone evoke it.

to a quick forward jerk of the leg, with an immediate fall. It is often absolutely essential, especially in women, to bare the tendon struck and have the leg tested quite free from constricting clothes. Should no reaction be obtained by one of the methods mentioned, the following will be found efficient should the knee jerk, although extremely sluggish, not be absent. The patient sits naturally with the feet upon the floor, the knee flexed at a right angle, and the thigh muscles free from voluntary contraction. The quadriceps extensor group is grasped with the left hand in such a manner that the slightest quiver of contraction may be readily communicated to the hand, although perhaps inappreciable to the eye even with the leg bared. Complete uncovering of the leg is not essential, although by it it is interesting to note simultaneously with the eye the slight contraction appreciable to the hand. I have used this method in preference to any other for a number of years.

The patient's attention being diverted for an instant, the tendon of the knee is then sharply struck, either with the ulna border of the hand or, preferably, with the percussion hammer. If no contraction is to be readily obtained by whichever of the methods employed—of which this last described is far and away the most exact—the method of Jendrassik or some of its modifications of reinforcing inappreciable movement should be tried.

In this the patient is told to perform some voluntary muscular effort not involving the action of the leg muscle when the patellar tendon is percussed. The fingers of the two hands may be interlocked in such a way that separation is not readily affected when traction is made on the hands; or the fore fingers of both hands may be placed in the claw position, and, on being so interlocked, pulled against. This voluntary muscular effort is supposed to either heighten muscular tone throughout the body by increasing generally the radiation of nerve energy, and thus to intensify response to reflex stimulation, or to synergize reflex action entirely through removal of cerebral control. J. S. Risien of London has recently[1] shown that mental processes, such as adding figures together, dividing and multiplying them without the aid of writing, reinforce the knee jerk much more sharply than physical effort. This strongly corroborates the view he ingeniously supports that reinforcement is in reality a removal of cerebral inhibition.

Should motor reinforcement not succeed in eliciting the knee jerk—and it may fail through the natural awkwardness of the subject, resulting in interfering contraction of the posterior thigh muscles—some form of mental reinforcement should be tried, such as having the patient count the numerals forward and backward, multiply figures, etc.

Should no knee jerk be thus obtained, it must be considered absent and other symptoms of tabes carefully searched for.

Cause of the Loss of the Patellar Tendon Reflex in Tabes.—This loss is effected by disease in some part of the afferent or sensory tract. It is not yet settled whether this interruption occurs peripherally in the sensory and tendon nerves (which have often been found the seat of disease in tabes) or in the posterior nerve roots or in the posterior external columns of the cord. The seat of interruption, especially in the early stage of tabes, may vary. It is, however, very probable

[1] *Amer. Journ. Med. Sci.*, Mar., 1896.

that disease of the peripheral muscle nerves alone is often the cause of the loss.

The cutaneous or superficial reflexes in the early stage of tabes, are sometimes much heightened. Subsequently, with the appearance of cutaneous anæsthesia, the superficial reflexes disappear, the plantar first becoming lost, followed by a cessation of the gluteal, cremasteric, and abdominal reflexes. Derangements of the sexual, bladder, and rectal reflexes are all common in the first stages of tabes.

Loss of sexual desire and power is common in the disease, and often early in its history. Occasionally a marked heightening of the sexual reflex occurs, in which there is extreme desire with little ability for gratification.

Bramwell[1] mentions a case in which both desire and power existed in an extraordinary degree. Attacks of paroxysmal satyriasis occurred, in which the sexual act was performed four or five times in twenty-four hours for a number of consecutive days. Each paroxysm lasted several days, and was succeeded by a period of complete sexual apathy, with subsequent curious return.

Derangement of the bladder and rectum is often manifested by frequent urination, the act being painful in the extreme, accompanied by pelvic, perineal, and urethral pains or twinges in the neck of the bladder. Paroxysms of frequent and painful urination may sometimes occur, somewhat analogous to the radiating pains manifested in the legs.

In a later stage, with the persistence in the urinary difficulty, anæsthesia of the mucous membrane of the bladder, with loss of its expulsive power, is common. Such tolerance may be acquired that the patient may remain a day or longer without desire to urinate. In other cases weakness of the sphincter, at first slight, causes a condition of incontinence, especially for a short time after urinating. In a later stage, through paresis of the sphincter, persistent incontinence results.

Constipation is somewhat common in tabes, and rectal tenesmus is often noted in the early stage, with occasional involuntary evacuation of fæces. This last is more usual in the later stage, when there has occurred complete anæsthesia of the rectum.

Among the early and important symptoms for which search should be made, none is more suggestive than reflex iridoplegia, or the Argyll-Robertson[2] condition of the pupil, present in about 90 per cent. of the cases. This consists in abolition of the pupillary reflex to light, while until late in the disease associated contractions to accommodation are retained, with vision unaffected. It is generally accompanied by double myosis, which in turn is associated with loss of reflex dilatation on stimulation of the skin,[3] a symptom less important diagnostically than the first named.

[1] *Diseases of the Spinal Cord.*

[2] Marie states that this as an early sign of tabes was in reality first described by Vincent and Coingt, and not by Argyll-Robertson, although now known by the latter's name.

[3] Sharp stimulation of the skin in any part of the body normally is followed by momentary reflex dilatation of the pupils. This reaction, as shown by Erb, fails in many cases of locomotor ataxia, especially when myosis is present.

In testing for reflex iridoplegia it is important that a strong diffuse light be used. As suggested by Gowers, when artificial light is employed it is best to have the patient view an object in a distant part of the room, and then bring a light suddenly in front of the eye. The light must not be directly looked at, unless it is held at least 4 feet from the eye; otherwise accommodation, in fixing the object, may result in pupillary contraction. If the light is viewed near the eye, the other eye must be so shaded as to fix a distant point and maintain relaxation of accommodation. Each eye should be separately tested, with its fellow covered from the light employed.

The loss of the light reflex in tabes is in all probability due to disease situated in the fibres connecting the corpora quadrigemina with the nucleus of the third nerve. The preservation of accommodative power and of sight in reflex iridoplegia indicates that the afferent and efferent part of the reflex arc are unimpaired.

In cutaneous reflex iridoplegia (loss of the dilating power) the lesion is probably situated in the efferent fibres of the centre for dilatation in their descending course through the cervical portion of the cord.

Instead of myosis, more rarely mydriasis may be present in tabes. Inequality of the pupils is very common, with perhaps mydriasis of one and myosis of the other. In myosis the pupil may be very small, with sight unaffected. With inequality of the pupil it has been noted that on the side in which myosis was most marked there was present indication of paresis of the sympathetic, such as redness of the cheek and conjunctiva, local elevation of temperature, and diminution of the palpebral fissure, and in a few cases unilateral swelling (Ross). One or both of the pupils may be also irregular in outline.

In about 20 per cent. of the cases of tabes, amaurosis, ending in complete blindness, due to double gray atrophy of the optic nerve,[1] occurs, and is an early symptom. A gradual diminution in the visual acuity, with concentric restriction of the field, at first for white and colors (especially green and red), and secondarily for the recognition of objects, occurs. Blindness may appear in a few weeks or may be delayed for years. The average period between the onset of symptoms of atrophy was noted by Berger to be three years, with variations occurring from two months to seventeen years. The cases of tabes with optic atrophy seem to constitute a special form in which the disease appears to expend itself on the organs of vision, leaving symptoms referable to the lower part of the cord (such as inco-ordination and the characteristic pains, but especially the former), if already existing, in abeyance.

Déjerine[2] has recently directed especial attention to the forms of tabes associated with blindness, and has compared cases of the sort with the common classical type. His comments are most interesting and deserve notice. He speaks of three forms of tabes in which amaurosis occurs:

"(1) In by far the largest class the optic atrophy supervenes at a period longer or shorter after the appearance of lightning pain, and

[1] Which, opthalmoscopically, has all the appearances of a true, primary degenerative atrophy, and not that the result of neuritis.

[2] *La Méd. mod.*, Mar. 20, 1895, quoted in *Brit. Med. Journ.*, May 18, 1895.

inco-ordination never appears. In these cases as the blindness is being established the pains diminish in frequency and severity.

"(2) In the second variety the amblyopia appears simultaneously with, instead of shortly after, the lightning pains, and the tabes develops with the blindness, stopping when the latter is complete.

"(3) In a few cases blindness exists without pains. Such cases are mostly seen by ophthalmologists. In them the knee jerks have disappeared, whilst, as regards the pupil, loss of the reflexes to light exists, and that for convergence is preserved. Gowers has pointed out that lightning pains appear if the cases are sufficiently long observed. No satisfactory explanation yet exists, Déjerine thinks, of the antagonistic action of optic atrophy on the symptoms of tabes. In almost all cases the pains diminish considerably, and sometimes even disappear. Once the author saw the patellar reflex reappear after an abolition of several months. In the rare cases in which blindness supervenes on inco-ordination no improvement of the latter takes place, though the pains amend. The features of the optic atrophy are—(1) It attacks the eyes successively; (2) it is rapid in its course—six to eighteen months; (3) pupillar inequality, myosis, mydriasis, or normality indifferently exist; (4) though due to syphilis, treatment is of no avail. Romberg's sign is absent in these cases and the gait is easy. Sensibility in its various forms is unaffected."

Unilateral or bilateral temporary (often but ephemeral, and not showing a recurring tendency) paralysis of the external ocular muscles is very common in the preataxic stage, causing, if slight, double vision, or, if more decided, strabismus. The external rectus is most frequently involved, following which are the superior and inferior recti and the superior oblique. Rarely all the external and internal muscles of the eyeball are affected, causing ophthalmoplegia externa and interna, suggesting an associated nuclear lesion of luetic origin. Unilateral and less frequently bilateral ptosis, associated with external strabismus, is also frequent in the preataxic stage of tabes, as is a slight narrowing of the palpebral fissure. In a considerable percentage of tabetics implication of the auditory apparatus occurs, due to a unilateral atrophy of the auditory nerves similar in character to that affecting the optic nerve. In these deafness may be of slowly progressive onset or occur quite rapidly.

Disorder of the sense of smell, and less often absolute anosmia, sometimes occur in tabes, due also, perhaps, to atrophy of the olfactory nerve special to tabes.

Involvement of the trifacial nerve, indicated by neuralgiform or lancinating pains in the area of distribution of the nerves, may also be looked for in tabes. These, when occurring in paroxysms without other symptoms of tabes very evident, may be mistaken for ordinary hemicrania. Accompanying the attack there may be conjunctival injection, increased lachrymation, and photophobia. Cutaneous hyperæsthesia succeeds an attack. Later in the disease anæsthesia occurs, occupying the region of the conjunctivæ, nasal fossæ, and also perhaps the mouth, tongue, and pharynx (Ross). The motor branch of the trifacial is less frequently involved than the sensory, leading to partial or more complete paralysis of the muscles of mastication.

Transitory indications of irritation or of paralysis of the facial nerves, unilateral or both-sided, may occur, leading, on attempts of phonation, to grimaces and to ataxic or indistinct speech.

Tabetic Crises.—These, when present, are frequently among the prodromal symptoms of the disease. The most common and interesting is that affecting the stomach. In this attacks of the most violent gastralgia occur, the pains of which may be of agonizing severity. With it there is prolonged and uncontrollable and prostrating vomiting. The gastric crisis may be associated with lancinating pains in the extremities, and occur during a paroxysm of pains or appear with these absent. The attacks continue for a time varying from that of a few hours to several days, with an interval of a week to a month. They are of precipitate onset, and are usually totally without premonitory symptoms or assignable cause, and tend to abrupt subsidence. During the paroxysm constant and prostrating retching and vomiting occur. Vertigo is common. Food or drink, even in the smallest quantity, cannot be taken. The result of an examination of the vomited matter in a number of cases indicates that there is usually during the seizure hypersecretion of the gastric juice with hyperchlorhydria. This, however, cannot be expected to be an invariable accompaniment, since the attacks are not dependent upon a local cause, but upon either a systemic intoxication or upon disease of the vagus or its nucleus.

Pharyngeal Crises.—Attention to the occurrence of these has in late years been called by Oppenheim.[1] There is much pharyngeal pain, congestion of the face, and profuse perspiration, accompanying a series of frequent repeated efforts at deglutition, the attack lasting ten to thirty minutes.

Intestinal crises are less common than the gastric. These consist in attacks of sudden and violent colic, often concomitant with profuse watery, mucoid diarrhœa, perhaps mixed with blood.

Renal crises have been occasionally noted in tabes. In these symptoms occur resembling the passage of a calculus, save no blood appears in the urine and no calculus is voided.

Crises resembling *hepatic colic* are also among the less frequent symptoms of tabes.

Laryngeal crises are of grave but of somewhat rare occurrence in tabes. These consist of paroxysms of dry, violent cough, resembling that of pertussis, due to spasmodic contractions of the laryngeal muscles. In such a crisis there is urgent dyspnœa, burning pain in the neck and upper part of the spine and shoulders, and perhaps vertigo. Prostration, and even syncope, are common. Sudden death has occurred in an attack.

So-called *cardiac crises* are occasionally seen in tabes. These are, however, in most cases, apparently but examples of true angina pectoris complicating the tabetic condition, and perhaps owing its existence to disease of the coronary arteries or to another common cause of the disease. It is interesting that in tabes aortic regurgitation is not unusual, although perhaps often overlooked. It probably is dependent upon syphilitic disease of the aorta and its valve leaflets. Attacks of angina in tabes occurring in subjects of aortic regurgitation are in all

[1] "Neue Beiträge zur Path. der Tabes dorsalis," *Arch. f. Psych.*, xx., 1888.

probability intimately relative to the coincident aortic disease, as is usual independent of tabes. Tachycardia independent of anginal attacks occurs in tabes. Special attention was long ago called to this by Charcot, who found a pulse of 100 to 120 usual in his cases.

Trophic and Vasomotor Disturbances in Tabes.—*Trophic Disorders.*—These, although comparatively rare in tabes, when present are among the most interesting of the early symptoms of the disease. The most important of the trophic disorders are changes in the nutrition of the bones and joints, perforating ulcer of the foot, falling out of the teeth and nails, and the appearance of certain cutaneous eruptions. Single or multiple so-called "spontaneous" fractures of the bones, appearing painlessly and apparently causelessly, or from violence most slight in proportion to the amount of injury, may occur very early in tabes.

Through disturbances in enervation the nutrition of the bones suffers, and they may become abnormally friable. These fractures are more common in women, and most frequent in the bones of the lower extremity, more especially the bones most exposed, such as the femur, its shaft and neck, and the tibia and fibula, although they may occur in any part. The slightest cause often leads to the fracture, such as merely crossing the legs while sitting or the act of pulling on the boots. It is quite common for no traumatic cause to be apparent. As remarked, the fractures are painless. The fractured ends of the bones unite readily enough if maintained in position, which last is not always easy, because of the freedom from pain tempting the patient to use the limb involved. Chiefly because of the inability to maintain the fractured ends in position, a large amount of callus is usual and shortening of the limb is common. Fracture of the bones of the upper extremity, such as the humerus, the radius and ulna, the clavicle and scapula, also occur when the disease affects the upper segment of the cord. Fracture of some of the bodies of the vertebra or of their arches or processes is occasionally encountered, causing curvature of the spine. It is usually associated with arthropathy of the intervertebral articulations. Associated with spontaneous fracture of the bones there is, more rarely, a tendency, also present in these cases, to rupture of the tendons, such as the tendo Achilles or of the quadriceps extensor cruris.

The most extraordinary trophic affection in tabes is that involving the joints, and is known as "arthropathies des ataxiques" of Charcot.[1] These, which are of much more frequent occurrence than fractures of the bones, usually appear in the early stage of tabes, involving at first the joints of the lower extremities. They most commonly assert themselves abruptly and during very severe paroxysms of lancinating pains. They are curiously, as with the crises and the optic atrophy, more common in the preataxic stage and in those cases in which typical fully developed tabes does not occur. The affection, as with spontaneous fracture of the bones described, is usually of painless and rapid onset. There may have been creaking of the joint for a few days preceding, or the first noticeable symptom may be pronounced swelling about the joint. The knee-joint is usually first involved; following which the hip-, shoulder-, and elbow-joints may be attacked. Here, as with the other joints, the whole seg-

[1] Tabetic arthropathy was first recognized and described by Charcot in 1868.

ment of the limb is generally implicated. The swelling is usually firm, unyielding, and painless to pressure, and shows no indication of being of inflammatory character. The skin about the part is pale, and heat and pain, as mentioned previously, are absent. Charcot pointed out two forms of the affection: one the mild, in which the effusion vanishes in a short time, leaving the joint, apart from the continuation of crepitations apparently normal. In these mild forms relapses are common. In the other or more serious forms the œdema attains considerable size and is permanent. Changes in the articular ends of the bone and in the inter-articular fibro-cartilages rapidly and painlessly occur, similar in pathological character to those of rheumatoid arthritis, and differing clinically, chiefly in abruptness of appearance and in absence of pain. The disease may have onset in bone, cartilage, or ligaments—soon all these parts become involved, the joint surfaces destroyed, and the bones dislocated. Very interesting characteristics of tabetic arthropathy are, after a time, the extreme mobility and curious deformity of the affected joints, induced and perpetuated through the influence of the muscular contractions upon the joints when subjected to the various movements necessary for locomotion and fair station after inco-ordination has occurred.

One of the most interesting arthropathies of the disease is the so-called tabetic foot described by Charcot and Féré, in which, through a trophic lesion in the tarsal and metatarsal bones, the foot becomes suddenly swollen, chiefly in the part near the tarso-metatarsal articulation. As a result the inner foot border becomes thickened and rounded, and the arch of the foot flattened or even convex; shortening of the foot occurs in some cases. If the swelling of the involved joint surfaces subsides, as it may after a long time, complete and persistent ankylosis is usually evident.

Trophic Affections of the Skin.—The most common trophic disorders occurring in tabes affect the cutaneous surfaces. These are various eruptions, notably erythematous, urticarial, lichenoid, and herpetic, which are common on the affected parts during paroxysms of lancinating pain. The nails, especially of the large toes, may suddenly become detached, renew themselves, and again repeatedly appear and be re-shed; perforating ulcer of the foot is a very important trophic affection of the early stage of tabes. It usually has origin as a corn which subsequently ulcerates centrally. The ulceration may soon heal while still superficial, or it may extend into the deeper structures, involving the articulations and bones. The usual seat of perforating ulcer is about the head of the first metatarsal bone or it may be below the heel. It is most apt to occur at the situation at which the greatest amount of pressure is exerted by the footwear. Anæsthesia usually exists above the site of the previous corn, indicating that the perforating ulcer is due to changes in the nutrition of the skin due to alteration in the cutaneous nerves. Loss of the teeth without previous caries also occurs in some cases of tabes, due, probably, to a trophic disorder involving the fifth nerve. One or more, or indeed all, of the teeth may gradually become loosened and fall out. In these cases atrophy of the alveolar margin of the jaw has been observed, with atrophy also of the crown of the teeth.

The causes of the trophic and vasomotor disorders of tabes are in all likelihood implications in the degenerative processes of certain por-

tions of the gray matter of the cord (central and intermediate gray matter and the vesicular column of Clarke, situated at the junction of the posterior horn with the intermediate gray matter), in which lie the vasomoter centres controlling the nutrition of the bones, joints, and skin.

Symptoms of the Ataxic Stage.—The interval succeeding the development of the earliest of the most usual symptoms of tabes, such as the lancinating pains, reflex iridoplegia, and loss of the knee jerk, and the appearance of noticeable ataxia, is most variable. In a small percentage of cases motor inco-ordination may be the first apparent symptom: less infrequently this may not occur for many (even twenty or thirty) years after the undoubted development of the first stage. Commonly, after a period varying from two or three or more years, during which such of the symptoms enumerated have appeared, the condition before described as static ataxia—Romberg's symptom—is accidentally discovered by the patient. It will then usually be very apparent on having the patient attempt to stand erect with the inner edges of the feet in juxtaposition. Unsteadiness will be still more evident if the eyes are closed or if the patient is directed to stand on each foot alternately, first with eyes open, again with eyes closed. It is likewise most noticeable if he is made to rise abruptly and walk immediately after rising, or if in walking he is checked abruptly and made to turn about. With the continuation in the other symptoms described, or perhaps their aggravation, especially as concerns disappearance of reflexes, the increase in the genito-urinary difficulty,[1] heightening and diffusion of anæsthesia, and appearance often of analgesia, of impairment or loss of the temperature sense and of the muscular sense, dynamic ataxia or difficulty in walking insidiously develops, and the second stage is fully entered.

In this stage co-ordination is so defective that, from the irregular contraction of the muscles concerned in balancing, this power is usually lost when the inside of the feet are placed in juxtaposition, or even with them widely separated, unless the feet are encased in shoes with broad soles: this is still more marked if considerable plantar anæsthesia exists. The patient is unable to walk comfortably at all in the dark (or with eyes closed), and tends to fall in turning or in stepping backward suddenly. With the guiding sense of vision ordinary locomotion may be accomplished on the level without very marked oscillations, but on an irregular surface or in descending stairs the ataxic gait is very apparent and characteristic. The patient is compelled to fix the eyes steadily upon his feet and the space in front of him. He then can progress reasonably well, and in a straight line with little or no oscillations, provided he proceeds with great deliberation and slowness. Should the eyes be closed momentarily or be removed from the feet, the movements become unsteady and he may fall to the ground. The attitude assumed in progression is very peculiar. The irregular and tonic contractions of the extensors and flexors of the body concerned in preserving equilibrium prevent the normal rhythmical, pendulum-like swing of the legs. The motions, on the contrary, are jerky and irregular. The feet are raised

[1] Sexual weakness, if not previously excessive, now advances to impotence, and incontinence of urine, or, less often, retention, with anæsthesia of bladder and urethra, occurs.

more than is necessary, thrown forward and outward too far, and the heels dropped too suddenly and usually with a decided thump. Oscillalations occur from the pelvis as the patient progresses, leading to alternate elevation and depression of the hips to clear the ground with the feet. Displacement of the line of gravity, due to irregular contraction in the muscle groups concerned, becomes more and more marked in locomotion as the inco-ordination insidiously increases, and finally all station is lost and balancing power is impossible without aid. At first a single stick or the guidance of another's arm is alone necessary. Subsequently, walking is impossible without support to both arms, as by aid of two sticks. Rising to the feet from the semi-erect posture becomes after a time out of the question. The legs are thrust aimlessly about, and even after being raised to his feet the patient's legs slip away disorderly, forward and backward, affording no support whatever to the body. The inco-ordination is similarly shown in recumbency if the legs are moved purposely about. Inability to readily touch objects held within easy reach with the toe or the entire foot is manifest. This of course is much more marked if after viewing the object the eyes are closed before the effort is made. A similar test may be tried by placing a small object before the seated patient, the position of which object he is to first inspect and then attempt to touch with alternate feet, the eyes being closed. The trunk muscles subsequently may likewise become inco-ordinate through the upward extension of the disease in the cord, so that swaying occurs even while the patient is seated, and the maintenance of easy equilibrium, even in semi-recumbency, is impossible if the eyes are closed. With the extension of the disease to the upper part of the cord, ataxia, as with loss of muscular and tactile sense, appears in the upper extremities. Inco-ordination in the arms may be first shown by the patient's inability to perform the finer movements of the hand, as in writing, or in women it is manifest in sewing. Buttoning and unbuttoning the clothes without the guiding sense of vision is early impossible, even with but little anæsthesia present. The patient is then found to be unable with eyes closed to readily touch with a single finger a designated part of the body, as the tip of the nose, its bridge, or the ear lobule. This should be tested with the fore finger of each hand alternately. Or, if directed to bring the tip of a certain finger of each hand in juxtaposition, the eyes being shut, the act is impossible or only performed hesitatingly after several trials. In a later period all movements with the fingers and hands may become so inco-ordinate that eating and self-dressing are impossible.

Notwithstanding the most extensive degree of motor inco-ordination, in an uncomplicated case muscular power and nutrition remain extremely good. The patient, if able to be about, can often walk long distances without more than ordinary fatigue. There is of course no motor paralysis, save very occasional transitory weakness in certain muscle groups, unaccompanied by wasting or the reaction of degeneration. Even in the preataxic stage this weakness may appear very unexpectedly, the legs temporarily giving quite away, causing the patient to fall precipitately to the ground as if the influence of the will were suddenly withdrawn. This has been especially referred to by Charcot. In many cases also a sense of great weakness is experienced, quite independent

of exertion, due probably to involvement of the nerves of muscular sensibility.

The electrical reactions are never qualitatively altered, even in the parts most affected in inco-ordination, although quantitative changes in the way of, at first, simple increase, and, later, of diminution in electro-contractility, occur as might be expected.

Perhaps the chief factor in the production of inco-ordination is failure of the muscular sensibility, which is a prominent symptom of the ataxic stage of the disease. A great deal of doubt has existed as to the chief cause of the inco-ordination in tabes. It seems, however, well established that mere tactile anæsthesia is little concerned in its production, and that the prime cause, to which the anæsthesia can be but contributory, is widespread disease of the fine sensory nerves which are concerned in the reception and conduction of afferent impressions of muscular sensibilities to the brain. Disease of these nerves in a minor degree leads to abolition of the knee jerk, although it may not be the sole cause of the loss. A much greater degree of disease, as Gowers states, is required to lead to inco-ordination, and a still greater to produce abolition of pressure sense.

Muscular sensibility becomes finally so markedly deranged that the patient is often totally unable to recognize the position of his extremities if passively moved in a direction unknown to him, and he is quite unable to discriminate between various degrees of pressure exerted, as, for instance, when tested by weights of different degree. In examining as to the condition of the muscular sense the patient's eyes should be closed or bandaged, and with him recumbent he is told to perform, in rotation, various movements with each limb, such as to bring the heel of one foot slowly and carefully to the great toe of the other, or he is asked to describe the position in which his limbs are placed. The muscular or pressure sense is also ascertained by noting with objects of similar dimensions, but of different weights, if the degrees of difference, which normally are very apparent, can readily be appreciated. The areas of hyperæsthesia or hyperalgesia remarked in the first stage, associated perhaps with plantar anæsthesia, give place later to pronounced tactile, pain, or temperature loss.

Anæsthesia is most often present in the plantar surfaces and upon the heels and toes, but may extend upward as far as the trunk, and sometimes involves the latter. Rarely the trunk, and not the limbs, is the seat of anæsthesia.

As a rule, pronounced and diffused anæsthesia in the upper extremity is rare. Moderate blunting in tactile sense may, however, be encountered in the fingers in the second stage; as Charcot pointed out, especially noticeable in the area of the distribution of the ulnar nerve (along the inner border of the forearm, hand, and middle finger). The patient is often unable to feel the floor under the feet, and when the anæsthesia involves the hand he is often quite unable to determine the primary quality of objects grasped. The anæsthesia need not be solely tactile, but may involve the muscles, bones, and articulations, thus explaining the painlessness of the arthropathies and fractures which may occur. Grave danger exists to ataxics from injuries, such as burns, through the absence of the appreciating warning sense of pain.

Pain accompanying visceral disease may likewise be absent. Gowers points out that a severe attack of pleurisy may thus go unrecognized.

The deep impairment of sensibility may be so pronounced that contraction of muscles with the strongest faradic or galvanic current is quite painless. The three forms of sensibility are not usually affected equally, and pain or tactile sensation may be alone involved. Temperature sense, on the contrary, is rarely singly diminished, and its impairment may be but partial, as to heat or cold. Often, with the accompanying diminution, there is a marked delay in the appreciation of tactile pain or temperature sense amounting to a number of seconds. With tactile and pain impairment the instrument stimulating both senses may cause appreciation of one some seconds before the other. Sensation may not be markedly impaired, yet the seat of stimulation may not be easily, if at all, recognized. So that the titillation of one part—as of the foot, for instance—may be referred to the knee or calf, or *vice versâ*, or a single point of stimulation may be felt in many places (polyæsthesia). A combination termed allochiria sometimes is encountered in which the sensation is referred to quite the same spot stimulated, but on the opposite limb. The condition of tactile sense is readily noted by the use of a feather or a piece of absorbent cotton, the tip of the observer's finger, or the æsthesiometer. This last is not more especially useful than the others. Whatever is employed to estimate tactile loss should not simultaneously tend to stimulate the temperature or pain sense (*i. e.* should be of the body temperature, and not sharp-pointed). In testing, the patient's eyes must be closed or screened, and, unless the skin be the seat of unduly thick epidermis, the touch should be very light. For practical purposes there is no better æsthesiometer than the intelligent application of the finger-tip, employing varying degrees of pressure according to lack of result.

If the æsthesiometer itself be employed, the extent at which normally the two points of the instrument are recognized at the slightest degree of separation in the part examined must of course be known. These measurements will be found described in all works on physiology, so need not be enumerated here. It must be borne in mind that the degree of sensitiveness often varies in different persons, and that the power of appreciation is much increased normally by practice.

The condition of pain sense may be ascertained in various ways readily suggesting themselves to any one; as, for instance, by the prick of a pin or, better, the point of a penknife. A hint of Gowers in this connection is of value. It is that too fine a point must not be employed in stimulating pain sensibility, lest in the naturally least sensitive parts, in which the terminal perceptive nerve plexus is wide, the prick of a fine point may be unfelt, although no loss of pain sense exists.

Temperature sense is often affected in tabes, anæsthesia existing either to heat or to cold, or to both, perhaps with perceptible delay in appreciation. Normally, perception of temperature sense is less promptly felt than pain, on account of the time consumed in raising the temperature of the part stimulated. This delay is usually much accentuated in tabes. Temperature sense may be so altered that a moderately cold body causes burning, and *vice versâ*.

The condition of the temperature sense is tested by applying cold and hot agents to the skin. The writer usually employs test tubes containing water of varying degrees of temperature.

The Terminal Stage of Locomotor Ataxia.—Tabes is essentially a chronic disease. As previously stated, many years may elapse before the onset of marked ataxia, and even after entering the second stage the patient, although unable to go about without assistance, may follow his usual vocation for years without further increase in the symptoms, the disease, as it were, tending to remain stationary. More usually the progression, though slow, and perhaps with occasional temporary remissions in symptoms, is steadily downward, until, finally, inco-ordination becomes so extreme that locomotion is altogether impossible even with assistance, as is also the maintenance of the sitting posture. The patient is then compelled to remain abed, altogether helpless. Anæsthesia and loss of the muscular sense are extreme. He is quite unaware of the position of his limbs without the aid of sight. The bladder, if it has not previously become so, is now paretic, requiring the steady employment of a catheter, and chronic cystitis has usually supervened with perhaps secondary kidney disease. The rectum may be so anæsthetic that involuntary evacuations of fæces occur. The patient may continue in this, the third stage, in fair condition, although helpless, for even twenty years, although the average duration of life after the first appearance of the disease is considerably less than this, not exceeding eight to twelve years. The muscular strength and nutrition throughout the course of the disease usually remain unimpaired, other than the weakness natural from mere flaccidity and wasting of disuse. Subsequently, through diffusion of the sclerotic process to the anterior cornua —of not infrequent occurrence if the patient is not carried off by an intercurrent malady, such as renal disease, pneumonia, or phthisis pulmonalis—diffuse wasting and flaccidity occur, with the appearance of a true paralysis, with reaction of degeneration in the affected muscles. Or the disease may in its upward progress in the cord reach the medulla, causing death from respiratory implication. Bedsores, although common in the third stage, are rarely the cause of death.

Forms of Locomotor Ataxia.—Apart from the typical form of tabes described, general mention must be made of certain recognized aberrant types, no lengthy description of which can, however, be given here.

The mild form is that described by Charcot, and incidentally before referred to, in which, although the disease undoubtedly exists, the symptoms are not prominent nor troublesome, and after appearance remain stationary for many years. Lancinating pains, although occurring, are of no severity, but loss of the knee jerk, slight static ataxia, and reflex iridoplegia occur.

Tabes dolorosa (Remak) is a term applied to what is also called the *neuralgic form* and the *abortive form*, characterized by the extreme violence of the lancinating pains. Pains with absent knee jerk may be the only symptoms for upward of twenty years. To the abortive type *amaurotic tabes* essentially belongs, since, frequently, with the onset of early optic atrophy, other symptoms of implication of the lumbar cord, save the loss of the knee jerk if present, sink into the background.

The severe form, described by Duchenne, is that in which nearly all the classical symptoms occur and are remarkable for their intensity.

An *acute form* is also described, in which the symptoms run a rapid course, and the third or paralytic stage is arrived at in nearly as many months as it ordinarily takes years to reach.

The *cervical form*—quite rare—is that in which the symptoms have onset in the arms. The legs are but little if at all affected at first. Cases have been reported by S. Weir Mitchell, J. K. Mitchell, H. C. Wood, Martins, Bernhardt, and Eichhorst. Marie urges caution in not confusing this form with syringomyelia.

The *hereditary form* (Friedreich's ataxia) is described in another section (p. 225).

A rare form is the *hemiataxic*, in which the symptoms, although bilateral, are much more pronounced on one side.

Certain other forms are described in the books which are simply tabes complicated with other spinal or cerebral disease. Thus the *paraplegic form* is that in which the sclerosis has invaded the lateral columns, causing what is termed by Gowers *ataxic paraplegia*.

The *meningitic form* is that in which implication of the spinal meninges in the disease gives rise to symptoms referable thereto, such as severe spinal pain and tenderness and diffused cutaneous hyperæsthesia.

A not infrequent complication of tabes is general paresis of the insane. Some regard the coincidence of the two diseases as representing a special form. Certainly the two maladies are often combined, not infrequently starting with symptoms of tabes, and terminating with these plus those of general paresis. It must be borne in mind that the diseases have one symptom in common, the loss of the pupil light reflex, and that syphilis is a common antecedent in both.

Myelitis (acute or chronic) may also occur during the course of tabes, adding its special symptoms to the complexus present.

Muscular atrophy complicates tabes with some frequency. This may be *general*, occurring as a definite affection either in the second or third stage (true progressive muscular atrophy) as a result of the disease invading the ganglion cells of the anterior cornua; or *local*, due either to involvement of trophic centres or more usually (as Déjerine has shown) to degenerative atrophy of the peripheral nerves.

Occurring as a part of muscular atrophy arising from invasion of the trophic centres are general amyotrophy, ophthalmoplegia, and bulbar palsy.

Hemiatrophy of the tongue is of not very infrequent occurrence in tabes, and is due to a degenerative central (medullary) lesion. This occurs as a late symptom, and was first remarked in connection with tabes by Charcot. It should be stated in this connection that hemiatrophy of the tongue has been noted occurring in general paresis, and as a late lesion of syphilis apart from tabes and general paresis.

Prognosis.—The presence of indications of tabes must of course always be viewed with the greatest seriousness, and a guarded prognosis, even in the earliest stage, given. It must be apparent that the earlier in the disease the case comes under observation the greater the opportunity for arrest and improvement. Since the disease is dependent upon

a neural degeneration the immediate cause of which we are quite ignorant, and for the cure of which no specific yet exists, it is easy to comprehend the natural progressive tendency usually shown, and how futile the hope of actual cure can be in a case sufficiently advanced to indicate symptomatically distinct destruction of tissue by the sclerotic process. The most important point I have in mind in the prognosis and treatment of tabes when the disease is encountered in its actual incipiency is that the pathologic condition has probably not yet advanced beyond a condition of mere molecular alteration—one of deranged nutrition. If this nutritional alteration can be stayed and the toxic blood state underlying the disease, if not specifically antidoted, measurably overcome by proper hygienic means and by drugs, as is plausible, much may be hoped for in the line of actual cure. It is easily understood, in considering the pathology and natural history of the disease, that the nature of the prognosis largely depends not only upon the stage in which the case is encountered, but upon the individual peculiarity or susceptibility, as is shown in certain cases by a natural tendency toward induced or spontaneous arrest, and in others by a rapid downward tendency despite all that can be done.

That not only arrest, so that the ataxic stage is never reached, but actual cure, may occur in the first stage there is slight doubt, and even in the second stage much may be commonly done toward arrest of the most troublesome symptoms for long periods, if not permanently. In the third stage little is to be hoped for, although occasionally bedridden tabetics have been practically returned to the second stage. In true tabes preceding syphilis seems not to influence the prognosis, since rarely if ever is an antiluetic treatment of avail.

Concerning the prognosis of special symptoms a word should be said: Lancinating pains, usually regarded as the most obstinate, as they are unquestionably the most distressing, symptoms of the disease, are so often helpfully influenced by suspension, especially in the cases in which the disease is stationary, that unless a markedly progressive downward tendency is shown by the tabetic, hope of benefit may be extended.

The bladder symptoms and impotence, as well as static ataxia, and indeed marked dynamic ataxia, are also often improved by suspension and other measures to be later discussed.

The crises, especially when dependent upon degeneration of the nerves involved, are not usually amenable to treatment. When dependent upon a toxic blood state, improvement may occur under proper hygienic and medical measures.

For optic-nerve atrophy, if at all advanced, little can be done. As before stated, these cases usually steadily progress to complete blindness. Rarely, and then in the most incipient cases, arrest under treatment has occurred. The influence of the occurrence of optic-nerve atrophy on the purely spinal symptoms has been before considered.

Complications, such as chronic cystitis, renal disease, phthisis, and aortic regurgitation, naturally render the prognosis much more unfavorable, materially shortening the life of the ataxic.

Diagnosis.—The diagnosis of tabes, more especially in the early stage, is often beset with great difficulty: it is well to recognize this at the start, lest unfortunate error arise.

In cases with paucity of symptoms the age should always be taken into account and compared with that already stated at which tabes is most common, and a searching inquiry should be made as to evidences of previous specific infection. While the mere history of syphilis cannot suggest tabes, since from the standpoint of syphilis alone tabes is not common, yet the fact of its past occurrence, with the presence of symptoms suggestive of tabes, lends additional weight to their probable character. The physician should not readily be satisfied with a mere negative rejoinder in answer to a general inquiry as to lues, but should, in as guarded and politic a manner as the nature of the inquiry demands, ascertain if there have been noticeable evidences of a primary sore, or of secondary skin, and mucous-membrane lesions, or of tertiary manifestations years before; and, if so, if these received a *thorough* mercurial course. This fact is of value not only diagnostically, but also therapeutically, as will be pointed out hereafter.

With or without a history of syphilis, should several of the common symptoms of tabes be present, such as the Argyll-Robertson pupil, loss of the knee jerk, lightning pain, with or without Romberg's sign, the diagnosis may be accepted as established quite beyond question. But this assemblage of symptoms is not always usual, and we are often called upon to decide cases in which perhaps but one signal symptom exists, and only a few minor general ones of no very direct diagnostic importance. The value of each symptom must then be carefully weighed and placed with that of the others. It must be considered in what other disease or diseases, if any, these are of likely occurrence, and if any of the symptoms are peculiar to tabes. This can be said of the triad above mentioned, which includes the loss of the pupil reflex to light. Since, as I have before narrated, the knee jerk may apparently, very occasionally, be absent in health, and since also several other affections of the nervous system have this symptom, with, also, ataxia, it cannot be regarded as of as much value as is the presence of the Argyll-Robertson condition of the pupil—the presence of which should at once suggest a strong suspicion of either tabes or general paresis.[1] This symptom would be regarded as of more value than the loss of the knee jerk were it as constantly present. Regarding loss of the knee jerk, the question arises as to its exact value in the diagnosis of tabes. Alone we cannot regard this absence other than with mere suspicion, but, noted to accompany lancinating pains and slight inco-ordination, the diagnosis is quite assured, especially, as will be later pointed out, if care be taken to eliminate a form of neuritis in which pains, inco-ordination, and loss of the knee jerk are present. This is the easier if the pains are, as stated, of the characteristic lancinating sort. When loss of the knee jerk and but one or two minor symptoms are present, the diagnosis is often puzzling in the extreme. To illustrate this a few examples may be cited: Thus, three years ago I saw a case with Dr. Lewis Brinton in which with history of early syphilis there had been present indications of neural derangement of some months' duration. The patient was a man, aged forty, engaged in a business which necessitated long and continuous mental application. There was a history of overwork dating for

[1] Occasionally the Argyll-Robertson pupil has been noted occurring in other degenerative diseases, and sometimes alone as a single sequel of a long preceding syphilis.

years. His health had stood the strain until recently. The symptoms (largely mental) were to my mind those of pronounced, though simple, nerve exhaustion of easy assignable cause, plus eye-strain, which was found to exist. Certain of the mental symptoms were temporarily quite removed during the use of atropine pending refraction. But there was also present loss of the knee jerk, even to reinforcement. This naturally caused tabes to be suspected. A thorough search for other symptoms revealed nothing characteristic. No other assignable cause for the loss of the patellar tendon reflex was evident. Dr. Brinton then recalled that, in seeing the patient several years before for another ailment, examination for the knee jerk as a matter of routine showed its absence. The patient now informed us that its absence had been noted by himself when a boy at school. Subsequently, under proper hygienic and medical treatment, all symptoms disappeared. This case is interesting because of the presence of obscure nervous symptoms with absence of the knee jerk, which might have led one to the diagnosis of presumable early tabes, especially in view of the preceding common causal factor, syphilis.

The most common affection with which tabes is likely to be confounded is peripheral neuritis, especially that occurring as the result of diphtheria, diabetes, or the abuse of alcohol. In that of diphtheria[1] and diabetes but slight or no ataxia may be present, the case well simulating the first stage of tabes. But in these affections, depending as they do upon a neuritis, we have indications of motor weakness in addition to the ataxia and undoubted qualitative electrical alterations (which should always be searched for) in the paretic muscles, and perhaps, as nearly always in acute alcoholic peripheral neuritis, tenderness in the affected muscles. In diphtheritic neuritis, too, a history of indications of preceding sore throat is usually evident, and often, subsequently, there is also found paralysis of accommodation and of the palate.

It must be recalled in this connection that all cases in which the knee jerk is abolished in diabetes are not necessarily those of peripheral neuritis alone, since changes in the posterior columns of the cord have been found in this disease, evidently due to the toxic blood state present,[2] as they have been also in other diseases, such as pernicious anæmia. In certain cases of presumed diabetic peripheral neuritis difficulty in diagnosis is occasionally very great. Thus Grude[3] records a case in which he first diagnosed tabes complicated with diabetes, since, in addition to pronounced glycosuria and symptoms such as might have been due to an accompanying peripheral neuritis (ataxic gait, paræsthesia, and lancinating pains in the limbs, with flabbiness and atrophy of the leg muscles, marked ataxia, absent knee jerk) there was bilateral myosis with loss of the light and accommodative reflex. Under treatment the diabetes greatly improved, and in the course of a year the pupillary immobility had disappeared, as had partly the ataxia. The knee jerk remained absent. This case, from the improvement in certain symptoms, Grube finally

[1] Loss of the knee jerk is very common following even slight attacks of diphtheria, with no other indication of nerve derangement.

[2] See a paper by Williamson: "Changes in the Posterior Columns of the Spinal Cord in Diabetes Mellitus," *Brit. Med. Journ.*, Feb. 24, 1894. Other references are Sandmeyer, *Deutsch. Archiv f. klin. Med.*, Bd. 50, and Leyden, *Wien. med. Woch.*, No. 21, 1893.

[3] *Neurol. Centralbl.*, Jan. 1, 1895.

regarded as one of peripheral neuritis due to tabes, although certain of the symptoms indicate that slight degenerative changes may have likewise occurred in the cord.

That loss of the knee jerk may occur in chronic malarial poisoning, with the presence of other symptoms simulating locomotor ataxia, and not be dependent upon discoverable neuritis, I have nowhere seen remarked. The following case of my own is of great interest in this regard, since for a short time I was myself inclined to view it as one of early tabes: J. L——, aged thirty years, stationary engineer, first came under observation in April, 1889. Of robust build, weight 190 pounds; denied lues. He had had for two or three years preceding an attack of intermittent malarial fever. Had noticed, for some little time before he consulted me, three or four times weekly feverishness, not preceded by chill nor succeeded by sweat. The eyes on these occasions seemed "full of sand," and he was markedly languid and soporose. Gastric symptoms were present, indicative of hyperchlorhydria, and there was intestinal indigestion. The spleen was notably enlarged; other organs normal. There had been lost nineteen pounds of flesh in nine months. He was given 12 grains of quinine daily, with arsenic, and occasional nightly doses of podophyllum or calomel. Rapid improvement occurred. Febrile and other symptoms disappeared and weight was regained. Treatment was discontinued in a short time. He re-presented himself in the spring of 1891, complaining only of inability for coitus: neither erection nor ejaculation was possible, and there was loss of sensation about the genitals and absence of the cremasteric reflex. Sexual power had slightly failed before he had previously seen me, but of this he then had made no remark. Now sexual loss was absolute. At this time no other symptoms were complained of except cold extremities and a tendency to vertigo. No further loss of flesh had occurred. I did not refer to the early notes of his case, and had entirely forgotten that he previously had had malarial infection. The knee jerks were both absent, even to reinforcement. Station was normal and pupillary light reflex present. Plantar reflex was absent and slight plantar anæsthesia present; cold feet were habitual. Strychnine, arsenic, zinc phosphide and valerianate, and damiana were ordered, either combined or singly, and continued for several months. Slight erective power returned under this treatment pursued for some time. The knee jerk continued absent. I now sent him to my service at the Jefferson Hospital, where I began suspension, employing it three to five times weekly, all drugs being discontinued. Sexual power, which had ceased to improve beyond ability to obtain semi-erections, with premature very small ejaculations during coitus, under the drugs used, returned after a few suspensions (one month of treatment). Knee jerk had then reappeared, at first sluggishly and to reinforcement only (noticeable only as a slight crossed reflex). But subsequently without reinforcement the knee jerks were readily obtainable and were manifest both as a direct and as a crossed response. Suspension was discontinued after a two months' trial, and in a short time he relapsed into the former condition of impotence, with the renewed sluggishness of the knee jerk. There was at no time muscular wasting or other indications of paresis. The electrical reactions, taken on several occasions, were normal. Now, more closely pursuing the

early history, it was noted that he had previously suffered from malaria and been treated for the same with success. Examination again disclosed very decided enlargement of the spleen. He was placed on full doses of quinine with arsenic, and given small doses of podophyllum at night. Two Turkish baths were taken weekly. This treatment was continued steadily, with occasional intermissions, for a number of months. The spleen had greatly decreased in size in three weeks, and he was perceptibly brighter, probably due to improvement in erective power and in circulation. A few weeks later it was noted all symptoms had disappeared. The knee jerks were active, plantar anæsthesia gone, sexual power nearly normal. He has remained well to the present.

Other forms of pseudo-tabes, with which locomotor ataxia in its more developed form is likely to be confused, are especially peripheral neuritis of the ataxic variety, due to alcohol and to arsenic, and mere functional ataxia, the so-called neurasthenic pseudo-tabes. The diagnosis in multiple neuritis usually hinges on the less chronic onset (usually acute in multiple neuritis) and the association with the symptoms of inco-ordination, those of distinct paresis or paralysis of certain groups of muscles, such as (in alcoholic neuritis) the extensors of the feet and hands (causing foot- or wrist-drop), which soon exhibit qualitative and quantitative electrical alteration and some wasting. Tenderness on pressure is felt in the muscle groups affected.

In peripheral neuritis, however caused, the pains are never of the distinct lancinating, deep-seated character present in locomotor ataxia. They are, on the contrary, of the nature of neuralgia, usually being limited to certain nerves, and are apt to be associated with areas of tenderness on pressure over the paralyzed muscles, and in alcoholic neuritis with unprovoked muscular pain.

In functional or neurasthenic pseudo-tabes there may be pains, inco-ordination, partial impotence, and diminished knee jerk; but none of these symptoms are of the precise character apt to be encountered in a well-marked case of tabes. The symptoms are also usually of abrupt onset, more common in women than in men, and there is usually no history of syphilis.[1]

The diagnosis between tabes and cerebro-spinal sclerosis is commonly easily made, even with the presence of ataxia in the latter, and, as occasionally occurs, optic atrophy. The ataxia is of a coarse, jerky character, such as is observed in cerebellar tumor, and affects oftentimes as much the muscles of the trunk and upper as well as the lower extremities. This, together with tremors, characteristic stammering speech, vertigo and nystagmus, exaggerated knee jerks, absence of lancinating pains and pupillary symptoms, renders the diagnosis a matter of slight difficulty.

Paraplegias of ordinary form, such as spastic due to primary or secondary lateral sclerosis, or that due to the disease of the posterior and lateral columns, show exaggerated knee jerks, ankle clonus, with absence of pains and anæsthesia. In the former there is no trace of ataxia, and there is a typical spastic, paralytic gait. In postero-lateral sclerosis there is at first ataxia, succeeded later by a spastic condition of the muscles and the gait of lateral sclerosis.

[1] In the diagnosis of neurasthenic pseudo-tabes Marie lays stress on the preservation in men of the cremasteric reflex, with the presence of partial or complete impotence.

The separation of cerebellar disease, such as tumor, from locomotor ataxia is accomplished by taking cognizance of the difference in the character of the ataxia. Thus in cerebellar disease the gait is reeling, like that of a drunken man, the trunk muscles being as much or more implicated than the legs. There are also absence of lightning pains, and the presence always of severe headache, and usually of optic neuritis, vomiting, vertigo, and increased but unequal knee jerks.[1]

Syphilis of the spinal cord is sometimes indistinguishable clinically from true tabes, from which it differs totally in histological character. Cases of this sort must therefore be treated as those of tabes. It must be borne in mind that syphilis of the cord may occur not only as early as the third month after the primary lesion, but as late as twenty years, so that a distance in time of great or minor degree separating the primary lesion from the appearance of symptoms of the spinal malady, cannot especially be allowed to weigh in favor of either affection in diagnosis. Usually cases of cord syphilis presenting symptoms that really confuse are apt to display certain irregularity of manifestation which serves to assist in the differentiation. Thus the paraplegic symptoms tend to predominate more on one side, as Gerhardt has pointed out, and there are often cerebral manifestations. When syphilis affects the blood-vessels as well as the meninges, sudden exacerbation of the spinal symptoms is not uncommon, with rapid onset of paraplegia. Gerhardt remarks that should the syphilitic process invade the spinal cord, the cervical region is that generally selected.[2] A true syphilitic affection, such as a gumma, may coexist with posterior sclerosis, causing symptoms of its own and a blending of those of the two maladies, not only puzzling to comprehend, but impracticable to accurately diagnose, even when the fact of the possible coexistence of syphilis of the cord and a true posterior sclerosis is borne in mind.

Treatment.—As before dwelt upon, although tabes seems in the vast majority of cases to be dependent upon preceding syphilis, it is not histologically a syphilitic disease, the lesions of the typical affection approaching nowise those of tertiary syphilis. These latter are rather to be considered, as pointed out by Strümpell, as degenerative sequels of syphilis, the result, probably, of the production of a non-organized toxin in the body, over which antiluetic treatment is without influence. This fact seems quite well established, and is of the utmost importance, since the mere clinical relation of tabes to syphilis has led many in the past to infer an existing pathological sequence, and hence to expect therapeutic results in a line with those obtained from the employment of mercury and iodides in syphilis. This view has not only given rise to much disappointment, but has actually been provocative of harm, since it has led often to the injudicious use of agents, the too free employment of which, when not indicated, could only be detrimental. Thus in many cases of typical locomotor ataxia in which the general condition of physical tone has been depressed, an energetic and long-continued mercurial course, pursued far beyond that necessary for diagnostic or for intelligent therapeutic ends, has seemed finally to hasten rather than arrest the progress of the disease. All who have had

[1] If optic atrophy is encountered, it is always post-neuritic, and not primary.
[2] *Berl. klin. Woch.*, No. 50, 1893.

experience in the treatment of tabes have seen instances of this sort. It cannot, therefore, be too strongly urged that, however close the connection between tabes and lues, the former is in its typical pathological character not syphilitic, and hence not amenable to luetic treatment. This rational inferential fact is now most abundantly supported by clinical evidence. Yet, notwithstanding this, it is also unquestionable that in repeated isolated instances in the experience of all who have seen many cases of disease of the spinal cord it occurs that the judicious use of mercury in the early stage of what has been symptomatically diagnosed locomotor ataxia has resulted in not only staying the disease, but in removing all symptoms thereof. The occasion of this is the occurrence of a syphilitic affection of the cord which from mode of onset and general clinical character and situation (due either to syphilitic new formations, such as meningeal gummata, or to syphilitic disease of the bloodvessels) originates symptoms totally indistinguishable from those of true tabes. Instances of this are now so numerous in literature that the fact of their frequent occurrence cannot be neglected. These cases must be classed clinically with those of tabes, since they are often totally inseparable.

Recently Ewald[1] relates a most instructive case of this sort, in which there were present static and dynamic ataxia, absent knee jerk, partial tactile anæsthesia, and temperature sense loss, with absence of the pressure sense and the pupillary light reflex. The necropsy showed no changes indicative of true tabes, but instead a chronic fibrous and gummatous spinal arachnitis and a diffuse chronic interstitial myelitis, associated with endarteritis. The process was syphilitic purely. It began as an interstitial proliferation, which proceeded from the periphery, affecting the nerve fibres secondarily and in limited areas. The occurrence of these cases unquestionably indicates that, although antisyphilitic remedies are without avail in true tabes, and sometimes when improperly employed are even harmful, their use cannot be practicably unqualifiedly condemned in the treatment of the disease, as is done by some clinicians. Thus in a recent writing on locomotor ataxia[2] this whole subject is dismissed with the words: "Antisyphilitic treatment is of no value, even though the history of syphilis be clear. It is true that medical literature abounds with reports of cases which seem to oppose this statement, but I have no doubt that in such cases the diagnosis has been incorrect." It is interesting, in view of the foregoing, that Wood himself but a year or two previously drew attention to a form of syphilitic pseudo-tabes (due to infiltration along the spinal dura mater, involving the posterior roots), the diagnosis of which, in a case studied by himself, he totally failed in, regarding it as one of true tabes until the necropsy revealed its true nature.[3]

If Wood's view as to the inutility of antiluetic treatment in tabes was generally adopted, it might not infrequently occur that cases regarded as those of locomotor ataxia, but in reality those of specific cord disease, perhaps indistinguishable from tabes, would proceed to a fatal issue in a manner similar to what occurs in the progressive affec-

[1] *Berl. klin. Woch.*, No. 12, 1893.

[2] H. C. Wood in *An American Text-Book of the Theory and Practice of Medicine*, vol. i. 786.

[3] *Lancet*, Aug. 1, 1890, p. 289.

tion, which otherwise might promptly be cured if active treatment were begun sufficiently early. The vital danger attending mistake in pathological diagnosis, as far as concerns the withholding of the specific remedy in syphilis of the spinal cord and the slight harm that can occur from its judicious employment, renders it almost imperative that in every case of tabes,[1] however typical clinically, coming under observation, in which there is reason to suspect past syphilis, the case should receive for diagnostic purposes a short course of treatment by mercury alone or by mercury and potassium iodide. Especially should this be done if it can be ascertained that, with evidences of past syphilis, such as the presence about the trunk or limbs or on the penis of cicatrices, there has been no thorough mercurial course pursued. Evidences of this, as of any marked indications of secondary or tertiary lues, are often absent, since, as before remarked, it is commonly the cases with mild primary, secondary, and tertiary symptoms—in other words, of practically untreated syphilis—from which those of nerve syphilis develops. Space does not permit lengthy discussion as to the best mode of applying mercury or mercury and potassium iodide. Suffice it to say, that for promptness of effect and certainty of results from the use of mercury the hypodermic method is far and away preferable to mouth administration, and succeeding that, when its hypodermic use is impracticable, that by inunctions, although disagreeable of application, is closely second in value. For hypodermic use mercuric bichloride is preferred by the writer to the many highly lauded insoluble preparations. It may be employed in 1 per cent. solution, and a daily dose or alternate daily doses of gr. $\frac{1}{8}$ to $\frac{1}{4}$ (or even a larger dose should syphilitic infection have been recent and some features of the case suggest a typical lues) from two weeks to a month, and then finally abandoned or intermittently continued, the subsequent use to depend upon the result obtained.[2] If distinctly beneficial, decided amelioration in certain symptoms having occurred from energetic treatment, the remedy should, of course, not be abandoned, the case now being viewed as one of probable nerve syphilis. The use of mercury in this way, observing common-sense rules against overdose, even when unindicated in pure tabes, is unlikely to be harmful, and may be of the greatest value diagnostically. Potassium iodide may be coincidentally administered by the mouth. It is preferably used alternately with a short course of mercury, the latter being employed steadily for a period of ten days to two weeks, succeeded by the potassium iodide for a similar time in steadily increasing doses, until, if not markedly disagreeing, from a minimum amount of a half-drachm daily, this same quantity, to a drachm and a half three times daily, is reached. It should be administered very largely diluted in water or milk. Potassium iodide must be employed with caution, since even in moderate

[1] Except those cases in which optic atrophy is in process. Then the use of mercury is harmful and may hasten the onset of blindness.

[2] The various methods of the hypodermic employment of mercury, as well as the proper technique of the use of inunctions, most important to clearly understand, are fully described by Taylor in *The American System of Therapeutics* (Lea Bros. & Co., publishers), vol. ii., to which the reader is referred for particulars. If the hypodermic or inunction method cannot for any reason be employed, the best salt for internal administration is probably the protoiodide, in doses of gr. $\frac{1}{4}$ to $\frac{1}{3}$, three times daily, guarded by a small quantity of opium and belladonna. (See also Vol. I. of this System, p. 500.)

dosage in some cases of tabes its administration disturbs the stomach and much debilitates the patient. It is quite impossible to predict which of the two drugs is indicated. If mercury is found of striking benefit, trial of potassium iodide is of course unnecessary. Potassium iodide, as a rule, is more distinctly useful in tertiary syphilis than mercury, and yet cases are not infrequently encountered, especially of nerve syphilis, in which potassium iodide is without utility and mercury alone is rapidly curative. Indeed, there are some neurologists—such, for instance, as Althaus—who claim that potassium iodide is of far less utility than mercury in nerve syphilis. Althaus finds that mercury acts as a true specific in all primary luetic nerve lesions, just as it does in all the earlier manifestations of the secondary period. My own experience has been decidedly more favorable to the use of mercury than potassium iodide in nerve syphilis, and not to the employment of the two drugs in combination.[1]

The reason for the greater efficiency of mercury is difficult to understand: it may be explained, as I have remarked elsewhere,[2] by the fact that "it is well known that in many cases of nerve syphilis primary and secondary symptoms have been so slight as to pass unnoted by the patient, while the symptoms of involvement of the nervous system are the first that distinctly appear. In these, of course, there has been no specific treatment. May it not be that in these cases the syphilitic virus still retains the contagious and transmissable qualities of its earliest stage, and is only capable of being brought into subjection by mercury?"

A short and energetic, although intelligent, employment of mercury being without marked effect, it should be discontinued and the use of potassium iodide begun. This also proving resultless, luetic treatment should be finally abandoned, it being properly presumed the case is one of true tabes, from which no benefit by antisyphilitic means can be expected. This, of course, premises the case yet to be in an early stage, in which damage to important structures is not irremediable. With the destruction of nerve centres or of afferent paths by the disease process, however specific the remedy may be, it cannot restore lost parts.

In true tabes, for which at the start it must be acknowledged there is no known specific, much may be done by properly directed hygienic and therapeutic measures toward not only arrest, but in a few cases actual cure, of the disease if encountered sufficiently early; and even in the later stages as well not only temporary amelioration of symptoms, but arrest for a long period, may be often produced, although less frequently than in the early stage.

It is important to remember in the treatment of the disease that in its incipiency the pathological condition is one probably of mere molecular alteration, which, should it be encountered sufficiently early, is probably susceptible of check and removal.

Hygienic measures are of the utmost importance. It has sometimes

[1] In this particular see the history of a most instructive case of my own of cerebro-spinal syphilis, in which the combination of mercuric chloride in moderate and potassium iodide in large doses failed to relieve symptoms, rapidly yielding to moderately full doses of mercurous iodide alone (*Medical News*, April 11, 1891).

[2] *Medical News*, Apr. 11, 1891, p. 401.

occurred that a total suspension of injurious habits, such as late hours, excesses in venery, alcohol, and tobacco, have served in themselves to arrest the progress of the disease in the early stage, entirely without the assistance of drugs.

Few can avail themselves of all the conditions favorable for recovery, such as will be related, but all should be enjoined to do so as far as is possible.

The patient should be so far cautiously impressed with the gravity of the ailment as to recognize, without the occasion of undue depressing worry, the importance of attending to the general health. This comprises a course of living over which the physician must maintain a careful scrutiny. Open-air life, preferably in the country in a dry and warm climate at low altitudes, should be advised. Moderate daily exercise should be taken, but over-fatigue must be most carefully avoided. Walking long distances should be prohibited. Physical over-exertion may easily distinctly increase the inco-ordination. Exposure to extreme cold or to inclement weather tends to aggravate the disease. If the patient be much debilitated, so that moderate exercise produces overtire and seems to heighten ataxia, or fulgurant pains be excessive and obstinate, rest in recumbency in freely ventilated apartments, during which massage by an experienced masseur is employed, is important. Regular hours for sleep should, of course, be enjoined. The greatest care must be urged on the patient or his attendants, that causes of physical or mental strain, if existing, be removed. If able to be about, the patient to be in best condition usually requires to be occupied in some diverting manner, but the usual previous vocation, if it involves too continuous application or wearisome demand on mind or body, must, if possible, be abandoned. Recreations of a harmless sort should be encouraged.

In the preataxic stage a cool or cold sponge bath or needle douche in the morning, perhaps preceded by a general immersion of the body in moderately hot water and succeeded by energetic friction by the hand and coarse towel, is of use to impart tone to the system. In more advanced cases the simple warm bath (95° F.) from fifteen to twenty minutes daily may be tried. In any case, should discomfort or aggravation of symptoms result from the hot or cold baths employed, they must at once be discontinued, and such baths afterward employed consisting of extremes of temperature not exceeding 70° to 100° F. Venereal excess, however slight, for reasons easily understood, is unquestionably harmful to the tabetic. Although it is quite impossible to lay down any fixed rule as to frequency, it may be said, however, that the sexual act should not be permitted more that once weekly in the married, and that extreme continence in the unmarried tabetics is absolutely imperative.[1] Curtailing venereal excess is extremely difficult in some cases accustomed for years to habitual over-indulgence.[2] Yet to obtain the best results from treatment it is imperative that the necessity for it be laid before the patient.

[1] Should nocturnal emissions occur on cessation of sexual indulgence, they must be controlled by appropriate means.

[2] This refers more particularly to the preataxic stage of tabes, in which loss of sexual desire and ability may not have occurred.

While hope for cure or for decided amelioration in symptoms exists tobacco must be allowed only in extreme moderation. It unquestionably has an injurious influence on the muscular and nervous system in a disease like tabes. As to the effect of even moderate smoking in health, it is interesting to note that Lombard has shown that smoking but one cigar of no undue strength will reduce neuro-muscular energy to one fifth its previous amount, and that, although the depression is but temporary, more than an hour elapses before complete restoration to the normal occurs.[1] A moderate use of alcohol in the shape of a light wine or, preferably, malt liquor, is advisable, especially if the appetite be impaired or insomnia exist. Excess must always be guarded against, since too free indulgence in alcohol will apparently sometimes excite severe exacerbation of symptoms.

Careful attention is to be paid to the state of the digestive apparatus. Should symptoms of indigestion exist, the condition of the motor and secretory functions of the stomach should be ascertained by appropriate measures, and suitable remedies then be employed. This is highly important, since without good digestion nerve tone must suffer and the disease assume the ascendancy. Care must be taken to distinguish between symptoms of indigestion due to hyperchlorhydria—of not infrequent occurrence in tabes—and those of lowered secretory tone and mere atony of the stomach (the last of which may exist alone or with either of the former),—all fruitful sources of gastric and intestinal trouble.

The diet should be of an appetizing though of a simple and digestible character. Excess in eating of even the simplest fare must also be guarded against, as likely to provoke indigestion if absent or to aggravate it if present. It is well recognized that attacks of lancinating pains are often directly induced by disorder of the stomach or by constipation. Regularity of the bowels must be maintained by appropriate means.

Apart from drugs which may be required for the digestive condition, such as various forms of the vegetable bitters, none of which should be employed unless indicated, and then only for periods of a few weeks at a time, certain remedies which have an influence for good on nerve tone are often required and should be employed. Chief of these is arsenic. That this drug often is distinctly beneficial in tabes, through a retarding influence it apparently may exert on the degenerative process, is quite generally acknowledged.[2] Arsenic may be used in the form of the acid or the alkaline solution (the liquor acidi arseniosi and liquor potassii arsenitis), the former of which should be preferred as much the more stable and certain. It should be given in doses of from 3 to 5 minims three times daily for periods of three to four weeks, and then intermitted for a time, perhaps with another drug, such as silver. Of course attention should be paid in its administration to avoid toxic symptoms should the dose be excessive through undue susceptibility. A proprietary solution of gold and arsenic chloride is now much lauded in nervous affections as of greater value than arsenic alone. I have

[1] See the *Lancet*, Dec. 31, 1892, p. 1501.

[2] Gowers states that most of the cases in which he has seen the greatest improvement occur were on arsenic at the time. He attributes the amelioration in these to this drug.

employed it somewhat with apparent benefit, but not sufficiently to judge of its claims to superior virtue. Succeeding arsenic, zinc valerianate is often of utility, especially in those cases in which a neurasthenic element is prominent. I have used this drug a good deal in neurasthenia, when existing as an independent disorder, with great success, and have employed it somewhat in tabes. It should be given in doses of 2 to 3 grains, three times daily, in capsule or gelatin-coated pill, and may often advantageously be combined with arsenious acid—gr. $\frac{1}{40}$ to $\frac{1}{20}$; strychnine sulphate, $\frac{1}{60}$ to $\frac{1}{30}$ (or extract of nux vomica, gr. $\frac{1}{8}$ to $\frac{1}{3}$), and with sumbul, gr. 1 to 2, the last of which is of some value as a nerve-bracer. This combination, in various modifications, I employ a great deal as a nervine in other affections than tabes. It may be of good service in the early stage of this disease. Strychnine is of no special utility save as a general nerve tonic. It may be used alone or in combination with arsenic or the other drugs mentioned when apparently indicated, especially to meet symptomatic indications, such as for the relief of urinary incontinence and of impotence.

Silver nitrate is highly recommended in tabes by some authorities; others, again, condemn it utterly. The fact is, that it is probably of some utility through a direct influence on nerve tissue the seat of the degenerative process, in which the metal is deposited. The variability in results obtained from its use are doubtless largely due to methods of employment. Apparently to obtain the best effects it must be employed in such a manner that decomposition does not occur before or during its ingestion, and it must be taken in rather large doses (gr. $\frac{1}{8}$ to $\frac{1}{3}$ three times daily) for a comparatively long period. It is best administered dispensed and taken in distilled water and on an empty stomach. There is, of course, great likelihood of blueing of the skin and mucous membrane if continued sufficiently long, but this is of itself of slight consequence, and should not be considered by the patient, who must always be informed of what may occur from its continued use, if permanent benefit not accruing from other drugs is to be obtained. The urine should be examined from time to time during its administration, and on the appearance of albumin the silver should, of course, be discontinued. Nephritis has been noted from its too long use, as has also wrist-drop.

Among other drugs sometimes employed in the treatment of tabes may be mentioned gold and sodium chloride, ergot, belladonna, phosphorus, physostigma, aluminum chloride. They are, however, all of doubtful utility.

Electricity is of some service in tabes for its general tonic effect. Its employment is too often neglected in this connection. It, of course, has no curative value in tabes, yet may be of distinct utility employed in the form of general galvanism and faradism or combined general galvano-faradization in imparting general vigor and relieving certain of the symptoms. Large well-moistened sponge electrodes are used—one applied to the nucha, the second along the spine and over the abdomen and extremities; or the patient may sit upon a large pad, made by folding several thicknesses of towel over a zinc plate; the other electrode, a large sponge, should be stroked over the extremities, along the spine, and about the chest and abdomen. The feet may be placed upon a

similar pad and the upper electrode become the labile. The sitting should be ten to thirty minutes daily or alternate daily. The measured strength of galvanic current employed will depend upon size of the electrodes and susceptibility of the individual. In general, 10 to 30 ma. may be used. Should vertigo occur from galvanization, the strength of the current must be reduced. The galvanic current should not be interrupted, and should be controlled by a rheostat, so that shocks are avoided. The faradic current (the rapidly interrupted induced) is used as strong as can be agreeably borne.

Locally, faradism, especially the slowly interrupted current, is of utility, and should be employed as a stimulant to flabby muscles, especially if the patient is unable to take exercise. The faradic current is also sometimes of service in relieving attacks of fulgurant pains, but more especially the superficial pains. It is of great utility applied with the soft wire brush to the dried skin to overcome tactile anæsthesia. Atony of the bladder is often benefited by deep urethral or intravesical faradization, the indifferent pole being placed in the suprapubic region or on the perineum.

Counter-irritation to the spine by means of wet cups, blisters, or the actual cautery formerly was much employed in the treatment of tabes. It can be of service only in cases with pronounced meningeal symptoms. Blisters must in any case be used most cautiously, especially in those in which there is a tendency to trophic derangements.

A method of treatment which, when at first introduced, was too extravagantly lauded and subsequently has fallen into most unmerited disuse, is that by suspension. It has now few adherents, although in the writer's opinion no single remedy has been of more avail for the alleviation, and at least temporary arrest, of several of the most troublesome symptoms of the disease. As I have remarked elsewhere [1] concerning this method, the unfavorable criticism directed against it was largely due to illogical expectations as to what may be accomplished in a class of cases in which at the best no more than arrest of the disease process can be hoped. Cures were expected in ailments the very nature of which precluded such a possibility. Other critics, less of the *quid-nunc* than of the ultra-therapeutic order, with preconceived notions as to utility based on ignorance of *modus medendi*, refused to perceive a trace of good in the method, and held all improvement as either psychical or of post-hoc nature, either arising through the mental impression produced by the novelty of the method and the imposing apparatus employed, or through fluctuations which under no treatment characterized the disease in a large proportion of cases; or, finally, perhaps, through the influence of the drugs, such as potassium iodide, coincidentally used. The singularly warped judgment exhibited by critics for a method of such value is so generally manifest that too earnest a protest cannot be entered against it, preventing as it has its employment by many, and especially by those who depend less on their own judgment than on that of recognized authorities for knowledge of utility of therapeutic means. To illustrate how far prejudice may lead, reference must be made to a leading article which appeared some time ago in the *Lancet*,

[1] *Medical News*, Sept. 12, 1891: "On the Utility of Suspension in Disease of the Spinal Cord."

which doubtless has done much to bring the method of suspension into unmerited disrepute. In citing the failure of clinicians at a later date to obtain the higher percentage of improvement under the suspension treatment noted by the earlier reporters, and commenting upon what was styled the "negative results" of the former, the reviewer remarked that time had not been more kind to this mode of treatment than to many others which had been introduced to remedy intractable diseases; finally predicting that before long the suspension apparatus would be relegated to obscurity. These so-called negative results, with improvement noted in 25 out of 85 cases treated in Mendal's wards by Rosenbaum, represented improvement in at least 29 per cent. in an ailment the treatment of which under other methods is no more encouraging. This so-styled negative result really represented, as indicated by the figures stated, 29 per cent. of improved in 85 cases. Even this, as was remarked by Althaus, could scarcely be considered bad from a single method of treatment in a malady so intractable as tabes; and a more careful perusal of Rosenbaum's paper, in which the 85 cases reported showed, as Althaus pointed out in his criticism, that there were actually but 61 cases of tabes from which fair deduction could be drawn as to result, because of certain of the 85 suffering from other ailments, and of others who were under treatment too short a time for them to be utilized in the analysis. So that of the 61, improvement was shown in 25 cases, or 40 per cent.—as fair a percentage as that obtained by any other method of treating tabes.

Naturally, one places little credit on continued adverse results in the hands of those who both receive the method and pursue it with doubt and half-heartedness, and often with imperfect technique,[1] trusting its application to novices. This is more especially the case when abundant testimony of its value from personal experience and from that of earnest, unbiassed investigators is at hand.

Suspension apparently acts by separating and breaking down adhesions the result of the sclerotic and secondary inflammatory process, releasing compressed though still healthy nerve tissue. In cases in which tabes have become stationary, yet, with marked inco-ordination and pains present, permanent benefit can early be expected, and has been noted, from its use. The amount of elongation (stretching) of the cord by the method of suspension in vogue—*i. e.* by the head and arms—has been shown, in reality, not to be very great. As the results occur through the mechanical effects on the diseased tissue, and in many cases are probably *nil* solely through failure to obtain a proper amount of extension, several years ago I abandoned the use of ordinary suspension for the simpler method of forced extension of Bonuzzi. According to Bonuzzi, this method of extension produced an elongation of the cord perhaps three times greater than that obtained by ordinary suspension from the head and arms. This method is valuable also in that its simplicity robs it largely of the suggestive nature of treatment inherent in the old method of suspension by complicated apparatus. The method as suggested by Bonuzzi consisted in having the patient recumbent, with

[1] H. C. Wood (*The American Text-Book of the Theory and Practice of Medicine*, vol. i. p. 789), who questions the value of the method, recommends a daily suspension of from five to fifteen minutes!

head placed and maintained in an elevated position by means of a bolster. The lower extremities are flexed upon the body through a semicircle, the knees being placed upon the chest of the patient, the legs remaining straight. Subsequently the divergent feet, seized by the ankles, are carried strongly toward the floor.[1] The method so practised must be employed very cautiously at first, lest unpleasant symptoms arise. As used by myself, now since 1892, in a number of cases of cord disease in hospital and private practice, I have seen no ill result save temporary spinal ache, and in a large proportion of cases have noted great benefit following. The patient is placed recumbent on a couch in the dorsal position, with the head supported by a rather high pillow. The thighs are flexed upon the trunk and the legs upon the thighs. The flexed knees are now grasped by a rolled or closely folded small sheet or large towel, and with the operator at the head of the couch and supporting his position by placing one of his knees against the same, the flexed knees in apposition are cautiously pulled toward the patient's forehead, so that the spine is strongly semiflexed. Should the traction upon the upper or lower spine be too great or too suddenly exerted, severe pain in the cervico-dorsal or dorso-lumbar part of the cord may be complained of. Traction must then be slowly relaxed. If but little discomfort is experienced, or that felt is shown by a further trial to have no ill effect, as has been invariable in my hands, the body is maintained in this position, with the flexed knees resting either at the chin or upon the brow, or even beyond the head, from one to four minutes. The flexion must be so done as to be at least slightly felt in some portion of the spine. The patient subsequently rests quietly recumbent for a half hour or more, and should take little or no exertion the remainder of the day. The proceeding is repeated three times weekly, and so continued steadily for two to three months, after which, as in ordinary suspension, should occur an interval of about similar or less time.

A case recently so treated deserves brief mention here: T. W——, aged thirty-four; Englishman; first seen in March, 1893. Had had syphilis ten years previously; no subsequent history of the disease. Came to America in 1890. Health then began to fail. He suffered from indigestion and had fainting spells. In the spring of 1892 had severe headache, with dimness of vision, lancinating pains, and slight inco-ordination. He then consulted a physician, under whose treatment he remained for eighteen months, taking steadily mercury and potassium iodide. This plan of treatment had greatly increased his debility, and had been apparently without influence for good on the spinal symptoms. Headache had disappeared, but lancinating pains had continued paroxysmally. When seen they had been rather constant for four months. He had then totally absent knee jerk (even to reinforcement), marked static and moderate dynamic ataxia, and slight tactile anæsthesia. There was increased sexual desire. No other symptoms of the disease evident. No indications of neuritis. No electrical alterations. Smoked immoderately and indulged somewhat to excess in venery. Was very sleepless. Could walk quite long distances without fatigue, despite the ataxia. Walking at times lessened the leg pain. His habits were carefully corrected; tobacco entirely suspended. Chloride of gold and

[1] See the *Revue Therapeut. générale et thermale*, 1892, No. 2, p. 25.

arsenic ordered. Various drugs were at first tried for the relief of pain, but without result. Extension of the cord begun by Bonuzzi's (modified) method Jan. 15, 1894, and repeated subsequently at intervals of four days until four extensions were used, then about three times weekly; duration, one and a half to two minutes. Perceptible strain was always felt along the spine during and subsequent to stretching.

No severe pains occurred after the second suspension. Gait quickly and visibly improved, as did the static ataxia. Patient himself regarded the prompt change wrought in his condition by so simple a method of treatment as extraordinary. All medical treatment was suspended in a short time, and that by suspension alone continued. The patient was under observation until a short time ago, about two months from this writing (Feb., 1896). Extension of the spine had been intermittently continued at his home by a relative. Pains, which had ceased entirely very soon after the treatment by extension had been begun, had not returned, and all ataxia save slight static remained absent. The knee jerk has continued absent. The case may thus be regarded as an arrested one of tabes in the early part of the second stage. In many cases so much cannot be expected. Concerning the influence on symptoms of extension of the spine when applied preferably by the method just described or by that with the arm and head apparatus, my experience has been that, as a rule, pains are greatly lessened, and not infrequently are entirely removed, and that inco-ordination, if not too long existing, is also greatly ameliorated, though much less often entirely removed. Urinary incontinence is often improved or cured, as is loss of sexual desire or ability. The method seems of most utility during the second stage of the disease. I have never noted the return of knee jerk recorded by other observers in a few cases. It is probable that the utility of the method might be still further enhanced if small doses of mercury or of potassium iodide were given coincidentally. Especially might this be in those cases in which a myelitic or meningitic condition accompanies sclerosis. The influence of these drugs on the subinflammatory condition would thus be more decided, one might suppose, than if employed without extension. I have, however, never employed mercury or potassium iodide in a case of tabes while treating by extension, lest this should introduce an element of doubt as to the cause of beneficial result.

Treatment of tabes by the use of hypodermic injections of the glycero-phosphate of calcium or with sodium phosphate has been advocated recently, and good results reported. The former of these is asserted to be the ingredient on which the virtues of the testicular extract are based. This is, however, very doubtful. Some extended trial with the glycerophosphates by mouth ingestion has not convinced me that they are of utility in the conditions in which their employment is urged. The method first suggested by Brown-Séquard in 1889, which brought on his head great and undeserved ridicule, that of the hypodermic use of a sterile testicle extract, has in more recent time received rather general praise in the treatment of various forms of nervous disorders, among which was notably tabes. Although the writer has had but little personal experience with this remedy in tabes, his special acquaintance with it being limited to its use in general neurasthenic conditions, he has always regarded it with favor, believing that much symptomatically might

be expected from its employment, especially in those cases in which indications of cessation of testicular activity on the part of the patient exists. In these, apart from any influence the agent may have upon the symptoms, analogy indicates it may be of special service as a recuperative agent. This could be regarded as established were it certain that a function of the testicle, as of certain other glands of the organism, is to furnish, besides its external secretion, an internal one necessary for the maintenance of perfect health.

Considerable literature, and more especially French, now exists on the utility of the Brown-Séquard treatment in diseases of the nervous system, and especially in tabes. Space forbids special review of the subject here. Suffice it to say that apparently reliable testimony is on record of cases of tabes arrested by this means, and very numerous examples of marked symptomatic improvement.

Recent experiments in a series of 19 cases of tabes, extending over some time, at the Infirmary for Nervous Diseases in Philadelphia, by F. S. Pearce, under the direction of S. Weir Mitchell, tends to support the claims made by the French for this remedy. Every effort was made to exclude the effects of suggestion, the patients being unaware of the nature of the remedy employed. Pearce notes remarkable stimulating and temporary rejuvenating effects in locomotor ataxia. Symptomatically, pain and crises were relieved for a time and inco-ordination and sexual power helped. This remedy, therefore, seems well worthy of extended trial in tabes. It is highly important that the preparation employed, besides being of unquestionable activity, should also be aseptic, lest abscess result from its use. The greatest care should also be observed as to the cleansing of the syringe, needle, and site of puncture prior to each injection. The injections should be made daily or on alternate days. The fluid should be thrown into the tissues of the back, loin, or the nates. As the injections are quite painful, prior refrigeration of the area of puncture with ether spray or by the application of ice is to be recommended until tolerance to their use, as concerns sensation, is established.

Mention should be made of a preparation of a combination of animal extracts of considerable merit which has recently come into favor as a powerful reconstructant in debilitated, and especially in neurasthenic, conditions, and hence which presumably may be of utility in tabes. It is a combination intended solely for mouth administration, and is made from brain, spinal cord, and testes, the extractive matter of which preparation is asserted to contain a definite proportion of lecithin ($57\frac{3}{10}$ per cent.) and of spermine, nuclein, and cell nucleins of so-called phosphorized albumins ($43\frac{7}{10}$ per cent.). This remedy, which, although it is a proprietary preparation, is non-secret and non-patented, and is, so far as I am aware, unadvertised, is prepared under the name of "syrup of di-olyl lecithin (phospho-albumin)." It was brought to my attention only recently, but in the somewhat extensive trial I have already made of it in neurasthenic conditions I have found it unequalled by any other single remedy or combination of remedies as a bracer, not excepting the hypodermic use of testicular fluid, than which it is much more practicable of administration. It is well worth a trial in tabes.

Treatment of Special Symptoms.—Special treatment is usually required for the relief of the lancinating pains in tabes. The influence of spinal extension (suspension) is oftentimes quite extraordinary in not only jugulating obstinate paroxysms, but in preventing their early return. This should be borne in mind before drugs are resorted to, the ultimate effect of which, when more or less constantly used in so chronic a disorder, cannot but have an evil influence on bodily well-being. The drugs that afford most relief, excepting of course opium, are, in the order named, phenacetine, antipyrine, exalgine, acetanilid, and sodium salicylate. These may be used singly or in combination. Phenacetine is now the favorite. I have employed it frequently with good results. As with antipyrine, if used singly, it requires to be administered in a full dose (10 gr.), repeated perhaps three or four times at hourly intervals. Antipyrine and phenacetine in combination seem often more serviceable than either drug alone. These should be employed in doses of 5 to 8 grains each. A good preparation of cannabis indica is often also of use in relieving the pains when the latter are not too severe. Usually it is best administered in combination with one of the above in doses of $\frac{1}{2}$ to 2 minims of the fluid extract or gr. $\frac{1}{8}$ to $\frac{1}{2}$ of the solid extract.

It should be remarked that disappointment in the use of this remedy in other affections than tabes is frequently due to the preparation employed being inert. Apparently a reliable preparation may be recognized by noting if the precipitate, when the alcoholic preparation is added to water, is of a decidedly green hue; if brownish, the preparation is probably worthless.

Local remedies are usually of scant avail for the relief of true lancinating pains, since the latter are usually too deeply situated and of too great severity to be easily influenced. For more superficial pains, especially if localized, relief may be often obtained by the application of a lotion of menthol in alcohol (gr. 10 to 30 to the ounce), or by the use of the following liniment:

Menthol,		
Chloroform,	*āā.*	1–2 dr.;
Oil of cinnamon or oil of cloves,		$\frac{1}{2}$ dr.;
Soap liniment,	up to	$\bar{3}$j.

The tincture or the oleoresin of capsicum may be substituted for the oil of cinnamon or cloves, but added in smaller amounts (5 to 10 minims). A combination of oleate of aconitia (2 per cent.), menthol, of each 2 drachms, tincture of capsicum, $\frac{1}{2}$ drachm (or the oleoresin, ♏xv), in chloroform 1 ounce, may be painted over the painful area. This is of use for the relief of superficial neuralgias. Or a small quantity of chloroform alone, sprinkled on spongiopiline or on lint, may be of use.

Warm baths and the warm pack sometimes exert a decided sudorific influence during the paroxysm of pain. They are worthy of trial.

In cases of tabes yet at a period at which arrest of the disease, with return to usefulness, seems at all possible, opium or morphine should of course be only exceptionally resorted to until other means of relief have

been tried without result. Morphine hypodermically is often necessitated for the alleviation of the more intense paroxysms, especially for the gastric crises. Hypodermic administration should not be entrusted to the patient, and its employment by the physician should be as infrequent as possible. If morphine is given by the mouth, the patient must be cautioned against its steady continuance, the danger of the opium habit being depicted to him most vividly. Of course if no real benefit is obtained by other remedies, the drug must be freely used for effect.

For the relief of the painful crises, if severe, as is usual, morphine hypodermically, in full doses, is necessary. Little else can be of avail in paroxysms of tabetic gastralgia. A trial may also be made of sinapisms to the epigastrium and nucha, or, if the attack is prolonged and unyielding to these, small blisters may be applied in the same situation. To check vomiting often nothing save morphine hypodermically is of avail. There may be tried, also, however, nitro-glycerin (1 or more drops of the 1 per cent. solution on the tongue every half hour); 20 per cent. solution of menthol in olive oil, of which 10 drops are ingested on a small quantity of crushed sugar, with a sip of water; or menthol may be prescribed in brandy, to be taken on a little crushed ice. These and other remedies suggested for the relief of gastralgia and vomiting, such as dilute hydrocyanic acid taken with bismuth subnitrate and spirit of chloroform in mint water, are all of far more value to relieve simple nausea than to check the severe cramp or obstinate vomiting attending a gastric crisis in tabes.

For the relief of laryngeal spasm free inhalations of amyl nitrite, or nitro-glycerin hypodermically, may be used. Morphine, also hypodermically, may be required.

Treatment of the Ataxia.—This, as remarked, is often materially helped, and, indeed, in a fair number of not too advanced cases even largely, at least temporarily, removed, by the employment of extension of the spinal cord. Mention must here be made of the treatment of the ataxia by the use of what is termed compensatory gymnastics, introduced by Fränkel, and now attracting much attention abroad. By the steady, judicious employment of these exercises marked improvement in inco-ordination in cases of advanced ataxia has been lately recorded. This method has for its object the execution of certain definite movements solely with the purpose of re-education of the impaired or lost muscular sense.

As described by Belugou,[1] who has applied this method with success, these movements are: "No. 1 consists simply of flexion, extension, abduction; No. 2, of simple, co-ordinated movements in a definite direction against resistance; No. 3, of combined co-ordinated movements. The first of these consists of the garter, which is applied round one leg at the level of the knee. On this is a small piece of wood, which is fixed to the front of the patella by the garter. The patient then raises the other leg; then, flexing it, brings it rapidly down so as to touch this piece of wood with the heel. Another movement is obtained by holding a stick just above the middle of the legs. The patient then passes one foot alternately over the stick and back again without touching it. A modification of this is the hoop, through which one foot

[1] *Archives gén. de Méd.*, Feb., 1896, and also *Brit. Med. Journ.*, epit., Mar. 28, 1896.

is passed without touching it. The advantage of this is that it can be held in different positions so as to combine the action of various muscles. For the upper limbs many of the same exercises are employed, but in the third class a special series is used, necessitating delicate co-ordination. One of these consists in suspending balls of different sizes in a frame at different heights; these are put in motion, and the patient is obliged to seize each moving ball."

An excellent review of Fränkel's work, with the results obtained, has appeared in the Paris letter to the *Medical News*, April 3, 1897. These results have so important a bearing on the treatment of the most distressing symptoms of the later stages of tabes that they should claim our earnest attention. It is narrated that certain of the worst cases of ataxic inco-ordination at the Salpétrière, two of which had been bedridden for years, were distinctly improved under the use of Fränkel's method. It is stated that "one of these, a woman who had not stirred from her bed for six years, now is able to go out into town alone with the help of a single cane. She can go up and down stairs, and can even walk with her eyes shut." Naturally, the Argyll-Robertson condition of the pupil and the absent knee jerk persists as before. It appears that Raymond's results with the method in cases seen at his clinic in all stages of the disease gave 75 per cent. in which distinct improvement occurred. The following is quoted from the Paris letter:

"The method is an extremely simple one, and consists only in teaching the patients to make the movements necessary for any given action with exactitude. In walking, for instance, the patient is made to stand up as straight as possible, and to advance one foot a certain distance, but without slinging it, and then to place it once more beside the other. Then he puts it a certain distance forward and to one side, and takes it back again; then to the rear, and replaces it. Then he goes through the same movements with the other foot. At first his feet will need the constant supervision of his eyes, but after a while he will be able to dispense with that. Gradually his eyes may be fixed farther and farther away from his feet, but on some fixed object that will help his equilibrium, until finally the walls of the room at the height of his eyes will be sufficient, or even the ceiling, and then with eyes shut he can accomplish all the movements. The important points with regard to the exercises are that they should not be overdone, and should be carried out with the greatest possible exactitude. The avoidance of over-exercise is very important, because ataxics, owing to loss of muscular sensibility, are devoid of proper sense of muscular fatigue, and may easily be encouraged to pursue exercises that cause exhaustion rather than education of muscles. The impaired nerve paths quickly resent the passage of unaccustomed impulses, and the intensity of attention required to carry out the exercises properly soon induces cerebral fatigue. For these reasons Fränkel insists that patients should not be asked to exercise for more than ten minutes at a time, with an interval of at least ten minutes, after which the motions may be repeated once more, usually not oftener. Twice a day such a séance of a half hour may be given. Exactitude in the accomplishment of the motions decided upon is the whole secret of the treatment.

"When patients are bedridden or when they are not easily able to

stand up during the exercises the purpose of the motions may be made more definite by putting around one leg a garter to which, in place of a buckle, is affixed a metal plate. The heel of the other foot is made to touch the plate, placed at different heights on the leg, until the patient can accomplish the purpose readily even with eyes closed. Variety in the exercises is to be sought for, for they must be continued for weeks, sometimes for months, and Fränkel insists that the true end of the exercises must never be allowed to sink out of view. Not muscular exercise in the ordinary sense of the word, such as may be obtained from the repetition of any movement, is the end, but the re-education of muscular attention by the careful repetition of movements with an exact and definite purpose.

"Usually patients themselves are very much encouraged from the outset. At the end of about two weeks there is a distinctly noticeable improvement in the co-ordination of movements which they have been practising, and this improvement continues for some time, until a certain stage of co-ordination, dependent on the extent of the disease, is reached. Whatever is gained remains as long as the disease itself remains stationary. Further advance in the disease will demand renewed education of the muscular sense, and at times the progress in co-ordination will render latent the extension of the sclerotic process in the cord.

"For the more exact co-ordinate movements required of the hands—such as writing, for example—much finer and more detailed exercises are required. It is surprising to see, however, how much improvement may be obtained in handwriting after a careful course of exercises in tracing various curved, spiral, and angular figures. In other relatively delicate movements for ataxics, such as grasping small objects, buttoning clothes, etc., the same improvement is noticeable after a course of exercises directed more especially to these ends, such as grasping at a given signal variously colored swinging balls or picking out variously sized objects from a number scattered over the table.

"As to the cases in which the treatment, when applicable, does not succeed (and they are rare), they are mainly those in which the intensity of attention required to direct the movements through the diseased nerve fibres is too great for the patients to accomplish the effort demanded of them. Fränkel points out that they are almost always people who have been characteristically awkward in their preataxic life, and whose general muscular sensibility is supposably congenitally impaired, capability for delicate movements being the nice adjustment between muscular sense and muscular motility.

"In a certain number of cases the treatment is distinctly contraindicated. In acute tabes, where the pains are almost continuous, it would do harm rather than good, and when tried has always led to aggravation of symptoms. In patients suffering from arthropathies it is also contraindicated, or where for any reason there is suspected to be present that abnormal friability of the bones that causes so-called spontaneous fracture. Where severe vesical trouble exists, with a tendency to frequent recurrence of vesical crises, absolute tranquillity is the only thing for the patient. Any amount of movement leads inevitably to aggravation of the extremely troublesome symptoms."

Impotence in tabes is sometimes very materially improved by suspension. Strychnine, zinc valerianate and phosphate are of value. Injections of testicular fluid seem to have considerable power in restoring tone to the flaccid organs and of promoting a return of procreative power. Suspension, strychnine, and the Brown-Séquard treatment are also of utility for the bladder weakness. Careful attention from the first must be paid to the condition of the bladder, the patient early guarded against the imminent danger of prolonged retention, difficult often to recognize in tabes unless on the watch, because of the attending anæsthesia of that viscus. The patient must be taught the use of the soft catheter, which should be employed at stated intervals daily if free voluntary evacuation of the contents of the bladder be impossible. The strictest injunctions must be laid down as to cleanliness in the use of the catheter, it being thoroughly washed in weak carbolic-acid solution or in saturated boric-acid solution, and either kept in the latter or again washed in the boric-acid water, or preferably in boiling water, before use.[1] Should cystitis have occurred, every effort must be made to control the inflammatory condition before implication of the pelvis of the kidney in the process occurs.

The bladder should be cleansed daily or on alternate days by irrigation with a boric-acid solution of about half saturated strength, or with any other simple, non-irritating antiseptic solution. In cystitis, with painful micturition, infusion of buchu containing tincture of hyoscyamus, minims 10 to 30, and spirits of nitrous ether, $\frac{1}{2}$ drachm, is of value, as will be found also in all chronic forms of the affection the fluid extract of pichi, in doses of a $\frac{1}{2}$ drachm to 1 drachm, three times daily. Salol, combined with pichi or with a few drops of a reliable sandalwood oil, is of great service in chronic cystitis.

For pain accompanying cystitis or for the relief of neuralgia of the neck of the bladder or of rectal spasm the remedy of promptest utility is a rectal suppository containing cocaine muriate, gr. $\frac{1}{3}$ to 1; extract of hyoscyamus, gr. 1 to 3; iodoform, gr. 3 to 5. The combination of morphine with the above, while it will unquestionably increase its immediate efficiency, is not usually essential. Incontinence of urine may perhaps be controlled by the judicious use of suspension and by the administration of full doses of belladonna and strychnine.

Attention to constipation, which is common in tabes, is essential. Accumulation of fæces in the lower bowels must be swept out by copious warm-water enemas and the stools maintained in a suitable condition by small doses of cascara, or of aloin preferably combined with strychnine and belladonna or hyoscyamus.

Because of the tendency to disturbance in the trophic nervous system in tabes it is most important to caution tabetics against permitting a corn to be cut or allowing blisters on the feet to go unattended to.

In place of cutting the corn a method commonly employed is to soften the epidermis with an alkali and then rub it off with a piece of pumice-stone.

The onset of optic atrophy necessitates the most careful attention to

[1] In urging the use of the catheter in tabes Gowers states that while he has seen many melancholy instances of death in consequence of unsuspected retention, he has never seen any case in which the early and frequent use of the catheter did harm.

the general health, the avoidance of sexual excess and the too free use of tobacco. Strychnine should be tried in full doses, and the Brown-Séquard treatment or the use of the testicular, brain, and spinal-cord combination mentioned previously employed (p. 209).

PRIMARY LATERAL SCLEROSIS.

SYNONYMS.—Primary sclerosis of the crossed pyramidal tracts; Spastic paraplegia: Spastic spinal paralysis; Erb's palsy; Spasmodic tabes dorsalis (Charcot).

DEFINITION.—Primary sclerosis of the crossed pyramidal tracts.

This is a somewhat rare system disease. Its presence is characterized by a group of symptoms common to a number of ailments with which it is very frequently confounded. These symptoms are chiefly motor weakness and rigidity in the lower extremities, with marked heightening of the deep or tendon reflexes, but without muscular wasting or sensory disorder.

The occurrence of this disease as a primary affection, since first described by Erb in 1875, and the extreme paucity of necropsies in these because of the disorder not appreciatively shortening life, has led many to question if the ailment really deserves a place in neural nosology. It is indeed questioned if such a disease really exists, since a necropsy has not shown it[1] in an uncomplicated form. But, although the ailment is of rare occurrence and mistakes in pathological diagnosis are common, good authority has established that it deserves a place in the nomenclature of cord affections.

ETIOLOGY.—The infrequency of the occurrence of the disease as a primary affection has left its etiology largely a matter of conjecture. The onset of the disease is least uncommon in adult males between the ages of twenty-five and forty-five years, although it is said typical cases have been encountered earlier and much later than these extremes. Unlike the case with posterior sclerosis, syphilis is in no wise a causative factor. The cases in which syphilis is operative are really those of secondary sclerosis, the result of local syphilitic myelitis, originating secondary degeneration of the lateral tracts, one of the common forms of so-called spastic paraplegia.

The determining causes are generally stated to be traumatic injury (spinal concussion), long exposure to cold and wet, over-use of the affected muscles, and lactation. It is doubtful in what manner these are operative. The pathological character of the disease would indicate that it may have origin in a toxic blood state. Bramwell suggests that the limitation of the disease at first to the crossed pyramidal tracts, with onset at the lower part of the cord, indicates as a prime etiological factor defective nutrition of the cortical cerebral centres. The fibres of the pyramidal tracts are of great length, and, deriving their nourishment from the cortex, could be easily subjected to disease in their lower end, at which, too, the blood supply to the cord is least well maintained.[2]

[1] Dana: *Text-Book of Nervous Diseases.*

[2] See Bramwell: *Diseases of the Spinal Cord*, Edinburgh, 1895, p. 131.

It is not improbable that chronic lead-poisoning as a factor in the production of the disease is too little recognized.

Pathological Anatomy.—Necropsies in pure cases of this variety of cord sclerosis have been very infrequent. This is due to the fact that our knowledge of the disease is of comparatively recent date, and that the ailment does not materially shorten life. Until recently the only case quoted was that of Dreschfeld,[1] in which, with long-continued symptoms of the disease, there was found post-mortem what appeared to be sclerosis of the crossed pyramidal tracts. Slight changes in the anterior cornua were also present, but were regarded as of supposedly secondary occurrence. Déjerine and Sottas[2] very lately report a case with typical symptoms in which, death occurring from pneumonia, the sclerosis was found to be practically limited to the lateral columns.

The sclerosis in this affection involves the fibres of the crossed pyramidal tract, the chief function of which is conduction of voluntary motor impulses from the cerebral cortex to the multipolar cells in the anterior cornu. As a result of the sclerosis, therefore, there occurs more or less motor paralysis in all parts below the seat of the lesion, and, as the affection is bilateral, the palsy is paraplegic. The separation of the anterior cornual cells from the pyramidal fibres removes these from the inhibitory influence of the cerebrum; in consequence, reflex action is no longer perfectly, if at all, under cerebral control. As a result, marked heightening of the reflexes and rigidity of the muscles occur. This fact is regarded by many as the cause of the condition of rigidity and spasm occurring in the affected muscles in sclerosis of the pyramidal tracts. It is questionable, however, if mere removal of cerebral inhibition is the true explanation of this phenomenon, and if, indeed, the actual cause is not more likely a local one, such as might occur from the irritation produced by the lesion on the adjacent centres for reflex action, the multipolar cells, inducing in these likewise a condition of irritation.

Gowers believes that the degenerative process probably has origin in the terminal fibres of the crossed pyramidal tracts in the gray matter of the anterior cornu, and that from these fibrillæ the sclerosis subsequently pursues an upward course in the lateral column. This may explain the absence of readily discoverable lesion in an early case presenting symptoms of the disease dying from an intercurrent malady. An affection of these parts is difficult to detect, as the diseased fibrillæ would be interlaced with similarly healthy fibres. In functional curable cases it is probable that these fibrillæ are chiefly, if not alone, affected, mere molecular alterations occurring, the nature and cause of which are purely conjectural.

In many cases of lateral sclerosis a sclerotic process shows early tendency to extend beyond the lateral columns of the cord. The sclerosis may involve the multipolar cells of the anterior cornu, thus originating, in addition to the condition of spastic paraplegia, those of progressive muscular atrophy (amyotrophic lateral sclerosis); or, more commonly, a combined sclerosis of the lateral and posterior columns may occur. Occasionally lateral sclerosis is but an early indication of a disseminated degenerative process in the cord.

[1] *Brit. Med. Journ.*, Jan. 29, 1881. [2] *Comptes rendus Soc. de Biologie*, Dec. 6, 1895.

Symptoms.—The disease is of very gradual onset and most chronic course. The earliest symptoms are usually weakness and stiffness and a tendency to momentary spasm in the muscles of the lower extremities, most present after exertion. The stiffness and spasm also are very noticeable after rising on first attempting to use the limbs, and are distinctly aggravated by exposure to cold or to external irritants or by physical over-exertion. This condition increases very slowly; walking becomes progressively difficult, and the early slight stiffness of the limbs gradually deepens into a state of decided rigidity and spasm, associated with a great increase of activity in the reflexes and the development of certain of those not normally present.

The gait in a typical case of spastic paraplegia is most characteristic. The assistance of a cane or canes is necessary. Rather short steps are taken, and these with difficulty, the active limb being raised by an effort on the part of the upper and lower trunk muscles, with little or no flexion at the knee or hip. The leg is dragged rigidly forward, and in its progress, with a tendency to interlocking of the knees and adduction of the foot as the latter is spasmodically advanced, the ball and toes of the feet scrape the ground with each step. Often in progression the contact of the ball of the foot and toes with the ground develops a clonus in the limb, which, too, is common with the patient sitting when the ball of the foot rests insecurely on the ground. In a well-developed case loss of power is very apparent, most noticeable in the flexors of the hip, knees, and ankles. The muscles of the legs are rigid and usually very well nourished.[1]

The muscular tension and spasm are so decided in the legs oftentimes that in recumbency, with the legs extended, passive flexion at the knee is impossible, and passive elevation of one leg causes its fellow to ascend with it. The spasm is chiefly in the extensors, and is most manifest while these are in activity (while the limb is extended). After flexion at the knee has been accomplished the spasm may entirely relax, to at once return on a second extension at the knee. This sudden departure from the condition of flaccidity of the leg, with the knee flexed, to one of rigidity and spasm on its extension, has been aptly comparable to the backward fly of a penknife blade by virtue of its spring after having been almost completely opened by the hand. It has thus been termed "clasp-knife rigidity."

The reflexes, superficial and deep, in the affected part of the cord are in a condition of greatly increased activity, although the muscular spasm often present may not always permit more than a partial demonstration of this increase. Heightening of the superficial reflexes in the limbs may be shown only by an increase in the tonic spasm or rigidity, already present, on stimulation of their various centres. The plantar reflexes may be so heightened, however, that with the patient in recumbency stimulation of the sole will throw the legs into a condition of partial tetanus.

The knee jerks are invariably increased, and commonly very markedly so; a slight tap on the ligamentum patellæ—or often, in fact, on any portion of the knee or on the thigh muscles—will cause the leg to

[1] More than slight muscular wasting does not occur, save in those cases in which there is involvement of the anterior cornual cells.

quickly fly out. Clonus of the rectus may often be developed by sudden depression of the patella with the fingers, the patient being in recumbency. Typical ankle clonus is usually present. It is best obtained by incomplete extension of the thigh and knee with the patient sitting. The leg is held at the calf with the left hand and semiflexed at pelvis and knee in order to obtain as complete relaxation as possible; the ball of the foot near the toes is now quickly and lightly grasped by the right hand and a sudden energetic dorsal flexion made and maintained. Sudden stretching of the tendo Achilles after this manner will usually result in the production of ankle clonus, a phenomenon never observed in health. The clonus consists of rhythmical series of rapid alternate contractions and relaxations at the ankle, continuing so long as the pressure on the foot by the hand is maintained.

In a later stage of the disease with the coincident upward progress of the sclerosis the arms may become affected similarly to the legs. Stiffness and rigidity, with muscular weakness and heightening of the reflexes, associated here, however, with less paroxysmal spasm, are then manifest. Commonly one arm is more affected than its fellow. Heightening of the reflexes is shown by exaggerations of the triceps and wrist jerks, and the presence and increase of other periosteal and tendon reactions. A jaw jerk or clonus may then be obtainable. Involvement of the trunk occasionally occurs, shown by attacks of spasm in the abdominal and dorsal muscles.

The electrical reactions in the affected muscles are practically unaltered save perhaps in the manner of simple increase to both currents in the early and middle stages of the disease, and to simple diminution in the later stage. The reaction of degeneration never occurs when the lateral columns alone are the seat of disease.

Sensory symptoms in uncomplicated cases are usually quite absent. Muscular aching may be complained of, occurring merely as the result of fatigue. Neuralgiform or rheumatoid pain sometimes occurs, as do occasionally subjective numbness and tingling. Appreciable anæsthesia indicates involvement of some part of the sensory apparatus in the disease.

Any marked affection of the sphincters is uncommon. Constipation is usual, and is due probably to lack of exercise on the part of the patient.

Sexual power is rarely lost. Eye symptoms are uncommon. Optic atrophy is regarded as rare.[1]

Complications.—These have been already alluded to in discussing the Pathological Anatomy of the disease (page 216).

Diagnosis.—Primary spastic paraplegia, as has been remarked, is a somewhat rare disease. Certain conditions mistaken for it are those which originate a secondary lateral sclerosis, such as in chronic dorsal myelitis, caries of the vertrebræ, or spinal tumor. It must also be differentiated from amyotrophic lateral sclerosis, postero-lateral sclerosis, disseminated cerebro-spinal sclerosis, and from hysterical paraplegia.[2] Its

[1] I have encountered it in two typical uncomplicated cases.

[2] As primary lateral sclerosis never occurs in childhood, its separation from double cerebral hemiplegia or from cerebral tumor occurring in children need not be here considered.

differentiation from all of these, save certain cases of the presumed hysterical form, should not usually be a matter of difficulty to the acute observer. In a case presenting symptoms of the diseases described inquiry should be first made as to the mode of onset and character of preceding symptoms. If the ailment has not been of gradual development, and sensory symptoms—such, for instance, as a girdle pain, detectable anæsthesia, muscular inco-ordination, muscular wasting or qualitative electrical alterations, paralysis of the bladder or rectum, or bed-sores—have been present or have later appeared, the case, although clinically a spastic paraplegia, is not one of primary or, at least, *uncomplicated* primary, lateral sclerosis. Its separation from the unilateral spastic paraplegia occurring as the result of cerebral hemiplegia need not be mentioned, since unilateral primary sclerosis of the crossed pyramidal tracts is unknown; were it not, the preceding history and the affection of the arm and face should leave no doubt.

The two ailments the separation of which may be most difficult are disseminated sclerosis in its initial stage and hysterical spastic paraplegia. Disseminated cerebro-spinal sclerosis in its early stage may for a long time simulate primary spastic paraplegia, as it may also pure hysteria. In these cases counterfeiting lateral sclerosis the sclerosis is evidently primarily limited to the crossed pyramidal tracts, and subsequently becomes disseminated. In such a case a mistake in diagnosis is at first inevitable. If this fact, now well recognized, of the not infrequent simulation of primary lateral sclerosis by cerebro-spinal sclerosis is borne in mind, the early recognition of the latter and the separation of the affections will be the easier, as a constant watch for other symptoms of the disease should lead to their prompt recognition and correct interpretation as they appear.

Hysterical paraplegia may present the typical symptom complex of lateral sclerosis, even to the presence of—although rarely—a true ankle clonus. Usually the history of the case, the occurrence perhaps of past hysterical seizures, the sudden onset of symptoms of the disease succeeding an emotional cause, the presence of areas of anæsthesia, and spells of remission in the contractions, and usually their complete relaxation during anæsthesia if this test should be tried, and the occurrence of contraction of the fields of vision, enable a separation to be made.

Concerning the question of sex so much stress may be laid as to totally mislead, since what is presumably lateral sclerosis (clinically, primary spastic paraplegia) is apparently scarcely less rare in women than in men. Cases are not infrequently encountered in women in which for a long time a differentiation may be simply impossible: this, my own experience, is that also of all who have had much to do with these diseases. A conjectural diagnosis alone in these cases can be made, which may be alternately adhered to with certainty, and again promptly abandoned *seriatim* as time passes. The exact status of the case may remain altogether in doubt until perhaps a cure suddenly occurs by means of some inert drug, on the use of which much faith has been pinned, or through the exertion of an outside mental influence. Even excluding questions of sex, it cannot be too strongly urged that a hasty diagnosis of hysteria should not be based merely on the presence of a few past or current hysterical indications, since, although organic

disease of the nervous system is frequently mimicked by hysteria, the contrary also is common. This fact is much too little recognized by the profession, the rank and file of which are often apt to regard symptoms of undoubted organic nerve disorder appearing in an emotional or hysterical woman purely as an indication of an hysterical functional condition. It seems certain that not infrequently cases that are diagnosed at first as hysterical spastic paraplegia, merely because they have occurred in young women of presumed emotional tendencies, and are perhaps accompanied by nervous symptoms which inferentially are regarded as functional, finally terminate as cases of typical disseminated cerebro-spinal sclerosis. These have frequently in reality been merely examples of organic nerve disease from the outset, in which the admixture of a few hysterical symptoms have served to mislead.[1]

Prognosis.—True primary lateral sclerosis, when sufficiently advanced to be recognized with certainty, is practically incurable. It is stated by some authorities that in its early stage not only arrest may be effected, but a cure obtained. It is doubtful if such cases are in reality those of the primary disease, or, if apparently so, whether the trouble has not been of the functional variety, not dependent upon an actual sclerotic process. Yet even in a well-developed case arrest of the disease may often be accomplished and much improvement brought about. As concerns life, the prognosis is most hopeful, since the tendency of the disease to hasten death is almost *nil*.

Treatment.—Save in a few particulars the management of the disease so nearly resembles that of locomotor ataxia that no lengthy separate discussion is necessary. If there is doubt based on good evidence, however slight, as to whether the case is a primary lateral sclerosis or a spastic paraplegia secondary to a chronic myelitis or to a localized growth of specific origin, leuetic treatment, as outlined in discussing the simulation of tabes by cord syphilis, should be tried. Attention to the general health, as in tabes, is most essential. Especial care in the treatment of primary lateral sclerosis must be taken to avoid exposure to cold, and prevent, if the patient is yet able to walk about easily, muscular over-exertion, both of which are very distinctly harmful in this disease.

No curative drugs for the affection are yet known. It is probable that, as in tabes, the employment of courses of silver nitrate, perhaps alternated with arsenic, may be of service in arresting the progress of the disease. Spinal counter-irritation by blisters or the actual cautery is also thought to be of some utility. Strychnine should be employed with great caution, if at all, because of its influence in heightening reflex irritability. Galvanism is useless, and faradism is harmful in this disease. No especially helpful drug is at hand to control the condition of heightening reflex irritation and spasm. Potassium bromide or hydrobromic acid perhaps enjoys the most confidence in this particular. As before mentioned, great stress must be laid on the avoidance of muscular fatigue. Gowers has found that attention to this often permits drugs to do good which otherwise seem useless or harmful. Gowers has noticed great improvement from courses of rubbing, espe-

[1] For a most interesting account of the Simulation of Hysteria by Organic Disease of the Nervous System see an address by Buzzard, published by Churchill, London.

cially upward friction, instead of mere kneading of the muscles. He conjoins this with a course of Turkish baths. The latter may be readily employed in a modified but distinctly beneficial form by the use of the portable and inexpensive cabinets now on sale at the shops.

Although suspension is much less distinctly of service in this disease than in tabes or postero-lateral sclerosis, I have occasionally seen most gratifying results from its employment.[1] It is always worth a trial, and should preferably be used by the modified method of extension, which requires no apparatus, described under the Treatment of Tabes (p. 206).

POSTERO-LATERAL SPINAL SCLEROSIS.

Synonyms.—Progressive spastic ataxia; Ataxic paraplegia; Combined fascicular sclerosis.

Definition.—A form of disease of the spinal cord in which sclerosis of the posterior and lateral columns occurs, the symptoms of which are those of lateral sclerosis usually preceded and always accompanied by those of motor inco-ordination.

It is questionable if this affection should be termed a system disease, since in other than very rare forms it is probably either dependent upon a preceding dorsal myelitis with secondary degeneration, or is an atypical form of tabes in which overflow of the sclerotic or subinflammatory processes occurs into the lateral columns.

Etiology.—Its etiology, when not dependent upon chronic myelitis or the causes that produce tabes, is doubtful. It occurs most frequently between the ages of thirty and fifty years, and is more frequent in men than in women.

Pathology and Pathological Anatomy.—Since the disease in the great majority of cases is, as remarked, that of tabes with overflow of the sclerotic processes into the lateral columns, or is secondary to a chronic dorsal myelitis in which secondary degeneration has occurred, the anatomical picture is most often that of one or the other of these diseases. In rare cases in which a necropsy has been had, and which are regarded by a few observers, such as Gowers, as representing a distinct system disease, there is supposed to have occurred a coincident primary sclerosis of the posterior and lateral columns. That in the posterior columns is apt to be more intense in the dorsal than in the lumbar part of the cord, and involves but little the root zone of the postero-external columns, in both of which features it differs from pure tabes. The degeneration in the lateral columns, even in these cases, is most often not strictly systemic, although it chiefly involves the crossed pyramidal tract fibres. It often extends into the mixed zone of the lateral columns, and may invade the lateral limiting layer situated between the pyramidal tract and the gray matter.

Symptoms.—The symptoms of this disease are essentially those of

[1] See especially Case IX. in my paper, "Report on the Treatment of Fourteen Cases of Disease of the Spinal Cord by the Method of Suspension," *Medical News*, June 1, 1889.

spastic ataxia, with which there is combined inco-ordination of movement in the lower extremities. It is a disease of very gradual onset. The early symptoms are much those described under Spastic Paraplegia, but with the gradual oncoming and stiffness in the legs there is early associated quite marked ataxia, such as is present in tabes. This last may at first be the most obstrusive symptom, and, rarely, may alone direct the patient's attention to his ailment.

The gait in a fully-developed case resembles a combination of that of tabes and lateral sclerosis. There is unsteadiness in walking, but the legs are not raised so high or brought down so suddenly as in tabes. The toes may first reach the ground and drag as in spastic paraplegia. The ataxia is usually obtrusively shown, as in tabes, in walking or in attempting to turn suddenly and in standing with eyes closed and feet together. It is likewise brought out in recumbency by having the patient attempt to touch an object with his toes, the position of which object he has first viewed before closing the eyes.

In a well-developed case there is usually, as in primary spastic paraplegia, a condition of increased muscular tension and tendency toward spasm in the legs, with marked heightening of the reflexes, chiefly the deep. The knee jerk is much exaggerated. Ankle clonus is commonly easily elicited. The limbs remain well nourished, although muscular weakness is more or less prominent as the disease progresses.

Sensory symptoms are slight. Lightning pains do not ordinarily occur, and any marked anæsthesia is uncommon. The motor weakness in the early stage may give rise to pains in the legs from fatigue on excessive use of the limbs. Not uncommonly dull pain in the sacral region is felt. In cases secondary to myelitis girdle pain is common. Sexual power is lost early in many cases, and paresis of the bladder may occur. Constipation is usually present.

Eye symptoms, such as the Argyll-Robertson pupil and optic atrophy, are not usual, nor are the vaso-motor and visceral derangements which occur in tabes.

Later in the course of the disease the spastic symptoms usually become so conspicuous that the ataxia, which ceases after a time to advance, is little recognizable, and the case may be mistaken for one of advanced lateral sclerosis, or, with increasing paraplegia, it may come to resemble one of advanced myelitis with contractures. Less commonly spastic symptoms, which in a few cases have never been very prominent, may disappear, the knee jerk ceasing to be obtained. The case then resembles one of tabes.

The symptoms, after being limited for a considerable period to the lower extremities and trunk, subsequently may involve the arms, and rarely the face. There is, then, as in the leg, ataxia of the implicated muscles, with a tendency to rigidity and spasm and increase in the reflexes.

Diagnosis.—As before remarked, many of these cases are those of atypical tabes or represent a degenerative process resembling a chronic transverse myelitis, so that in many instances they cannot be distinguished from these affections. In its early stage, because of the marked ataxia, the disease might readily be mistaken for posterior sclerosis, were it not that preservation, with usually excess, of the knee jerk and

slight or decided ankle clonus, with definite loss of power in the affected muscles, prevent error. Later the case may be regarded as primary lateral sclerosis if ataxia or a past history of it is not searched for. The disease is especially apt to be confounded with Friedreich's ataxia, the hereditary form of combined sclerosis—an ailment which occupies a position between postero-lateral sclerosis and pure tabes. But in Friedreich's ataxia the tender age at which the disease develops, the family history, the presence of nystagmus, and the peculiar defect in articulation as well, or later the presence of scoliosis and club-foot, render the separation of the affections easy. The disease is differentiated from cerebellar tumor by the fact that despite certain common symptoms, such as inco-ordination with exaggeration of the knee jerk, and perhaps paraplegia, there is present in the latter affection severe occipital headache, with vomiting and optic neuritis or post-neuritic atrophy.

PROGNOSIS.—The course of the disease is steadily although slowly progressive, as is the case with primary spastic paraplegia.

TREATMENT.—The treatment is that outlined under both Tabes and Primary Spastic Paraplegia. Suspension is of much more utility here than in the latter affection, especially in the cases which have followed a chronic myelitis. I have obtained strikingly gratifying results with it in diminishing ataxia and rigidity in a number of cases of this disease.

As syphilitic disease of the spinal cord often causes symptoms similar to those of ataxic paraplegia, more especially to those atypical forms of tabes classed with ataxic paraplegia, in a case in which there is the slightest suspicion of past syphilis, if it cannot be ascertained that the patient received a thorough mercurial course, the plan outlined in the management of syphilitic pseudo-tabes (p. 199) should be pursued.

Brief mention must be made of a form of primary combined sclerosis first described by J. J. Putnam and subsequently by C. L. Dana. It is a new type of spinal-cord disease, which previously had been confused with myelitis or with multiple neuritis. It affects chiefly the posterior columns and the pyramidal and cerebellar tracts. It occurs chiefly in enfeebled women, usually past middle life, and runs a rather progressively rapid course.

Associated with the sclerotic process in the parts named there occur diffuse or systemic areas of softening, with degeneration of the gray matter or the nerve roots.

Putnam's researches suggest that both chronic lead- and arsenical poisoning may be the chief factors in the production of this form of sclerosis. The disease begins with anæsthesia or paræsthesia in the extremities and the occurrence of progressive enfeeblement, leading ultimately to paraplegia. There are emaciation, anæmia, and perhaps obstinate diarrhœa. Spastic symptoms occur, with exaggeration of the knee jerk and presence of ankle clonus. The arms are involved, although less prominently than the legs. The average course of the disease is two years. Death occurs from exhaustion. Perhaps because the disease has not in the past been recognized sufficiently early, treatment has been futile to stay its progress. Temporary improvement in a few cases has been noted under tonics.

HEREDITARY ATAXIC PARAPLEGIA; SPINAL PROGRESSIVE MUSCULAR ATROPHY.

By CHRISTIAN A. HERTER, M. D.

HEREDITARY ATAXIC PARAPLEGIA.

Synonyms.—Friedreich's disease; Friedreich's ataxia; Hereditary ataxia; Family ataxia; Generic ataxia; Friedreich's form of locomotor ataxia.

Definition.—A form of ataxic paraplegia due to combined posterior and lateral sclerosis, which occurs in families, always begins in young individuals, and further differs from the common form of ataxic paraplegia by the presence of nystagmus and disordered speech.

Etiology.—The occurrence of two or more cases of the disease in the same family is a prominent characteristic. As many as ten cases have been known to occur in the same family in the course of three generations. The two sexes suffer about equally, there being probably a slight preponderance in favor of boys. In the same family the two sexes may suffer equally, but often the disease affects one sex to the exclusion of the other. Apparently isolated cases occur, but it is likely that at least some of these will become the progenitor of persons who will develop the disease. The first symptoms may appear in infancy or not until the twenty-fourth year, but in the majority of instances the commencement is from the seventh to the sixteenth year. When several members of a family are affected they usually develop the symptoms at about the same age. Acute infectious diseases may facilitate the onset in some cases.

Pathology.—The pathology of hereditary ataxic paraplegia is involved in obscurity and confusion. Recent investigations by means of modern methods have led to such widely varying views as to the nature of the lesions observed that they seem to have added to the confusion. Only a few autopsies have been made, and some of these were in cases that did not conform closely to the type described by Friedreich. It seems to be pretty well agreed, however, that the spinal cord in hereditary ataxic paraplegia is usually reduced to two thirds or three fourths its normal diameter, but whether this alteration is due to arrest of development or to atrophy of the nerve fibres or to the contraction of newly formed connective tissue is not clear. In every case the cord shows sclerotic changes involving various fibre systems, especially the posterior columns (the change being most marked and most extensive in the columns of Goll), the lateral pyramidal tract, and sometimes the direct pyramidal tract and the direct cerebellar tract.

The marginal tract of Lissaur is sometimes involved, sometimes not. The cells of Clarke's columns are often atrophied, and the cells of the anterior and posterior horns may be much reduced in number.

According to Weigert's recent studies, the proliferation of neuroglia cells is less marked in Friedreich's ataxia than in tabes dorsalis, but we are still in the dark as to why or how the sclerotic changes are developed.

Symptoms.—From the foregoing statements it is evident that the lesions of Friedreich's disease resemble in some respects the lesions of ataxic paraplegia, in others the lesions of locomotor ataxia. In some cases the lesions incline more to the former type, in others to the latter, and in some cases important lesions of the gray matter are added. As might be expected, these variations entail differences in the clinical manifestations of the disease.

The first and most important symptom is inco-ordination of gradual onset, first in the legs, later in the arms. The ataxia is shown first as unsteadiness in standing and walking, later in a drunken, reeling gait. When the patient stands with eyes closed and feet together there is usually considerable swaying, as in locomotor ataxia. The muscular sense is said not to be impaired (Sachs). After a time the head may be the seat of irregular ataxic movements. In most cases there is after a time some loss of power in the legs. The paralysis may eventually become considerable in degree, and in some cases is pronounced early in the disease. It doubtless depends on the implication of the anterior horns. Sensory symptoms are generally absent, and the absence of lightning pains is an important point of distinction from tabes. This exemption is said to be due to the fact that the peripheral nerves are not involved (Déjerine). But, according to Charcot, lightning pains have occurred in some cases. The early loss of the knee jerk is one of the most characteristic symptoms. In a small percentage of cases the knee jerk is normal or even exaggerated throughout the course of the disease. The sphincters are rarely implicated. Some time after the development of inco-ordination (usually several years) a disturbance in speech becomes noticeable. The patient hesitates in the enunciation of sentences, and there may be elision of syllables. The defect may remind one somewhat of the scanning or staccato utterance of multiple sclerosis. Another common but late symptom is nystagmus. Very often the nystagmus is apparent only during extreme deviation of the eyes to one or other side, and it is sometimes a question whether these movements, which are at times seen in apparently normal persons, have the same significance as the fully developed symptom. Ocular palsies occasionally occur, but the pupillary reactions are probably never lost. Late in the course of the disease there may be considerable mental failure. Vertigo and dizziness, perhaps due to the oscillatory movements of the head, are common symptoms. Pathological anatomy has not as yet thrown much light on the origin of the different cerebral symptoms. Toward the end of the disease there may be paraplegic weakness, contractures which cause club-foot, and lateral spinal curvature.

The progress of the disease is very slow and gradual, and there may be long periods in which the condition is apparently stationary. Death

generally occurs in from fifteen to thirty years, but the usual cause of death is some intercurrent disease.

DIAGNOSIS.—The diagnosis is seldom difficult. From locomotor ataxia the distinction is made by the early onset, the presence of nystagmus and speech defects, and the absence of pupillary symptoms and lightning pains. Usually the distinction from ataxic paraplegia is easy, but in the tabetic type of this disease, with loss of knee jerks, it may be necessary to base the diagnosis of Friedreich's disease upon the early onset and the occurrence in several members of a family. But even these criteria sometimes fail, and it may be impossible to make an absolute distinction between the two diseases. In multiple sclerosis the knee jerk is exaggerated, some mental failure occurs early, and the speech is typically staccato or scanning; in Friedreich's disease the knee jerk is lost, mental failure occurs late or not at all, and the speech disturbance consists usually in simple elision or hesitation, and not in well-developed scanning speech. In the rare condition known as cerebellar heredo-ataxia, due to atrophy of the cerebellum, the knee jerks are exaggerated, the pupils are Argyll-Robertson, the visual field is restricted, optic-nerve atrophy is common, sensation may be disturbed, and there may be considerable mental defect. These features usually serve to distinguish the disease from Friedreich's ataxia, but the conditions may have much in common (ataxia, speech disturbance, nystagmus, etc.).

PROGNOSIS.—The disease is not influenced by treatment, but the patient may live many years without much discomfort. Death generally occurs from some affection not directly related to the disease.

TREATMENT.—This consists in making the patient as comfortable as possible by attention to the general health. Arsenic may be employed in doses of 5–15 minims of Fowler's solution, t.i.d.

SPINAL PROGRESSIVE MUSCULAR ATROPHY.

SYNONYMS.—Wasting palsy; Amyotrophic lateral sclerosis; Aran-Duchenne type.

DEFINITION.—Chronic spinal muscular atrophy, more widely known as "progressive muscular atrophy," is distinguished by slow wasting and weakening of the muscles, the wasting being at first limited to some one part (most often the intrinsic muscles of one hand), but spreading and increasing gradually until it is extreme and wide in extent. The wasting of the muscles is due apparently to degenerative changes in the lower segment of the motor path; that is, in the anterior cornual cells of the spinal cord, in the corresponding anterior nerve-roots, and in the motor nerve fibres which spring from these cells. As a rule, the pyramidal tracts of the spinal cord are also the seat of degeneration, and this degenerative change may be traceable as high as the cerebral cortex, the upper segment of the motor path being thus implicated with the lower segment. Only the common form of the disease will be considered here (Aran-Duchenne type).

Etiology.—The disease occurs with much greater frequency in males than in females (3 males to 1 female, according to Gowers), and is essentially an affection of adult life, although in rare instances it begins in childhood or during senility. A neuropathic heredity is traceable in almost one half of the cases, but examples of direct inheritance of the disease are exceedingly rare.

The excessive use of particular muscles or muscle groups sometimes leads to wasting, but it is not probable that such over-use ever gives rise to the lesions of progressive muscular atrophy. Anxiety, fright, exposure to cold or to wet, a severe general concussion, a fall or blow injuring a limb, have each been succeeded by the development of chronic spinal muscular atrophy under conditions which make it likely that the onset of the disease was at least facilitated by one or more of these influences. Very rarely the local injury has been followed by atrophy in the injured part, the wasting eventually becoming general. Sometimes the disease succeeds syphilis, but it is not clear that the syphilitic virus has any other influence than that of impairing the vitality of the entire organism. In many cases in which marked symptoms have been noted after some apparently adequate cause it is likely that the disease had begun before the operation of the supposed cause. It must indeed be admitted that in a very great majority of cases no cause can be found to which the disease can be reasonably attributed, and it is likely that the various factors above mentioned act only by impairing the nutrition and vitality of the body, and not as true causes. The writer has been much impressed with the relatively rapid progress of the disease in persons who were receiving inadequate nutriment. The actual cause of the disease is probably to be sought in some congenital nutritional imperfection in the lower (and often the upper) segment of the motor path, which renders it subject to degenerative processes which may be hastened by relatively trivial conditions.

Pathological Anatomy.—The anatomical alterations in the spinal cord are similar to those found in advanced cases of infantile spinal paralysis. The ganglion cells of the anterior horns are in various stages of pigmentary and sclerotic atrophy, and there is more or less proliferation and induration of the neurogliar elements. These changes are to be regarded as degenerative, and not inflammatory, as in the case of infantile spinal paralysis. They are especially marked in the cervical region, but may be found in the dorsal and lumbar regions when the corresponding muscles are implicated. In many cases the bulbar nuclei are affected as in chronic bulbar paralysis. The fibres of the anterior commissure are often degenerated, and the anterior root fibres passing from the diseased cornua are extensively and constantly atrophied. The peripheral nerves contain many degenerated fibres (motor fibres), and the terminal branches to the wasted muscles are completely or very extensively atrophied. These fibres come only from the degenerated anterior nerve roots. The muscles affected are pale and reduced in size.

Histological changes are always present. Of these the chief are—(1) simple narrowing of the fibres without loss of striation, and with increase in the number of sarcolemma nuclei; (2) fatty degeneration with loss of striation; (3) a hyaline or vitreous degeneration apparently

not related to fatty degeneration; and (4) the appearance of longitudinal striation with coincident loss of transverse striation. The interstitial tissue is often increased in amount. It is to be noted that the entire bulk of the affected muscles does not waste at once, but that individual fibres or fibre bundles waste, while their neighbors are little changed in size. This doubtless corresponds to the successive and random involvement of individual anterior cornual cells.

The changes above noted relate to the lower segment of the motor path. In probably every case the upper segment of the motor path is the seat of at least slight degenerative changes. These affect especially the direct and crossed pyramidal tracts. All the fibres of these tracts or only a few of them may be affected. Where the sclerotic process is slight in degree it is usually confined to the motor path in the cord, but where it is intense the degeneration extends through the medulla into the pons and crus, and even into the internal capsule. The cortical motor ganglion cells may be extensively atrophied. As will be seen in referring to the symptoms of spinal progressive muscular atrophy, the variations which occur in the degree and extent of the lesions of the lower segment of the motor path as compared with the degree and extent of the lesions in the upper segment are of the utmost importance in determining the distribution and type (whether flaccid or spastic) of the resulting paralysis.

Symptoms.—The striking clinical feature of spinal progressive muscular atrophy is the slow wasting of the muscles. This muscular atrophy commences in the arms in more than nine tenths of all the cases. In the majority of the cases beginning in the arm the atrophy commences in the muscles of the hand (Aran-Duchenne type), but a moderate number commence in the muscles of the shoulder (scapulo-humeral type). Of the hand muscles, those of the thenar and hypothenar eminences (thumb and little finger) usually suffer very early. This commencement of the atrophy in the small muscles of the hand and interossei is so common as to be a characteristic feature of the disease.

The atrophy generally begins on one side, and does not appear on the other until some time (often a year) has passed. When the atrophy commences in the shoulder the deltoid generally suffers first, but, whether hand or shoulder first shows the wasting, it soon extends to other parts of the limb. Thus both the flexors and extensors of the wrist, the supinators, the biceps and triceps, may in time become, one or all, involved. Often the various muscle groups are unequally atrophied.

Usually, after a time the muscles of the back, especially the trapezii, suffer. A peculiarity of the trapezius atrophy is that its upper part (ultimum moriens) often remains intact when the rest is much wasted. The sterno-mastoid also is often involved. The respiratory muscles may suffer early or late, and thus threaten life. Both intercostals and diaphragm may be affected.

In a very few cases of spinal progressive muscular atrophy the wasting commences not in the upper extremity, but in the legs. These cases do not belong to the Aran-Duchenne type, and there is good reason to think that most of them (including the "peroneal type") are due to changes in the peripheral nerves and not in the spinal cord.

Although it is exceptional to meet with cases in which the atrophy commences in the leg, it is not very unusual for cases commencing in the arm to show slight wasting of the legs after a long lapse of time. The face is rarely involved in the atrophy, but the lips may be wasted, owing to the not infrequent complication of progressive muscular atrophy with bulbar paralysis. The muscles that undergo atrophy gradually fail in power. This loss of power is in a general way proportioned to the degree of the wasting, to which it is usually due. There is an important exception to this general rule. It is that the legs may gradually lose power where there is no wasting or such slight wasting that the loss cannot be due to this. The paralysis that accompanies wasting depends, like the wasting, on disease of the ganglion cells of the anterior horns; the paralysis that occurs without wasting or is greatly in excess of it depends on degeneration of the pyramidal tracts of the cord. In addition to the atrophy and weakness that occur in progressive muscular atrophy there are the following important conditions:

(*a*) *Fibrillary Contraction in the Atrophied Muscles.*—This is so common a symptom in progressive muscular atrophy as to be characteristic, but it is not of invariable occurrence. There is also increased mechanical irritability.

(*b*) *Changed Electrical Reactions.*—When the wasting is slow there is usually simple diminution of irritability to faradism and galvanism. When the wasting is rapid the faradic loss may be out of proportion to the galvanic loss, and there may even be partial or complete R. D. in certain muscles.

(*c*) *Changes in Reflex Action.*—As a rule, there is loss of myotatic irritability in the atrophied muscles. The knee jerk is lost as soon as there is even slight wasting of the anterior thigh group in those cases where the leg is affected. When the legs are paralyzed without wasting (that is, from degeneration of the pyramidal tracts) the knee jerks are exaggerated and there is clonus. There may, indeed, be true rigidity, and the leg symptoms may thus resemble closely those of spastic paraplegia.

(*d*) *Changes in the Tone of the Muscles.*—The atrophied muscles are ordinarily flaccid and without tone; they are in a state of "atonic atrophy." Occasionally the muscles are in a state of rigidity from the first—*i. e.* they are in a state of "tonic atrophy."

Besides the symptoms of progressive muscular atrophy that have been enumerated there may occur others of minor importance. Thus, an early symptom is aching pain in the parts that become atrophied, and later there may be numbness in the parts, but there is never any true anæsthesia. The unequal paralysis of antagonistic muscles may lead to various deformities; in the hand the bird-claw hand (*vide* Ulnar Paralysis, p. 62) is apt to occur. Sexual power is frequently lost. The sphincters are unaffected.

Some of the variations in the symptomatology of progressive muscular atrophy have been briefly stated (variations in reflex state, muscle tonus, and degree of wasting). It remains to be indicated how these variations form the basis of certain clinical types.

We may regard as typical forms of progressive muscular atrophy

those cases in which there is considerable atrophy of the atonic sort in the upper extremity and upper part of the trunk, combined with weakness and rigidity, without atrophy or with very slight atrophy, in the legs. The atonic atrophy in the upper part of the body depends on the degenerative changes, already mentioned, in the ganglion cells of the anterior horns. The weakness and spasm in the legs (with increased knee jerk and clonus) depend, on the other hand, upon degeneration of the pyramidal tract and on those fibres of the tract that pass to the leg centres. For, although the pyramidal tract may be degenerated throughout its entire extent, the degeneration of those fibres that go to the arm centres of the cord does not cause over-action in the arm muscles. This is because the ganglion cells of the anterior cornua of the cervical cord are so extensively degenerated that no degree of degeneration of the pyramidal tract can cause the arm muscles they innervate to over-act. Were these ganglion cells less degenerated, or were some of them intact and others degenerated, the case would be different. The degenerated pyramidal tract would in this case cause over-action in the cervical ganglion cells, with resulting rigidity of the upper-arm and upper-trunk muscles. This is precisely what happens in some cases—namely, the cases of progressive muscular atrophy in which there is tonic atrophy (often slight) in the arms, and simply weakness and spasm, or spasm and weakness with moderate atrophy, in the legs.

It is instructive to picture still another pathological variation in the same direction. Cases occur in which the degeneration of the pyramidal tracts forms the chief lesion, and in which the degeneration of the cervical ganglion cells is very slight indeed. These cases are closely allied to a condition that has been already described—viz. spastic paraplegia; in fact, they differ from them clinically merely in the slight atrophy of certain muscles of the upper extremity.

Finally, another variation, opposed in character to the last, must be noted. Cases occur in which the ganglion cells of the cervical and lumbar cord are all so extensively degenerated that, notwithstanding the presence of degeneration of the pyramidal tracts, there is no rigidity or spasm in the lower extremities. There is in such cases what may be called universal atonic atrophy: the arms and legs are atrophied (often extensively), flaccid, and have lost their myotatic irritability.

Enough has been said to show that in progressive muscular atrophy there are extensive variations in type based on corresponding variations in pathological anatomy. There is, indeed, every conceivable gradation in the combination of spasm, atrophy, weakness, and myotatic increase between the widespread tonic atrophy, verging on pure spastic paraplegia, on the one hand, and on universal atonic atrophy on the other.

The forms of progressive muscular atrophy in which spasm is marked have been designated amyotrophic lateral sclerosis, and have been considered pathologically distinct from the atonic forms. This view is untenable, since lateral sclerosis is present, as we have seen, even in cases that present no spasm or myotatic excess, the effect of the lateral sclerosis in producing over-action in the anterior cornual cells being rendered inoperative by the degeneration of those cells. It is, therefore, more in accord with the facts of pathology to regard all

cases coming under the designation amyotrophic lateral sclerosis as varieties of one varying pathological state than to attempt their establishment as a distinct condition.

Of the state of general nutrition in spinal progressive muscular atrophy little is known. The excretion of urea is somewhat increased, as might be expected, in proportion to the degree and rapidity of the muscular wasting; but if the appetite is much impaired, the urea excreted may be below the normal for a healthy person with the same bulk of muscle. The lime salts of the urine are said to be increased (Fromman).

Complications.—The chief complication is bulbar paralysis, which may develop at any time, and may even precede the spinal symptoms. The tongue may be little implicated in connection with swallowing and articulation. Locomotor ataxia or tabes is a very rare complication. The presence of spasm associated with the atrophy cannot be regarded as a complication, for, as already explained, it depends on a lesion which is to be regarded as an essential feature of the disease.

Diagnosis.—The diagnosis of the developed disease is usually easy. When the atrophy is as yet localized to an intrinsic muscle of the hand, as the first dorsal interosseus or the adductors of the thumb, it is possible to confound it with the local non-progressive atrophy which is occasionally seen in persons who have greatly over-used these muscles. The latter is a very rare affection, and should not be assumed as an explanation of the local atrophy, except in cases where no progress can be detected during many months. Most forms of multiple neuritis are distinguished by the presence of sensory symptoms, but in some cases of lead-poisoning with normal sensibility and no history of lead a diagnosis may be impossible, except after the lapse of a considerable period of time, when continuous progress speaks for spinal progressive muscular atrophy. From syringomyelia the distinction is made by the absence of the well-known dissociated sensibility. From tumors of the spinal cord and from pachymeningitis of the cervical region the absence of pain and other sensory disturbances is the chief point of distinction.

It is important, but not always easy, to distinguish between spinal progressive muscular atrophy and certain forms of progressive muscular dystrophy. The occurrence of muscular atrophy in two or more members of a family, especially if the subjects be young, creates a strong presumption that the disease is myopathic (*i. e.* of muscular origin), and not myelopathic (*i. e.* of spinal origin). If the atrophy begins in the shoulder girdle and spares the hands, or involves the face and upper-arm muscles, or begins in the peroneal group, the disease is very probably not of myelopathic origin.

Prognosis.—In every case of spinal progressive muscular atrophy the outlook is grave both as regards life and the ability to employ the wasting muscles. The disease, however, runs a different course in different persons, without regard to treatment. In a few cases it has proved fatal in less than a year from the onset; in a considerable number it has lasted ten and even fifteen years. In the majority of cases it lasts from three to five years. If the disease begins rapidly, it is very likely that its progress will continue rapid. If it begins slowly,

it is apt to run a slow course throughout. To both these statements there are conspicuous exceptions, and it must not be forgotten that the uniform cause of the disease may be broken by a rapid, almost sudden, increase of paralysis in certain muscles. Spontaneous arrest sometimes takes place early in the disease, but as a rule such arrest occurs only when the patient is bedridden—that is, at a time when the respite is of no use. It is thought by Gowers that progress is slower in cases where the wasting is strictly symmetrical and nearly simultaneous on the two sides than where it is irregular. In most cases the hands do not become useless in less than a year, and the arms can generally be used for two or three years. There is no doubt that impaired digestion and privation as regards food may materially hasten the course of the disease.

The chief danger to life comes from interference with the muscles of respiration and from implication of the bulbar nuclei. Marked bulbar symptoms therefore make the prognosis materially worse, but slight defect in articulation may exist for years without being followed by the bulbar symptoms.

TREATMENT.—Most observers agree that little can be done to stay the course of the disease by means of drugs. Gowers, however, believes that it is often possible to cause arrest of the process by means of injections of nitrate of strychnine into the muscles. This method has not been employed in enough cases to make it possible to pass judgment on its merits, but it should be tried in every case. The initial dose is $\frac{1}{100}$ gr., and this is increased rapidly to $\frac{1}{40}$ gr. once daily. The injection is made into any one of the affected muscles. While this treatment is being carried out it is well to give small doses of arsenic (Fowler's solution, 5–10 ♏, t.i.d.) by mouth. If the strychnine treatment is beneficial, its effects will be noticeable in the course of one or two months.

It is the belief of the writer that electricity is useless for the prevention of the atrophies, but it may be wise to stimulate the muscles to contraction with the faradic or galvanic current (using the current which most readily gives a response), in order to gratify the patient. A strong current may do harm. Massage and passive movements seem to be of some service and should be used in moderation. It is of the utmost importance that the general health be maintained. The patient should have fresh air, sunlight, abundant nitrogenous and fat-containing food, and moderate but regular exercise. Marked improvement in the strength of the wasted muscles occurs when the patient is removed from bad to good hygienic surroundings.

Other Forms of Progressive Muscular Atrophy with Spinal-cord Lesions.

Not many years ago the idea was gaining ground that the chronic muscular atrophies of spinal origin could be sharply separated from the chronic muscular atrophies of myopathic (muscular) origin, but recent observations show that there exists a class of cases which on pathological grounds must be classed with the spinal progressive muscular atrophies, while on clinical grounds they should be classed with the

myopathic atrophies. The recognition of this fact is a distinct step in advance, and yet it must be owned that we are still very far from understanding the significance of the various combinations of lesions and symptoms with which we meet. There are indications that the various parts of the lower segment of the motor path (anterior cornual cells, nerve roots, peripheral motor nerves, muscle plates, and muscle fibres) have not been sufficiently studied in their relations to one another, and that the consequences of the functional unity of the entire segment have not been fully realized. If we bear in mind the fact that a lesion located originally in one or more parts of the lower segment of the motor path may eventually implicate the remaining parts, it becomes less difficult to understand why in atrophies which are clinically myopathic we may find changes in the ganglion cells of the anterior horns.

The Hereditary or Family or Infantile Form of Progressive Muscular Atrophy of Spinal Origin.

Cases have been reported (especially by Werding and by Hoffmann) in which several children in a family have been affected in the latter half of the first year of life by weakness and wasting in the muscles of the legs and back, which in time (months or years) have extended to the muscles of the arms and neck. The muscles implicated earliest and most extensively are the glutei, the flexors of the hips, and the thigh muscles. The atrophy is symmetrically located. Neither hypertrophy nor pseudo-hypertrophy of the muscles has been observed, but the adipose covering the muscles is apt to be increased, and thus obscures the wasting for a time. The paralysis is flaccid in type, and is associated with well-developed R. D., complete loss of knee jerks, and partial loss of skin reflexes. Lordosis of the lumbar spine may occur. Occasionally bulbar symptoms, fibrillary twitchings, or contractions are observed. There is no pain or local tenderness, and no disturbance of sensibility. The sphincters are normal. Mental development is good. Death occurs in the course of a few years from implication of the respiratory muscles. The following lesions have been observed in these cases: (1) degenerative atrophy of the ganglion cells of the anterior horns; (2) marked degeneration of the anterior nerve roots; (3) less pronounced degeneration of the mixed nerves; (4) secondary simple atrophy of the muscle fibres, with increase of sarcolemma nuclei and occasional fibres which are the seat of degenerative atrophy or lipomatosis. It seems safe to regard this type as permanently established.

The Heubner-Strümpell Form of Progressive Muscular Atrophy.

A few cases have been observed in which progressive muscular wasting, similar in distribution to that seen in the Aran-Duchenne type, has occurred in early adult life in persons who gave a family history of the same disease. These cases have been further characterized by the absence of fibrillation and of degenerative reaction in the muscles. The muscles in these cases have failed to show the simple atrophy with preservation of striation which occurs in the Aran-Duchenne form, and

have further resembled the muscular dystrophies by the presence of markedly hypertrophied fibres. The peripheral nerves, nerve roots, and ganglion cells have been found much degenerated. It is claimed that the spinal-cord changes in such cases are secondary to the changes in the muscles, and that this form of muscular atrophy is to be regarded as a condition intermediate between spinal progressive muscular atrophy and the dystrophies. It has not yet been conclusively shown that this is the case. The name above given may be used to designate these cases provisionally.

SYPHILIS OF THE SPINAL CORD; TUMORS OF THE SPINAL CORD; SYRINGO-MYELIA; MALFORMATIONS OF THE SPINAL CORD.

By CHRISTIAN A. HERTER, M. D.

SYPHILIS OF THE SPINAL CORD.

Definition.—Under this title are included all diseases due to the pathological changes in the spinal cord or in the cord and its membranes which are clearly traceable to syphilitic infection. As will be observed in the section on Pathological Anatomy (p. 238), there are practically no lesions of the spinal cord or its membranes which can be certainly assigned upon purely histological grounds to the action of the syphilitic process. The structure of some gummata (which are rare) and certain features of the inflammatory lesions, especially the changes in the bloodvessels and the irregular and wide distribution of the cellular exudate, are in many instances highly suggestive of a specific origin, and in practice may sometimes be taken as evidence of such origin. In many cases the inflammatory lesions differ so little from those observed in the course of other infectious diseases that we have to depend on the clinical history for our knowledge of their nature, and it is only fair to say that the cases are very few where it is safe to assume the syphilitic nature of the affection in the absence of a clear history of infection. An important class of cases, the degenerative lesions which result remotely from syphilis (including locomotor ataxia), do not properly come within the scope of this section, and will be referred to only to ensure completeness.

Etiology.—Almost every form of spinal-cord syphilis is either a variety of meningo-myelitis or is the early consequence of the vascular lesions of meningo-myelitis. Hence the facts that relate to the etiology of syphilitic meningo-myelitis are in the main applicable to the lesions of spinal-cord syphilis taken as a class.

It may be said of syphilis of the spinal cord that it is with very few exceptions[1] the result of acquired syphilis; that the symptoms may begin in from three months to fifteen years after the initial lesion, but that a very large proportion of them begin between six months

[1] Cases of undoubted spinal syphilis developing as the result of hereditary infection are extremely rare, but a few cases are on record. Thus Siemerling reports a case of cerebro-spinal syphilis in a boy aged twelve who inherited the disease from the father. Gummata were found starting from the meninges of the brain and cord, and typical syphilitic changes were found in the vessels. Syphilitic spinal spastic paralysis has also arisen from hereditary infection (Hoffmann). Probably the changes are never confined to the cord, but affect also the brain. Cases have recently been reported by Friedmann and by Sachs which confirm these views.

and two years after infection,[1] that the period of life in which the largest number of cases occurs is from the twentieth to the thirty-fifth year, and that men are far more often affected than women. As regards the severity of the constitutional specific infection, it is likely that in the majority of cases it is of moderate severity only, but it is true that a large proportion of cases of spinal syphilis occur among those in whom the proper treatment of the disease has been neglected and the syphilitic process has been unchecked. In such neglected cases the onset of the symptoms after infection is apt to be early.

It is difficult to estimate the influence of over-fatigue, of violent muscular effort, of over-eating, of privation as regards food, of alcoholism, of excessive venery, of traumatism, and of exposure to cold and wet in determining the development of the lesions of spinal syphilis, but it cannot be doubted that they favor such development when acting on a syphilitic patient; and it is likely that this influence is proportionate to the degree in which these various causes lower the general vitality and nutrition of the organism.

Pathological Anatomy.—The lesions of syphilis of the spinal cord do not differ essentially from the lesions observed in syphilis of other organs, and the resemblance to the lesions of brain syphilis is particularly close. As in the case of the brain, the syphilitic inflammation has its origin in the pia-arachnoid, and usually begins as an infiltration of the meninges (at first about the vessels, later diffuse) with small spheroidal cells, accompanied with marked changes in the bloodvessels, especially the smaller arteries. In some instances the infiltration is circumscribed in places, and not diffuse, and constitutes foci of inflammation known as gummata. But these changes, though usually most pronounced in the meninges, are only rarely confined to them. The vascular changes and the cellular exudate invade the cord along the connective-tissue septa, and thus damage the nerve elements themselves, the extent of this damage varying greatly in different cases and at different levels of the cord in the same case. We may therefore say that the essential lesion of syphilis of the spinal cord is a meningo-myelitis, and it is probably safe to say that all damage to the nerve elements in syphilis of the spinal cord is the result of this process.[2]

The symptoms of syphilis of the spinal cord are exceedingly varied as regards character, intensity, and distribution, and these variations can be understood only by bearing in mind the great variations that occur in the intensity and localization of the meningo-myelitis and the different consequences which it entails upon the nerve elements. As urged by Sachs, it should be remembered especially, first, that the process is inclined to be exceedingly widespread, and is liable to implicate in some degree the pia over the entire length and circumference

[1] According to Gilbert and Lion, in 56 cases occurring before the third year, the maximum number developed about six months after infection. In 70 cases referred to by Boulloche, 46 occurred in the first four years. It would seem that syphilis of the cord is to be regarded as a secondary rather than as a tertiary condition in many cases. Comparing it with cerebral syphilis, spinal syphilis is probably of earlier development. It is also relatively uncommon if we compare cases of exclusively spinal-cord syphilis with cases of exclusively cerebral syphilis.

[2] It is possible that vascular disease may occasionally cause softening of the cord when there is no meningo-myelitis.

of the cord; and, second, that the intensity of the inflammation and the consequent damage to the nerve roots and the cord vary extremely in different parts. Throughout the greater part of the periphery of the cord the damage is apt to be slight in degree, and in many cases the damage is everywhere slight. Wherever gummata form (and they may occur almost anywhere—in the longitudional fissures, in the walls of the vessels, or in the nerve roots) the damage to the nerve elements is apt to be considerable.

In consequence of the changes just mentioned in the walls of the arteries it is common for thrombosis to occur in the vessels that feed the cord, the thrombosis being facilitated by the low blood pressure and slow current which prevail, partly in consequence of the free arterial anastomosis on the surface of the cord. The vessels of the cord being end arteries (Kadyi), their occlusion leads to softening of limited areas of the cord with surrounding inflammation. A comparatively rare condition of the cord, which is probably secondary to vascular disease in some cases, is the formation of patches of sclerosis.

The lesions of syphilis in the spinal cord possess no histological characters which serve to distinguish them from certain inflammatory lesions which are not the result of syphilis. This view is not in accord with those of some writers, though it is supported by most of the recent pathological observations upon this subject. But the nervous lesions of syphilis, though not distinctive, often show characteristics which enable one to say that they are probably syphilitic. When the syphilitic inflammation is diffuse the small spheroidal infiltration often shows an irregularity of disposition not usually seen in simple inflammation, but often observed in tubercular inflammation. When the infiltration is circumscribed, as in gummata, there is commonly a disposition for the mass to undergo degeneration centrally, but in this respect also there is a close resemblance to tubercular nodules. Again, the syphilitic process in the smaller arteries is apt to cause an unsymmetrical chronic endarteritis (endarteritis obliterans) or a swelling and hyaline degeneration of the arterial and capillary walls—conditions certainly very suggestive of syphilis, but similar alterations are known to occur in tubercular and other inflammations. The lesions of syphilis and of tuberculosis in the spinal cord may, in fact, be so similar as not to be distinguishable in certain cases. In general it may be said that the tendency of syphilitic inflammation is toward cicatrization, that of tubercular lesions toward degeneration and necrosis, but to this there are many exceptions. The discovery of the tubercle bacillus in the case of tubercular lesions (especially nodules) often settles the nature of a doubtful lesion; but this is not a histological criterion.

The changes in the walls of the arteries which constitute endarteritis are probably dependent on the action of the toxin produced by the specific micro-organism of syphilis (see Endarteritis of the Vessels of the Spinal Cord, p. 119).

Gumma of the Spinal Cord (Circumscribed Syphilitic Infiltration).

It is important to define the sense in which the word "gumma" is employed, for if we class as such all spheroidal-shaped areas of infil-

tration of the pia or cord which are visible to the naked eye, we should have to regard gummata as common, and even constant, features of diffuse meningo-myelitis. But if the term is used only to include nodules consisting of caseating or cicatrizing centres surrounded by a zone of granulation tissue which is enveloped in an irregular and usually dense area of small spheroidal cell-infiltration, the number of gummata becomes relatively small, and the condition is to be regarded as a rare affection of the spinal cord. This usage is to be preferred.

Gummata of the cord are usually single, occasionally multiple. They originate from the pia or arachnoid, and from it invade the adjacent cord. Generally they have been special incidents of a more general and diffuse meningo-myelitis, but instances have been recorded in which the sharply defined cicatrizing gumma has been the sole evidence of syphilis of the spinal cord (Charcot and Gombault).

Practically, we have to recognize the following clinical forms of syphilitic disease of the spinal cord:

Primary syphilitic lesions of the spinal cord.	*A.* Subacute and chronic diffuse meningo-myelitis. Variety: Erb's syphilitic spinal paralysis; meningo-myelitis simulating scleroses of the spinal cord. *B.* Circumscribed meningo-myelitis—gumma.
Lesions secondary to syphilitic vascular lesions.	*C.* Acute or subacute softening of the spinal cord—syphilitic myelitis. *D.* Scleroses of the spinal cord: 1. Disseminated syphilitic sclerosis; 2. Tabes dorsalis.

Diffuse Syphilitic Meningo-myelitis.

Synonyms.—Chronic syphilitic meningitis; Arachnitis gummosa.

Symptoms of the Various Forms of Spinal Syphilis.—In a typical case of syphilitic meningo-myelitis of gradual development and chronic remittent course the earliest symptoms are those referable to irritation of the meninges and nerve roots. In addition to pain referred to the dorsal, cervical, or lumbar spine, there are radiating pains in various nerve territories, especially girdle pains and neuralgiform pains in the limbs. The severity of these pains is extremely variable in different cases and even in the same case. They are rarely entirely absent. The usually wide distribution of these sensory symptoms is such as to suggest the diffuse or disseminated character of the lesions. It often happens that there are no symptoms referable to compression or irritation of the anterior nerve roots, for if the lesions are limited to the dorsal region, the effects of damage to these roots is so slight as to be overlooked. When, however, the anterior nerve roots are implicated in the cervical or lumbar region, there are muscular weakness, atrophy, and altered electrical reactions. But the paralysis and atrophy are usually partial in degree and limited and irregular in distribution, owing to the characteristic irregularity and incompleteness of the meningeal lesions. It is not uncommon to observe a coarse tremor, increased by effort, in the paretic limb, and this may be an irritative nerve-root

symptom. When the cord itself is invaded the evidences of damage to the nervous system become more pronounced, but even then the symptoms are essentially those of the partial interruption of the peripherally located fibre systems. These symptoms consist especially in partial or complete paralysis of one leg or of unequal paralysis of both legs, this inequality corresponding to the asymmetrical implication of the pyramidal tracts. As a rule, the paralysis is spastic in type, but if the mid-lumbar cord is damaged, the paralysis is flaccid and not spastic. In a certain proportion of cases the spastic paralysis presents the peculiarity (first noted by Erb) that the muscles paralyzed are only moderately rigid, even in the presence of ankle clonus and exaggerated reflexes. These motor symptoms are almost invariably accompanied with sensory disturbances, commonly of a mild type. Paræsthesiæ in the affected extremities are common, but actual loss of sensibility is exceptional. The various forms of sensibility may be reduced together, or the pain, temperature, or tactile sense may be separately impaired. Some degree of inco-ordination is not uncommon in the paretic limb or limbs. Incontinence of urine and of fæces is usual where the lesion implicates the lumbar cord, and there is often partial incontinence of urine when the lesion is located above the lumbar region. A combination of meningeal, root, and cord symptoms like that just sketched is suggestive of a syphilitic lesion in consequence of the multiplicity of the symptoms, their wide extent, and the incompleteness of their development. A further and most important characteristic of many of these cases is the marked variation of the symptoms within a short space of time. Thus a patient with paralysis of one leg develops paraplegia within a few days, the newly developed weakness often clearing up as rapidly as it developed, or the knee jerks which have been present one day are gone the next, but remain absent only a short time. Rapid fluctuations are also observed in the condition of the sensory symptoms.

While it would be both impracticable and useless to describe the almost endless combinations of symptoms that may be observed in syphilitic meningo-myelitis, it is desirable to take note of some of the more characteristic types, certain of which assume considerable importance owing to their resemblance to symptom groups which possess an entirely different diagnostic significance.

Chronic Dorsal Syphilitic Meningo-myelitis; Erb's Syphilitic Spinal Paralysis.

Symptoms.—The symptoms which constitute Erb's forms of syphilitic spinal paralysis are such as might be expected from a meningo-myelitis of the dorsal region of the cord with slight implication of the meninges, and a pronounced invasion of the structures of the cord itself, especially the periphery of the lateral columns. Although a lesion thus located best explains the symptoms of Erb's type, the pathological descriptions of this condition rest as yet mainly on inference, and if we regard it as a special form of spinal syphilis, we must do so almost wholly upon clinical grounds. At present, therefore, we may reasonably assume that Erb's type is merely a meningo-myelitis presenting certain peculiarities of distribution. The leading features of

this type are as follows: the gradual development of a spastic paraplegia with exaggerated knee jerks and ankle clonus, the almost regular presence of slight incontinence of urine and the absence or slight development of sensory disturbances in the legs (numbness, formication, slight anæsthesia). A feature of these cases is the disproportion between the considerable spasm noted in the walk, the increased reflexes, etc., and the slight degree of rigidity observed in the muscles when the legs are at rest. The onset of the symptoms is generally within three years of the time of infection. The progress of the condition is slow, there is a marked tendency to remissions in the symptoms, and in many cases all progress in the disease ceases.

Cases showing these clinical characters are by no means rare, especially in hospitals for chronic diseases, but, on the other hand, they are far from common. In the experience of the writer they are considerably less frequent than cases of locomotor ataxia. It should be said that while Erb's form of syphilitic spinal paralysis stands for a well-recognized group of cases, we cannot sharply separate it from allied conditions even upon clinical grounds. For example: it is probable that some cases of widespread meningo-myelitis with flaccid paralysis and loss of knee jerks develop under the influence of treatment into a condition which cannot be distinguished from that to which Erb first called attention, and which he claims, without full justification, to be a quite distinct type of spinal syphilis.

Gumma of the Spinal Cord.

Symptoms.—The symptoms of gumma of the spinal cord are those of tumor and are elsewhere discussed (see Tumors of the Spinal Cord, p. 254). This refers to the use of the term in the limited sense indicated in the section on Pathology. Using the term in the more general sense, and excluding gumma as already defined, it is probably safe to say that gummata do not cause sufficient damage to give rise to the symptoms of tumor, and their manifestations are sufficiently considered in the section on the Symptoms of Meningo-myelitis (p. 240).

Syphilitic Pseudo-tabes; Syphilitic Pseudo-ataxic Paraplegia.

Symptoms.—In a very small number of cases symptoms closely resembling those of tabes have resulted from a syphilitic meningo-myelitis which has implicated especially the posterior aspect of the spinal cord, and has invaded the posterior nerve roots and the posterior columns of the cord. In such cases there have been ataxia, lightning pains, loss of knee jerks, and disturbance of the functions of the bladder. In addition to these symptoms there have usually been present cerebral symptoms similar to those observed in tabes, especially third-nerve paralysis, pupillary paralysis, etc., but not identical with them. In true tabes the cerebral symptoms are essentially those that depend on a chronic degenerative process in the nuclei of the affected cranial nerves; in pseudo-tabes the symptoms are those due to an active process affecting the pia and the larger and smaller vessels. These symptoms will be elsewhere described. It is upon the character of these associated cerebral symptoms, the fluctuation in the spinal-cord

symptoms, and the relatively early onset after infection that the distinction of pseudo-tabes is to be made.

It is claimed by some authors (Dinkler, Kuhs, Sachs) that a specific meningo-myelitis is sometimes associated with the degenerative lesions of true tabes, the symptoms of which are overshadowed and obscured by those of the tabetic lesions. This is probably the case, although it is not absolutely clear in the few autopsies hitherto reported that the lesions were actually syphilitic in origin.

Occasionally we see in patients who have contracted syphilis within a few years a combination of symptoms strongly suggestive of ataxic paraplegia—namely, progressive weakness of the legs, with proportionate ataxia, rigidity of the muscles, and increased knee jerks with ankle clonus. It is likely that these symptoms are due to the simultaneous invasion of the posterior and lateral columns of the cord by a meningo-myelitis of syphilitic origin, but at present there is no proof of this. Such cases cannot always be positively distinguished from ataxic paraplegia, but the early onset after infection, the irregular progress with occasional regression of the symptoms, the presence of even slight disturbance of sensibility, the involvement of the bladder, or the presence of symptoms indicating cerebral syphilis are important aids in making a distinction from ataxic paraplegia.

A few cases have been described (Schmaus, Goldflam) in which the symptoms of a chronic poliomyelitis of the lumbar region of the spinal cord have apparently been due to syphilis. The changes in the anterior horns have usually been degenerative in nature, and in some instances have been associated with hyaline degeneration, more extensive in distribution, of the vessels of the cord. These changes may or may not be accompanied with chronic leptomeningitis. The cases referred to are so rare and their dependence on syphilis so uncertain that any further mention of them would be out of place.

Acute Syphilitic Myelitis; Acute Myelomalacia.

The question whether syphilis is ever the cause of acute myelitis, though much discussed, has never been decided in a definitive way. It is the view of the writer that cases occur, though not often, in which acute transverse myelitis or disseminated myelitis may with good reason be looked upon as syphilitic in origin. Yet there is nothing in the nature of the lesion observed in these cases in the spinal cord to distinguish them from the myelitis that occasionally follows other infectious diseases. A considerable number of cases have been reported[1] in which a focus of myelitis or softening (or many foci, distinct or confluent) has followed upon a clear history of syphilis under such circumstances as to make it all but certain that the cord lesions were syphilitic. In a certain number of these cases there has been associated meningitis, sometimes general, sometimes limited to the myelitic level, and in some cases the pia has been very slightly or not at all implicated. In a few cases (Siemerling, Osler) the myelitis has been complicated with a true gumma. In a number of cases of acute myelitis well-marked disease of the small

[1] Gilbert and Lion, B. Anduin, Lamy, Sottas, Goldflam, Winge, Homolle, Julliard, Leyden, Walker, Schmaus, Déjerine, Siemerling, Schulze.

arteries entering the cord (which according to Kadyi are terminal arteries) has been found. Usually the chief change has been a marked and often uneven thickening of the arteries, but, as a rule, there has been also periarteritis, endophlebitis, and periphlebitis. In a few cases the affected arteries have been the seat of hyaline degeneration, and have been obstructed by hyaline thrombi derived probably from white blood cells. Where the arteries have been the seat of inflammation the pia has likewise been implicated, but cases sometimes occur in which the pia is normal or nearly so, while vessels entering the cord are changed. There can be no reasonable doubt that in a large proportion of cases of acute syphilitic myelitis the process is in reality one of acute softening of the cord, owing to the obstruction of a number of the nutrient vessels of the cord, and that the inflammation is that which occurs about foci of softening. But it cannot yet be claimed that all instances of syphilitic myelitis are the result of vascular obstruction. Cases occur in which the vessels show no alteration except those found in all inflamed tissues. As suggested by Gowers, it is possible that thrombosis may occur in a minute vessel, and that the primary lesion may ultimately disappear in the intense inflammation which it excites; but this is nothing more than a suggestion.

The symptoms of acute syphilitic myelitis or myelomalacia do not usually develop suddenly, but rather with prodromata, such as numbness and slight weakness in the lower extremities or retention of urine, which vary much in duration and intensity before the condition is fully established. At times, however (as in the cases reported by Williamson), the onset is very rapid or even sudden. When the condition is established the symptoms are partial or complete paralysis of both legs, considerable and even complete anæsthesia of both legs (the anæsthesia is much more extensive and pronounced than in chronic meningo-myelitis), paralysis of the sphincters, and diminution or abolition of the knee jerks. Bedsores are apt to appear early. After a time the knee jerks are usually increased, sometimes even in cases where they are at first abolished, and ankle clonus is obtainable. Cases of syphilitic myelitis usually terminate fatally within a few weeks or months. They are little more influenced by antisyphilitic treatment than cases of transverse myelitis of non-specific origin. In a few cases, under especially favorable conditions, life has been prolonged several years, but there has usually been little or no retrogression in the symptoms. Occasionally the symptoms have begun in the arms.

The usual secondary degenerations occur in these cases if they live more than a few weeks, and in the cases of longer standing disseminated sclerotic patches have been found at the seat of the original lesion.

Syphilitic Inflammation of the Cauda Equina.

In a very small number of cases the cauda equina has been implicated in a syphilitic inflammation. The nerves of the cauda have been infiltrated and matted to one another and with the membranes by the exudate. The symptoms in such cases are pain, referred to the sacrum and in the regions supplied by the sacral nerves, partial loss of power in the legs, anæsthesia of the buttocks (saddle-shaped areas, etc.),

paralysis of the bladder and rectum, and impotence (see Localization of Spinal-cord Lesions, p. 87). Occasionally the cauda is involved in tubercular inflammation with results indistinguishable from those caused by syphilis. Usually the lumbar vertebræ have been the primary seat of the tubercular process.

Syphilitic Multiple Sclerosis of the Cord.

Patches of sclerosis are sometimes found scattered in an irregular manner through the spinal cord and brain, without reference to definite nerve tracts, in persons who have previously shown the symptoms of disseminated myelitis and encephalitis. In some cases thickened and infiltrated bloodvessels have been found in the centres of these patches, where the perivascular sheaths, distended with small round cells, have given evidence of a still active though subsiding inflammatory process (Déjerine, Köppen, Buss, Popow). As a rule, the patches themselves differ from the sclerotic islands of typical multiple sclerosis in that the axis cylinders are destroyed in company with the remaining parts of the fibres, not by the proliferation of the neurogliar elements, but by the necrotic or inflammatory process which precedes this proliferation. It is thus highly probable that some cases of insular sclerosis have their origin in vascular disease, and it may be that even the cases in which the sclerotic islands do not develop as a sequel to myelitic softening have their origin in chronic changes in the vessels.

The symptoms that result from disseminated sclerosis following syphilitic disease of the vessels of the spinal cord cannot at present be described in a satisfactory manner, owing to the small number of cases on which such a description would rest. In some cases the typical symptoms are probably found—inco-ordination, spasm, nystagmus, and defects of speech—but it is the impression of the writer that the clinical pictures vary a good deal from the classical pictures of multiple sclerosis in cases where the suspicion of syphilitic multiple sclerosis is justified, and that the disease is apt to run a course resembling that of lateral sclerosis or amyotrophic lateral sclerosis if cerebral lesions are absent. There is also reason to think that in syphilitic cases the condition is much more rapidly developed than in cases of the classical type. In many cases, perhaps most, a diagnosis cannot be made with reasonable certainty. But in a patient who develops symptoms suggesting multiple sclerosis within a few years after specific infection, or in whom these symptoms follow evidences of meningo-myelitis or myelomalacia, a disseminated sclerotic process is probable. Even the absence of the knee jerks in a patient who has had previous symptoms of meningo-myelitis does not materially lessen this probability. It should be borne in mind that the multiple sclerosis due to vascular disease may run a rapid course after other kinds of infection than that of syphilis (acute infectious diseases).

Tabes Dorsalis and Syphilis of the Spinal Cord.

As a rule, no traces of active syphilitic disease are to be found in cords of tabetic persons, but it is nevertheless an indisputable fact that

the lesions of tabes dorsalis are a remote sequel to syphilitic infection in a large majority of cases. Recent observations (Leyden, Marie, Déjerine) go to show that the degenerative changes in the posterior columns are secondary to alterations in the structure of the posterior nerve roots, and according to some observers (Obersteiner, Redlich) these changes in the posterior nerve roots begin just at the point where the latter pierce the pia mater to enter the cord, and depend on chronic meningitis and arterial sclerosis in this region. Syphilitic meningitis (and its vascular changes) has been found in some cases (Buzzard, Adamkiewicz, Sachs) of locomotor ataxia, but the relation of such changes to the tabetic process is uncertain.

It may be said that, on the whole, the facts favor the view that the lesions of tabes are usually the consequence of preceding chronic arteritis of syphilitic origin, but the evidence is not yet conclusive on this point.

Multiple Cerebro-spinal Syphilis.

It is a common thing to find some manifestation of cerebro-spinal disease associated with one or other of the forms of syphilitic disease of the spinal cord which have just been described, especially with meningo-myelitis. Indeed, it is the experience of the writer that spinal syphilitic disease of whatever form is more often associated with cerebral symptoms than not. This is the case even in tabes, where the Argyll-Robertson pupil and the optic-nerve atrophy indicate the implication of the brain in the degenerative process. The cerebral lesions which are associated with meningo-myelitis or its consequences—that is, with syphilis of the spinal cord, properly speaking—are essentially of the same nature as those found in the cord. In many cases there is a chronic syphilitic meningitis at the base of the brain, with or without the formation of gummata, involving the cranial nerves (especially the third nerve, with ptosis) in an irregular and often unilateral way. In other cases there is acute cerebral softening from the thrombosis of atheromatous arteries, especially the branches of the middle cerebral. In such cases there is usually hemiplegia. Sometimes the basal meningitis is associated with the vascular lesions just mentioned. It often happens that where a meningo-encephalitis and a meningo-myelitis are associated the former begins first, and it is no uncommon thing for the resulting symptoms (headache, ptosis, paralyses of the ocular muscles, hemiplegia, paraplegia, etc.) to so dominate the clinical picture that the spinal-cord symptoms are lost sight of. Sometimes the chief cerebral indications of syphilitic disease are local headache or myosis or immobility of the pupils to light and during accommodation. Occasionally the symptoms are those of a cortically situated gumma.

It is important in every case of suspected syphilis of the spinal cord to examine carefully for evidences of cerebral syphilis, for some of the conditions just referred to are sufficiently characteristic of syphilitic disease to strengthen greatly in certain doubtful cases the diagnosis of spinal syphilis.

Mixed Forms.—At the risk of some repetition it is desirable to call attention to the frequency with which different lesions of the spinal

cord resulting from syphilis are associated, for this association of different lesions in the same case leads to exceedingly puzzling combinations of symptoms. Thus a slight meningitis in the lumbar region may abolish the knee jerks at the same time that an acute or subacute softening of the spinal cord in the dorsal region secondary to arteritis gives rise to spastic paraplegia and ankle clonus. The association of ordinary meningo-myelitis with acute or subacute softening of the cord is perhaps the most frequent combination of lesions. Sometimes meningo-myelitis is combined with the sclerotic islands already referred to. Then there may result a puzzling combination of the symptoms of spinal syphilis and multiple sclerosis. The combination of meningo-myelitis and degenerative changes in the cord has been already described. It would be impracticable to undertake to describe in detail the almost innumerable combinations of symptoms that may arise according to variations in the position, nature, and degree of the lesions, the present intent being merely to call attention to one of the ways in which atypical cases arise. One of the commonest symptoms in syphilis of the spinal cord is the loss of knee jerk. It is liable to occur in any of the types described except that of Erb, and even in this form, when the meningo-myelitis is clearly dorsal, there may be enough implication of the lumbar pia and nerve roots to abolish it; and it is a question whether the mere loss of knee jerk should lead us to make a sharp separation from Erb's type if there is a correspondence in other symptoms. A spastic gait and loss of knee jerk are sometimes found in association. Probably meningitis implicating the lumbar roots is the chief cause of loss of knee jerk in syphilis of the spinal cord where there is no lesion in the lumbar cord itself, but it should be remembered that it is sometimes lost in transverse myelitis of the dorsal cord, in cerebellar syphilis and during and after syphilitic fever.

Diagnosis.—The features which characterize the various forms of syphilis of the spinal cord have already been sufficiently described. In the case of meningo-myelitis the symptoms are often sufficiently characteristic of a syphilitic lesion to justify making the diagnosis of syphilis of the spinal cord with considerable confidence even when the history of specific infection is wanting. The symptoms of acute softening of the cord are by no means characteristic of syphilis, for they are observed after various acute infectious diseases—as a rule, soon after or during convalescence. The symptoms of acute or subacute softening of the cord, coming on without apparent relation to any infectious disease (including tuberculosis, which sometimes leads to softening of the spinal cord—Williamson), are more likely to be due to syphilis which has been overlooked than to anything else. Care should of course be taken not to mistake the cord affections of Pott's disease without obtrusive signs of bone disease for syphilis of the cord. It is important to remember the following facts, neglect of which has caused many errors in diagnosis: (1) it does not follow that a given spinal-cord lesion is syphilitic merely because a patient has had syphilis; (2) many patients think they have had syphilis because they have had a chancroid, and physicians sometimes fall into the same error; (3) we cannot safely exclude the possibility of syphilitic infection unless we can also exclude the possibility of impure sexual relations on the part of the

patient, and (in the case of women especially) not always then; (4) the fact that a patient is not aware of having had secondary symptoms does not distinctly diminish the significance of a history of a chancre.

Prognosis.—The prognosis of syphilitic disease of the spinal cord depends (first) upon the nature and location of the pathological changes; (second) upon the vitality of the patient, which is essentially the capacity of his cells to repair the damage which they have sustained; and (third) upon the ability of the patient to obtain judicious antisyphilitic treatment and suitable hygienic surroundings. In speaking of the outlook for the patient's future it will be assumed that he is under appropriate treatment, for without such treatment the mildest lesions of the nervous system are almost certain to develop and do irreparable damage in the course of time.

The pathological changes which are most amenable to treatment are the changes in the walls of the vessels (arteritis, phlebitis) and the small round-cell infiltration into the pia and into the cord. These are the early changes in a large majority of cases of syphilis of the spinal cord. Before the substance of the cord is infiltrated, and while the symptoms are nerve-root symptoms only, it is almost certain that the symptoms will clear up under vigorous treatment. If the substance of the cord (lateral columns usually) is infiltrated and there is partial paraplegia, there is a very fair prospect that marked improvement will occur; but it is doubtful if the recovery will be complete, for it is likely that some nerve fibres will have become atrophied, and if the paraplegia has lasted more than a month, secondary degenerations may have begun.

The prognosis is better in cases of Erb's type than in cases of meningo-myelitis which have invaded the lumbar region, simply because with an invasion of equal intensity in the dorsal and lumbar regions the symptoms of the former (chiefly spastic paralysis) are less serious to the life of the patient than the symptoms of the latter (incontinence of urine, cystitis, etc.). Where patches of softening have been produced in the cord the prognosis as regards restoration of function is exceedingly bad, for softening which follows syphilitic endarteritis is no more amenable to specific treatment than the softening which results from arterial disease of other origin. Many cases of acute or subacute myelitic softening die in a few weeks or months uninfluenced by treatment. In a small proportion of cases life is prolonged for years, and the patients remain paraplegic, with slight remissions, until death. The outlook is naturally much worse when the softening is in the lumbar region than when it is dorsally situated. The cases in which sclerotic patches have formed in the cord are probably not amenable to treatment, although something may be accomplished, in some cases, in the way of checking their extension. The degenerative changes in the cord are probably not influenced by specific treatment, but symptoms due to coincident meningeal or vascular disease may be greatly improved.

It may be said that in the majority of cases of syphilis of the cord all that can be expected is more or less improvement. Although life may be prolonged ten or fifteen years in spite of a considerable degree of paraplegia, the morbid process does in the end shorten life. Even in the mildest cases, where the symptoms apparently disappear wholly,

it is doubtful if the bloodvessels of the cord are ever rid completely of the damage done by the syphilitic poison, and it is not uncommon for relapses to occur even when treatment is conscientiously continued. In patients whose digestive and nutritive processes are chronically and markedly deranged or who are cachectic the influence of treatment is likely to be much less favorable than in patients who are in comparatively good health at the time of onset of the spinal-cord lesions.

TREATMENT.—In every case of syphilitic disease of the spinal cord an energetic course of antisyphilitic treatment should be begun without delay. Unless there is some good reason to the contrary, the treatment should be "mixed"—that is, should consist in the use of both mercury and the iodides. In the purely sclerotic and degenerative lesions of the cord little or nothing is to be expected from mixed treatment in modifying these processes, and it is better to use the iodides alone; but this class of cases is not now under consideration.

There seems to be little doubt that the use of inunctions of mercury is more efficacious than any other way of giving this drug. The experience of the writer strongly favors this view. About one drachm daily of the blue ointment (or, preferably, of the oleate of mercury),[1] should be rubbed by means of rubber gloves into the skin of the inner side of the thighs and sides of the chest. This should be continued until about 70 or 80 drachms of the ointment have been used. At the same time that the use of mercury is begun iodide of potassium should be administered. In mild cases, where the loss of a little time will do no harm, the initial dose should be 20 or 30 grains three times daily, in Vichy or some alkaline water, after meals. This dose should be increased gradually (3–5 grains daily) to the point where treatment influences the symptoms distinctly or where the dose cannot be increased without causing marked gastro-enteric derangement. Usually the dose attained is from 60 to 120 grains three times daily. If the symptoms are urgent, the initial dose should be much larger (40–60 gr. t.i.d.), and should be increased rapidly to 2 or even 3 drachms three times daily. It is sometimes necessary to continue these large doses of the iodide many weeks before there is any effect on the symptoms. Then a point may be reached where the symptoms rapidly improve, often at a time when both patient and physician have become discouraged. The full doses of mercury and iodide should be kept up, if possible, until there is no longer any improvement. Then the mercury may be stopped and the iodide gradually reduced. If the patient holds his own, treatment may be discontinued for two or three months, but if the symptoms increase, the mixed treatment should at once be energetically begun anew. Even if the patient appears to be wholly rid of his spinal-cord symptoms, a course of treatment like that outlined above and lasting for two months should be carried into effect at least twice every year during the next ten years, or, preferably, during the remainder of the patient's life. After the first eighteen months or two years the inunctions of mercury may be stopped if the symptoms remain stationary, and the iodide may be continued by itself. If the stomach is at

[1] The oleate contains 10 per cent. of mercury, and may be employed in the same amount as the blue ointment.

all irritable, the sodium iodide may be substituted for the potassium salt in the same dose. It is apparently equally efficacious.

In most cases specific treatment seems to be rendered more efficacious by the coincident use of hot baths, especially sulphur baths. The Hot Springs of Arkansas and the Glenwood Springs of Colorado in this country, and the baths of Aix, Neudorff, and Nauheim in Europe, enjoy the best reputation for such cases. Probably all cases of syphilis of the spinal cord are benefited by the judicious use of such baths, which should only be taken under the supervision of a competent physician.

In every case of syphilis of the spinal cord it is of the utmost importance to see that the general nutrition is well maintained,[1] and the patient should be especially cautioned against exposure to cold, injury, over-exertion, and sexual excitement. The writer believes that all sexual excitement is dangerous in syphilis of the spinal cord, even in cases that have almost wholly recovered. Marriage should be interdicted until the symptoms have been slight and quiescent for at least a year.

In spastic cases in the chronic stage moderate walking should be encouraged. In long-standing cases the actual cautery along the spine may do some good. Massage should always be used, in conjunction with hot baths, to help in overcoming spastic conditions and contractures. Tenotomy may be employed to advantage if permanent and considerable contractures are present.

TUMORS OF THE SPINAL CORD.

DEFINITION.—The present section deals with tumors of the spinal cord in the clinical rather than the pathological sense, for it includes tumors which are not neoplasms (as massive tubercle and syphiloma), and necessarily makes reference to tumors which originate not in the spinal cord, but outside of it and its membranes. It also includes the tumors of the cauda equina.

Both on clinical and pathological grounds it is important to consider separately—

(1) Extradural tumors, which originate from the outer surface of the dura, from the vertebræ, or from structures lying between the vertebræ and the dura.

(2) Intradural meningeal tumors, originating from the inner surface of the dura or from the pia-arachnoid, and growing in immediate connection with these membranes.

(3) Intramedullary tumors or tumors originating within the substance of the spinal cord.

In many respects the first and second classes of tumors of the spinal

[1] The improvement which occurs in some debilitated patients both as regards power in the locally affected parts and as regards the dissipation of a cachectic appearance, is often rapid and striking where a change is made to improved hygienic conditions and more abundant and better food.

cord are much alike in symptomatology and prognosis, but the number of carefully observed cases is not yet sufficiently great to enable us to generally distinguish these classes in practice. It is desirable, however, that an attempt should be made to describe the differences which actually exist between these classes of cases, even though the undertaking be only partially successful.

Etiology.—The data for a statistical study of the period of onset, the sexual incidence, the influence of trauma, etc. in the case of the different tumors of the spinal cord are far too meagre at present to warrant definitive conclusions. Of extradural tumors, lipomata occur in early life, carcinomata late in life. Of intradural meningeal tumors, lipomata are congenital, myxomata belong to middle life, and syphilomata to early adult life especially. Of intramedullary tumors, the gliomata are apt to occur before maturity, and the massive tubercles, though occurring occasionally in childhood and in advanced life, develop more often between the fifteenth and thirty-fifth years. Taking the tumors of the spinal cord as a class, there appears to be a slight preponderance in the case of the male sex.

If we exclude the infective granulomata (tubercle and syphiloma) and the parasitic tumors, it must be said that we do not know any more about the conditions that favor the growth of neoplasms in the spinal cord than of those located elsewhere. A history of trauma certainly precedes the development of a considerable number of tumors. It is possible that in some cases it hastens the development of a tumor which already exists, and it is practically certain that in some instances where the tumor has been attributed to the influence of an injury to the spine the growth was present before the date of its infliction. Any considerable meningeal hemorrhage of traumatic origin is liable to give rise to cyst-formation, which is sometimes mistaken for a new growth. In a number of cases of tumor the first symptoms have come on soon after exposure to cold and wet. There is of course no reason to think that such exposure ever exerts an influence in the production of the growth, but it is not unlikely that it may excite secondary changes in nervous structures which have been previously damaged by the growth, and thus sometimes determine the first pronounced symptoms. In a considerable number of cases the first evidences of tumor have appeared during or soon after pregnancy, but the meaning of this fact is doubtful.

Tumors of the spinal cord are exceedingly rare. The number of published cases is probably still under 140. The number of reported cases of syringomyelia[1] is already considerably in excess of the number of spinal-cord tumors, although the former condition has only recently been recognized. Brain tumors are probably about forty times as frequent as tumors of the cord.[2]

Pathology.—As already stated, tumors which affect the spinal cord may be extradural or intradural and meningeal or medullary. Of 130 cases of tumors collected by Lloyd, 37 were located outside the dura, 19 lay between the dura and cord, 11 lay outside the cord, but

[1] Schlesinger analyzed 200 cases.

[2] Bruns observed 2 cases of spinal-cord tumors among 3500 cases of nervous and mental disease and 70 cases of cerebral tumor.

their exact position was not known, 19 were located within the substance of the cord, and in 14 the location was doubtful.

The extradural tumors which may damage the spinal cord or its nerve roots are lipomata, which originate from the normal extradural fat, echinococci, sarcomata, adeno-sarcomata, cavernous angiomata, carcinomata and sarcomata springing from the vertebræ (especially their bodies), and vertebral exostoses, which may be multiple. The varieties which originate in the vertebræ are not exceedingly rare; other varieties are very exceptional. Vertebral carcinoma is almost always secondary, usually to carcinoma of the mamma, but sometimes to cancerous disease of the stomach or uterus. It is said to be occasionally primary (Gowers). It is not uncommon for a carcinoma of the vertebræ to infiltrate the extradural fat and to surround the dura for a considerable vertical extent. Rarely the dura is ruptured. Sarcomas and osteosarcomas are especially liable to implicate the sacrum and may compress the cauda equina. The vertebral tumors are very apt to compress the nerve roots in the intervertebral foramina before compressing the spinal cord. The extradural tumors are almost always single, and seldom attain great size unless located in the region of the cauda equina.

The intradural meningeal tumors are chiefly fibromata, syphilomata, myxomata, and sarcomata. More rarely echinococcus or cysticercus cysts have been found, and lipomata, sometimes containing muscle fibres, myxomata, and myxo-fibromata, have been observed. Neuromata and myxo-fibromata sometimes develop on the nerve roots and may compress the cord. The syphilitic tumors of the cord almost invariably have their origin from the pia, but occasionally start from the dura. Like syphilitic tumors of the brain, they are far less common than is generally supposed if we use the word in its restricted and proper sense (see Syphilis of the Spinal Cord, p. 238). The mass is apt to develop at a point where the chief vessels enter the cord. The inflammatory tumor usually both invades and compresses the cord substance. Usually one side is involved before the other, but occasionally the mass is symmetrically located. Such tumors are sometimes rapid in growth, but their progress is apt not to be gradual. The intradural sarcomas usually do not begin in the membranes, but extend to them from other parts (vertebræ, lung). They compress or invade the cord, usually unilaterally at first. The rapidity of their growth depends on the nature of the cell elements of which they are composed, the round-cell sarcomas often growing with great rapidity. Occasionally sarcomata are multiple.[1]

The medullary tumors are few in kind. They are the massive tubercles or tubercular tumors, the gliomata and perhaps certain sarcomata. Occasionally an echinococcus cyst is found. As a class the medullary tumors are distinguished by the fact that they either develop symmetrically from the beginning, or, if they begin unilaterally (as in the case of massive tubercle), they soon extend to the other half of the cord. They also tend to develop with greater rapidity than intradural meningeal tumors, and cause pronounced medullary symptoms at an earlier period.

The massive tubercles of the spinal cord are almost always solitary. They are always secondary to tuberculosis in some other part, generally

[1] Of 28 cases of echinococcus cyst in the spinal canal, 23 were extradural (Souques).

the lung, occasionally the spine. Sometimes the extension of the inflammation can be clearly traced from the outer surface of the dura to the gray substance of the cord, which is the normal starting-point for the growth of the tubercle, probably because it affords a better nutritive medium for the tubercle bacilli deposited from the blood stream. Tubercular growths are rarely larger than half an inch in diameter, are irregularly spheroidal in shape, and are seldom larger vertically than transversely. Like syphilomata, they are rather more often found in the dorsal region than elsewhere. They are much more common than true gummata of the cord.

Of almost equal importance with the solitary tubercles of the cord are the gliomata. These tumors spring from the glia cells about the central canal of the spinal cord and grow toward the periphery (see Syringomyelia, p. 268). They show preference for the gray substance, but after a time extend to the white substance. They often invade and compress the nerve elements without at first destroying them. Their growth is rapid, and they generally invade the cord symmetrically. Probably the cervical region suffers more often than any other. In time the entire diameter of the cord is implicated, and the cord becomes considerably increased in size. Usually gliomata are ovoidal or spheroidal in shape, and involve only a small part of the length of the cord (one or two inches), but occasionally they implicate a considerable part of its vertical extent. When this happens the process is designated gliomatosis of the cord. Most sarcomas that damage the spinal cord spring from parts external to the cord and not from the cord itself, but it is possible that a sarcoma sometimes originates in the mesoblastic tissue which during development has been carried into the cord with the bloodvessels. It is the belief of the writer that most if not all of the tumors classed as gliosarcomata are in reality gliomata, certain cells of which may be indistinguishable from cells of the connective-tissue type. Gliomata may be highly vascular, and are sometimes the seat of hemorrhage.

The cauda equina is not rarely damaged by a tumor growing from the bones or from the membranes. Sarcomas, osteosarcomas, fibrosarcomas, fibromas, and angiomas have been observed. Occasionally the cauda equina has been damaged by extensive tubercular or syphilitic inflammation. Tumors in this locality are often very large, as they have ample space in which to develop. The nerves of the cauda may be very unevenly implicated.

The degree of pressure which a tumor exerts on the cord is of course dependent on its size and consistence, and is largely influenced by its situation. Tumors outside the dura exert far less pressure than growths inside it of the same size and consistence. A soft extradural tumor may grow very large without doing the cord much or any damage, whilst a small hard intradural tumor may seriously compress the cord. When the cord is compressed from without, it is indented or flattened, and may be considerably narrowed and softened. In the dorsal region the cord may be narrowed to the diameter of a quill and still retain considerable functional activity. Softening of the cord is due to inflammation. In tumors within the cord adjacent inflammation and softening are often absent, but there may be limited or extensive softening below

or above the tumor. It is important to remember that the regenerative power of the cord elements is extremely limited. It is doubtful whether there is any effective regeneration, even in the white substance, if it has once undergone degeneration. In the cauda equina extensive regeneration is possible, and renders the outlook much better than in the case of damage to the cord. Secondary degenerations, ascending and descending, are common in all forms of intradural tumors (except perhaps glioma), and in extradural growths which exert much pressure. These degenerations are frequently slighter than one would expect from the degree of apparent damage to the nerve elements.

The central canal is often obliterated by pressure, and may be moderately dilated above the level of the tumor. In gliomatosis or rapid gliosis of the cord a considerable cavity may be formed in the cord (see Syringomyelia, p. 268).

Medullary tumors are apt to set up local adhesive meningitis when they reach the periphery of the cord, and adhesions between the dura and pia are apt to occur in tumors originating from either membrane.

Symptoms.—*Extradural Tumors.*—As the symptoms depend on slow compression of the nerve roots and of the cord, often with evidence of vertebral disease, it is not surprising that they resemble the symptoms of tubercular disease of the vertebræ, which is by far the commonest cause of extradural compression of the cord. When the bones are involved the symptoms are local pain, often increased by coughing or sneezing, rigidity of the spine, irregularity of the spines, and local tenderness, severe and prolonged neuralgic pains (usually bilateral) at the level of the tumor, which are increased by movement, local hyperæsthesia, and after a time spasm of local muscle groups, and perhaps local (one-sided or bilateral) paralysis and wasting. When the cord is compressed in the dorsal region a well-marked paraplegia develops with exaggerated knee jerks and ankle clonus. The severe pain may continue notwithstanding the fact that the parts below the compression of the cord are anæsthetic. This combination of anæsthesia or paraplegia with root pains is known as anæsthesia or paraplegia dolorosa, and is particularly suggestive of malignant vertebral disease. Very rarely the cord is unilaterally compressed, with resulting Brown-Séquard paralysis. This occasionally occurs also in Pott's disease. Occasionally an extradural tumor (unconnected with the vertebræ) runs an almost painless course.

In the cases of extradural tumor in which the vertebræ are not diseased the symptoms are like those of meningeal intradural tumors, except that the cord symptoms usually follow more slowly after the the root symptoms (pain and rigidity), and that when the cord is compressed both halves suffer almost equally from the first. Extradural cervical echinococcus cysts have been known to cause laryngeal crises like those of tabes (Hirt, Gerhardt).

Meningeal Intradural Tumors and Medullary Tumors.—Although there are important differences between the symptoms of meningeal intradural tumors and the symptoms of tumors springing from the cord itself, the resemblances in the symptoms are more striking than the differences, and it is desirable to consider the symptoms of these two groups of tumors jointly before describing them separately.

The first symptoms of intradural as of extradural tumors are almost invariably irritative in character. In most instances pain is the first symptom, and is obtrusive throughout the course of the disease. The pain is usually one-sided at first, and may remain so for many months, but it is sometimes bilateral from the start. It is an important fact (first emphasized by Starr) that the pain seldom or never remains unilateral at a time when marked paraplegia has developed. The location of the pain usually corresponds to the termination of the nerve root which is first subjected to compression. As a large proportion of tumors affect the dorsal region of the cord, it is not surprising that the pain has been often referred to the trunk (chest, epigastrium, abdomen). In addition to pain in the parts at the level of the tumor, there is frequently less severe pain in the parts below (as in the feet); but pain is very seldom referred to parts above the level of the growth. The pain is usually neuralgic in character, and is described as burning, darting, or shooting. In most cases the pain is severe at times, and in some it is excruciating. Many patients are never free from pain, a dull aching pain alternating with acute exacerbations. Tenderness of the nerves in which the pain is felt appears to be an exceedingly rare occurrence—a point of some diagnostic importance. As a rule, spontaneous pain is not felt in the spine itself, but it is not uncommon for pain to be referred to the vertebral column when the spine is flexed or rotated. When spontaneous pain occurs it is commonly located one or two inches below the seat of the tumor (Starr). The spine may be tender to pressure or to percussion when there is no spontaneous vertebral pain, but this symptom is much less common than in extradural tumors. There are some exceptions to the rule that pain is a prominent symptom of intradural tumor, which will be referred to elsewhere. Numbness, tingling, and formication may accompany pain or develop in the intervals between the paroxysms. They are additional evidence of slight damage to sensory nerve elements. Hyperalgesia of the skin, especially at the upper level of the tumor, often develops early when pain is severe. There is often some rigidity of the muscles at the level of the tumor, before the development of the rigidity below the level of the tumor, which is dependent on compression of the cord. This is seen oftenest in the cervical region.

The irritative symptoms mentioned above do not long precede the development of symptoms due to destruction or compression of nerve elements, especially those of the spinal cord. These symptoms are paralysis and increased reflex action in the parts below the level of the lesion, loss of sensibility, and, in some cases, loss of reflex action in parts which were at first the seat of excessive reflex action.

Loss of power, gradual in development, occurs at some period in almost every case of intradural tumor. The paralysis very often, perhaps generally, begins in one arm or in one leg, or in the arm and leg of one side, before becoming paraplegic in distribution. In some cases the paralysis remains unilateral throughout the course of the disease. Frequently the paralysis is paraplegic in type from the beginning, and the two sides may be almost equally affected throughout. This occurs especially in the tumors which spring from the locality of the central canal and in those that lie in the anterior or posterior median fissure.

All four extremities may be paralyzed by a cervical tumor. In such a case either the legs or arms may be first affected or all the limbs may suffer simultaneously. The extension of the paralysis is slow, as a rule, and is directly related to the rapidity with which the growth compresses or destroys the elements of the cord. But it sometimes happens that there is a considerable increase in the paralysis in the course of a few days. This depends not on the tumor directly, but on the adjacent myelitis which it excites. Hence when the myelitis subsides the paralysis may decrease, and this may be mistaken for evidence of retrogression in the tumor. The loss of power due to the advance of the tumor is progressive, and seldom undergoes even temporary decrease. Anæsthesia of the skin is usually associated with loss of power, but is not usually marked until the paralysis is advanced. The loss of sensibility, which relates to tactile, pain, and temperature sensations, corresponds roughly to the motor paralysis, but it is important to note that the sensory loss frequently does not extend so high at first as it does at a later period, when the damaged segments are almost wholly destroyed. Ataxia is a rare symptom. It has been caused by implication of the posterior nerve roots by multiple tumors and by growths implicating the posterior columns of the cord. The condition of the reflexes depends mainly on the locality of the tumor. Early in the course of the process, before paralysis is considerable, the reflexes corresponding to the level of the tumor are lost on one or both sides, according to the tumor's position. When the lesion is above the lumbar enlargement the reflexes are increased in the parts below (skin reflexes and knee jerks exaggerated, ankle clonus present). Tonic spasm develops in the legs as the result of secondary degeneration of the pyramidal tracts. Contractures of the legs, often extreme, may be associated with the tonic spasm. But, though excess of reflex action below the tumor is the rule, there are two exceptions: First, a descending myelitis may extend downward and cause loss of reflexes by invading the lumbar enlargement; and, secondly, it sometimes happens that an active myelitis which stops short of the lumbar region inhibits the reflexes of the parts below by a mechanism but imperfectly understood, and causes an atonic instead of a tonic paralysis. The sphincteric reflexes correspond in a general way with the lumbar reflexes; they are destroyed when the tumor occupies the lumbar enlargement. Muscular atrophy and R. D. are confined to muscles that correspond to the level of the tumor. Vasomotor disturbances, such as œdema, alterations in the circulation and temperature, etc., are often early and marked manifestations at the level of the irritated nerve roots. Bedsores are prevented with difficulty in the late stage of tumor. The temperature of the body is usually normal. In tubercular tumors there is commonly fever due to the tubercular process to which the mass in the cord is secondary. In the late stage, when there is considerable exhaustion, there may be fever from hypostatic congestion of the lungs or from infection from bedsores. There are also cases where fever appears to be caused by the progress of the tumor. The writer has noted moderate continuous fever in cervical sarcoma, and high fever in rapid gliosis of the cervical cord.

In tumors occupying the cervical region of the cord rigidity of the

neck muscles is usually marked, and the arms are the seat of pain, paralysis, wasting, etc.; the legs are the seat of excessive reflex action, crossed anæsthesia with hemiplegia on the side of the tumor, and Brown-Séquard's paralysis may be present[1] (usually in partially developed form if the tumor is one-sided) and is not uncommon; the pupil may be contracted or dilated on the side of the lesion, and the palpebral opening may be narrowed and the eyeball sunken. If the tumor be making rapid progress in the substance of the cord, moderate or considerable fever may be present. Such a tumor may also extend upward and involve the medulla, with resulting dysphagia, loss of diaphragmatic breathing, etc.

Tumors of the dorsal region often produce well-marked signs of unilateral damage to the cord, followed by paraplegia and anæsthesia of the legs. The paralysis often involves the abdominal muscles, and the anæsthesia may extend up the abdomen or chest to a height which depends on the level and transverse extent of the lesion of the cord. Usually there is severe neuralgia on the side of the tumor. Wasting of intercostal muscles corresponding to the damaged segments is not appreciable, but in tumors of the lower dorsal cord it is probable that the abdominal muscles sometimes undergo perceptible wasting and develop the R. D. As with tumors anywhere above the lumbar enlargement, the reflexes below the level of the lesion are greatly exaggerated, but where complete transverse destruction of the cord occurs these reflexes are probably always ultimately lost. Early in the disease the patient is apt to experience an imperative desire to empty the bladder; if he delays, the bladder empties itself. A little later, before the implicated segments are wholly destroyed, there is intermittent incontinence; when the bladder is partly filled the sphincter relaxes spontaneously and the detrusor empties the bladder by gushes. Then the detrusor grows so weak that it cannot empty the bladder, and a more or less continuous dribbling from the full bladder sets in—there is overflow incontinence. In total transverse lesions above the lumbar enlargement the bladder reflexes may be wholly lost. Then the bladder is never full, because after a time the pressure of the urine overcomes what little elasticity remains in the sphincters; neither is it ever empty, because the tension in the bladder soon becomes so low that it cannot force the remaining urine past the sphincter. In this condition the urine can be readily expressed by the hand. Pyelitis and cystitis are grave dangers in this stage of the disease. The condition of the rectum is, generally speaking, similar to that of the bladder. There is obstinate priapism in all tumors above the lumbar enlargement which markedly compress or invade the cord. In tumors above the lumbar enlargement there is little liability to bedsores so long as the sensibility of the legs and of the skin over the sacrum is retained; when this is lost, bedsores occur as in tumors of the lumbar enlargement.

In tumors of the lumbar enlargement the tumor may at first involve

[1] In 35 cases of unilateral compression of the cord the symptoms began on the side of the tumor in 23; in 35 cases of unilateral compression of the cord the symptoms preponderated on the side of the tumor in 8; in 35 cases of unilateral compression of the cord the symptoms were unusually tardy in passing to the opposite side in 17; in 35 cases of unilateral compression of the cord the symptoms were typical Brown-Séquard paralysis in 3.

chiefly either the lumbar or the sacral segments of the cord; the precise distribution of paralysis may thus vary considerably (see Localization of Functions of the Spinal Cord, p. 80), and can be inferred from a knowledge of the functions of these segments. After a time both the lumbar and sacral segments suffer, the damage to the former usually preponderating. The knee jerks are lost early, unless the tumor is confined to the sacral segments of the cord, in which case they may be preserved. The sphincters of the bladder and rectum are wholly paralyzed and relaxed. There is also complete impotence. In women with vascular lumbar tumors exacerbations of the symptoms are apt to occur at menstrual periods (Guinon).

Tumors of the cauda equina, as might be expected from the fact that the nerve roots of the cauda are the neural representatives of the various lumbar and sacral segments, present symptoms closely resembling those of tumors of the lumbar enlargement. There are, however, some important differences. In cauda-equina tumors the symptoms almost always begin in the region involved by the sacral flexors (perineal muscles and those below the knee chiefly involved; sensory loss chiefly over sacrum, about anus and genitals, down back of thigh, and on outer side of leg and foot), the nerves of which lie nearest the median line and show a special susceptibility to suffer in all forms of damage to the cauda equina. As already stated, growths which damage the lumbar enlargement generally cause early and extensive paralysis and atrophy of the legs and thighs, but a tumor compressing the conus medullaris may produce the same symptoms in the region innervated by the sacral nerves as a tumor damaging the median strands of the cauda. On the other hand, a tumor of the cauda may occasionally damage only its lateral or lumbar roots, and thus give rise to symptoms of the type generally seen when the lumbar cord itself suffers. In practice, however, if symptoms begin in the lumbar territory and are only later succeeded by sacral symptoms, or if lumbar symptoms distinctly preponderate over sacral symptoms, this is to be regarded as practically excluding a lesion of the cauda equina. Cauda symptoms are very generally bilateral from the beginning, but frequently they are not symmetrical, though bilateral. In the rare cases where a cauda tumor is originally strictly one-sided a long period elapses before it gives rise to bilateral symptoms. In this respect such a tumor differs markedly from the tumors of the lumbar enlargement. The precise distribution of the anæsthesia is of great help in determining which nerves of the cauda or segments of the cord are involved. There is usually present some variety of the saddle-shaped area of anæsthesia on the buttocks (see Localization of the Functions of the Spinal Cord, p. 98), with or without implication of a strip of skin on the posterior aspect of the thigh and the outer side of the leg and foot. Severe pain in the region of the sacrum is a common symptom of tumors of the cauda. The affected muscles (*i. e.* especially those below the knee) early show marked wasting and R. D. Sphincteric troubles are of the same nature as those seen in tumors of the lumbar enlargement. Bedsores are very liable to occur early and to grow rapidly. An exceedingly important feature of most cases of tumor of the cauda equina is tenderness on percussion of the spine below the second lumbar vertebra. This

sign has great diagnostic significance when taken in connection with symptoms indicating a tumor of the cauda, and practically fixes the diagnosis. In the case of certain tumors (osteosarcoma, sarcoma) springing from the sacrum, the tumor, though not noticeable externally, may be felt by the rectum.

Multiple tumors of the spinal cord are apt to give rise to exceedingly complex symptoms. It may occasionally happen that the existence of two tumors is indicated by the successive development of similar focal symptoms at different levels of the cord. If there are many tumors, the smaller ones may not give rise to recognizable symptoms. If a brain tumor coexists with a tumor of the cord, the symptoms of the latter are usually overshadowed by those of the former, but the writer has known the reverse to happen in the case of tubercular tumors. The presence of multiple growths on the posterior nerve roots has been known to produce the characteristic symptoms of locomotor ataxia.

There are peculiarities in the symptoms and course of certain tumors which require special mention.

In massive tubercle of the spinal cord irritative symptoms are usually rather less pronounced than in other forms of tumor, and the indications of a destructive lesion of the spinal cord preponderate during the whole course of the disease. Usually the symptoms have progressed rapidly between their commencement and the time at which they have attained a marked degree of intensity, and in most instances the duration of the disease has been little more than two months. As a rule, there is a short stage in which the symptoms are wholly or mainly unilateral.

In cases of rapid gliosis or gliomatosis of the spinal cord (see Syringomyelia, p. 268) the symptoms may bear some resemblance to syringomyelia in respect to the sensory symptoms that are present. As a rule, they give rise to the clinical picture of subacute transverse dorsal myelitis or spastic paraplegia with hyperæsthesia or anæsthesia. They do not often cause Brown-Séquard's paralysis, their course is much more rapid than that of syringomyelia (one to three years), and they are subject to considerable fluctuations in their symptoms. In young children they have sometimes given rise to symptoms closely resembling Pott's disease. When located chiefly in the cervical region, as is apt to be the case, these new growths may be associated with a considerable continuous rise in temperature. The vertebræ over the tumor may be tender to pressure and rigid, and there is sometimes slight kyphosis. Dissociated anæsthesia is rare. Toward the end of the disease irritative symptoms may be prominent, but the severe pain so typical of spinal tumor is usually absent.

There are no constant and absolutely characteristic features of extradural tumors as distinguished from those which develop intradurally from the meninges or from the cord. Extradural tumors are apt to cause pain a long time before cord symptoms arise, and when these arise they are apt to be bilateral from the beginning, though they may be more pronounced on one side than on the other. Where the tumor springs from the vertebræ there is apt to be vertebral tenderness and pain on movement of the spine. Where the tumor springs from the

outer surface of the dura these symptoms may be wholly absent. There are no constant and characteristic symptoms of intradural meningeal tumors as distinguished from tumors that spring from the cord, but the following features are apt to distinguish these two classes of growths: The intradural meningeal tumors cause relatively severe root symptoms; cord symptoms begin later, are more limited in extent, and are more liable to remain unilateral, whilst spinal pain, local tenderness, and rigidity are relatively more common. In the case of tumors that spring from the cord itself the radiating pains due to irritation of the nerve roots are commonly absent or slight in degree (with the important exception of growths that irritate the structures of the posterior horns); cord symptoms are often bilateral and even symmetrical from the first, or if unilateral at first they soon become bilateral. Widespread muscular atrophy is also more likely to occur, but not much diagnostic weight can be attached to this fact. The early occurrence of extensive anæsthesia is a symptom of medullary tumors.

Duration.—The duration of the symptoms of tumors of the spinal cord is sometimes not more than six weeks (as in some tubercular tumors), whilst in other cases it may be many years. In a large majority of all cases (including tumors of the bones which damage the cord) the symptoms last from six months to three years, the average period probably being not far from one year. Death occurs earlier in medullary tumors than in meningeal intradural tumors, and the duration of life is longest in extradural growths. When the symptoms have reached their full development the duration of life may be only a few weeks. The cause of death is generally some direct or indirect effect of the tumor (pyelitis, septicæmia, damage to respiratory centres). In the case of massive tubercle death may be due to general tuberculosis or to tubercular meningitis.

Diagnosis.—The diagnosis of the existence of a tumor compressing or invading the spinal cord must generally be regarded as a difficult task. There are cases in which the indications are so clear and unequivocal that the diagnostic difficulties are unimportant, but, as a rule, the problem is one which requires for its solution the most careful balancing of evidence and the repeated examination of the patient. It is the conviction of the writer that a large proportion of the tumors of the spinal cord which come under medical observation are not recognized as such, and that when the diagnosis of a tumor of the spinal cord is made, even by the trained specialist, there is always a considerable probability that the condition will prove to be something else than tumor. But the fact that tumors may occasionally be successfully removed by operation gives their accurate diagnosis a practical importance which they formerly lacked, and makes their recognition worthy of the most painstaking efforts.

The evidence of the presence of a tumor consists in the signs of a slowly-growing focal lesion which both irritates and compresses the cord or the cord and its nerve roots. The presence of severe pain at the level of the supposed growth with slowly progressive motor and sensory paralysis is a highly suggestive group of symptoms. The presence also of rigidity of the muscles of the spine, of muscular contractions in the limbs, and of exaggerated knee jerks and ankle clonus renders highly

probable the diagnosis of tumor damaging the spinal cord. This probability becomes a practical certainty if in addition to the symptoms just mentioned the course of the disease is observed to be steadily progressive, and the symptoms have become bilateral after being for a time one-sided. In many cases, however, this typical combination of symptoms does not exist, and the diagnosis of tumor becomes a matter of probability, which is sometimes high, sometimes low. It only rarely happens that etiological factors aid in the diagnosis. The presence of tuberculosis in another part of the body or of sarcoma, carcinoma, echinococci, or vertebral exostoses may throw light not only on the question of the presence of a tumor, but on its pathological nature. A history of syphilis is apt to mislead, for it is probably present almost as often in other forms of tumor of the cord as in syphiloma.

Tumor of the spinal cord is apt to be confounded with myelitis. The distinction is based on the following facts: severe pain does not occur in myelitis uncomplicated by meningitis; if there be meningomyelitis, the pain is extensive in vertical distribution (and not limited chiefly to one level of the body, as in tumor) and accompanied by hyperæsthesia. It must be admitted that the distinction of tumor from myelitis may be impossible in some cases (*e. g.* some carcinomas of the cord). It is not singular that this should be the case when a tumor causes considerable adjacent myelitis, and there is no accurate record of the symptoms caused by the tumor itself. Cervical hypertrophic pachymeningitis may cause muscular atrophy of the arms and spastic paralysis in the legs. The vertical extent of the focal symptoms is greater in hypertrophic pachymeningitis than in any form of tumor except central gliosis of the cord. In the latter condition pain is never considerable, and may be absent; in the former pain is usually a conspicuous symptom throughout.

In the early stage of tumor the pains are often interpreted as neuralgic. Their persistence should suggest something more than a functional condition. It is probable that in most cases of tumor the characteristic tender points are absent, but too much weight must not be given to this fact. In neuritis the affected nerves are exceedingly tender to pressure in their course. In tumor this tenderness is usually, perhaps always, absent in the nerves to which the pain is referred. It may be necessary to wait a considerable time before the diagnosis of tumor can be established, but some of the evidences of organic disease usually appear early, and at least establish the fact that the condition is not merely functional. It is important to note the fact that the presence of the organic affection may favor the development of any functional derangement to which the patient may be disposed; but the presence of mental symptoms or convulsions or the signs of hysteria should in no way diminish the significance of the signs of structural disease which have been noted. In the case of tumors of the cauda equina pain is often first referred to the sciatic area. The pain of sciatica is, however, almost always one-sided; the pain of tumor soon becomes bilateral.

There are some important exceptions to the rule that tumors of the spinal cord give rise to pain. In central gliosis of the cord the gliomatous tumor soon destroys the paths for the conduction of pain which

lie in the central gray substance; when this has happened pain may cease to be an important symptom. Some tubercles of the cord growing from the anterior horns produce little or no pain for a considerable period. Soft, fibrous tumors (either extra- or intradural) may compress the cord without causing pain, and an extradural cheesy nodule has been known to run a painless course (Bailey). The painless course of a tumor of the spinal cord seems to depend less on the position of the tumor than on its slow growth and soft consistence, for such a tumor has been known to compress the posterior nerve roots and cause anæsthesia without previously giving rise to pain. Notwithstanding these exceptional cases of painless or almost painless tumor, severe and persistent pain is perhaps the most characteristic single symptom of a growth damaging the spinal cord. In the absence of this symptom the diagnosis is rendered exceedingly difficult, and is often impossible in the present state of our knowledge.

In view of the possibility of removing some tumors of the spinal cord by surgical means, it is of the utmost importance to make the diagnosis of the tumor as early as possible, for delay often greatly reduces the chances of successful operative interference. On the other hand, it is a serious error to subject a patient to operation and find that there is no tumor. Diagnostic errors will continue to be made, and it is not possible at present to promulgate rules the observance of which will prevent such errors; but certain dangers should be pointed out. A diagnosis of tumor is always risky unless unilateral irritative root symptoms (pain, spasm) of considerable duration (several weeks at least) are followed by symptoms of damage to the cord on one side, by extension of these irritative root symptoms to the opposite side, and eventually by the signs of damage to both halves of the cord. This is of course equivalent to saying that the diagnosis cannot be made with reasonable assurance until the cord itself has been considerably compressed or damaged on both sides. If severe persistent one-sided pain is followed by the gradual development of paraplegia, this combination of symptoms does not suffice to fix the diagnosis, as was recently shown (Pfeiffer's case). This serves to emphasize the fact that in a large majority of cases of tumors pain is rarely, if ever, limited to one side of the body in the stage when paralysis has developed. The indications by which multiple tumors of the cord may sometimes be recognized have been already mentioned.

If in a given case the presence of a tumor damaging the spinal cord seems probable, it becomes important to localize it as accurately as possible. The indications which help to distinguish from one another tumors which are extradural, intradural, and medullary have been sufficiently referred to in discussing the Symptoms of Tumor.[1] The

[1] The extradural tumors which originate from the vertebræ (carcinoma, sarcoma) have to be distinguished especially from tubercular vertebral disease, and this is always difficult or impossible in the early stage. The onset of the signs of bone disease in early life speaks for Pott's disease, but it is an important fact that vertebral tuberculosis is by no means a rare occurrence in advanced life. Signs of tuberculosis in other parts of the body are a very important aid to diagnosis, but their absence does not in itself much increase the probability of malignant disease. Severe and lasting root pains somewhat favor malignant disease. Paraplegia of rapid onset is more liable to occur in malignant disease than in Pott's. The discovery of a tumor mass external to the vertebral column

diagnosis of the relation of the tumor to the membranes and to the cord is often impossible and never certain, but in many cases a correct opinion may be formed.

The diagnosis of the level at which the cord is being damaged by the tumor is much more practicable. Unless the tumor is in the lumbar or cervical enlargement, where its lower end as well as its upper end can give rise to localizing symptoms, the diagnosis of the lower level of the tumor cannot be made, but its upper level can always be determined with considerable accuracy, and no effort should be spared to do this if an operation is being contemplated. This diagnosis of the upper level of the tumor is based on a knowledge of the functions of the individual segments of the cord and their nerve roots (see Localization of Spinal-cord Disease, p. 72)—*i. e.* on the symptoms that arise from the abolition or exaltation of function at the level of one of these segments. The symptoms which indicate the upper level of the tumor are the upper level of motor paralysis and wasting (especially available when the enlargements are implicated), the upper level of the anæsthesia, and the level at which pain and hyperæsthesia occur. If these symptoms agree in their indications, they fix the upper level of the tumor. If they do not agree, it is important to be guided by the symptom which refers the tumor to the highest level. It is a very important fact that the upper level of the tumor is frequently one, two, or three segments *above* the nerves corresponding to the highest level of anæsthesia or pain. Ignorance of this fact has led to serious errors. The discrepancy is most marked in tumors of the cord itself, and least pronounced in extradural tumors. As it is never possible to predict when the upper level of the tumor will be higher than indicated by the ordinary rules of localization, the surgeon should be prepared to expose the cord at a level higher than that indicated in the event of the lesion not being found. The nature of the tumor is generally very difficult to determine. A history of constitutional syphilis or evidence of tubercular disease in some other part creates a presumption that the cord lesion is of the same nature as the existing diathetic state. This is still more likely if the progress of the tumor is rapid, if the symptoms soon become bilateral, and if the patient is under forty. The presence of a tumor elsewhere is very strong evidence of the nature of the cord tumor, but this indication rarely exists. Multiple tumors are usually sarcomata or gliomata. A tumor in the substance of the cord is almost invariably a tubercle or a glioma.

is good evidence of sarcoma or carcinoma, but is seldom available. It should always be sought for if there are signs of sacral disease. The presence of abscess near the spine is strong evidence of tuberculosis. A history of cancer of the breast or stomach or uterus or rectum is of the highest diagnostic significance. Whenever a doubt exists as to the nature of bone disease with spinal-cord symptoms after the indications have been carefully balanced, the probabilities are always greatly in favor of Pott's disease on account of its relative frequency. An extradural echinococcus cannot be diagnosticated unless there are accessible cysts elsewhere. The presence of vertebral exostoses is sometimes rendered probable by the presence of exostoses elsewhere, as near the orbit or on the ribs. It should be noted that an eroding aneurysm of the aorta (especially of the thoracic aorta) occasionally causes vertebral pain and rigidity, severe intercostal neuralgia, and eventually compression of the cord. The diagnosis can sometimes be made by the presence of the signs of an aortic aneurysm. It is also noteworthy that arthritis deformans of the vertebral joints may cause rigidity and deformity of the spine, with consequent compression of the nerve roots or even of the cord.

In the absence of any indication of the nature of the tumor an extradural growth with evidence of bone disease is almost surely a carcinoma or sarcoma. If there is no sign of bone disease, it is probably a sarcoma, fibro-sarcoma, or lipoma. An intradural meningeal tumor is usually a sarcoma, fibro-sarcoma, or myxo-sarcoma.

Prognosis.—With the exception of syphilitic nodules compressing the cord the prognosis of spinal-cord tumors as regards life is almost hopeless unless the tumor can be removed by operation. Even if the tumor can be removed without killing the patient, the outlook is doubtful as regards the restoration of the functions of the cord, and, while it is right to advise operation in many cases of tumor, the prognosis should always be given in guarded terms.

The prognosis of extradural tumors is, generally speaking, better than that of intradural meningeal tumors. In the case of malignant tumors springing from the bone the outlook is exceedingly bad, although the patient may live for several years in a paraplegic condition if the lesion is in the dorsal region. Echinococcus cysts have been known to rupture externally with spontaneous recovery, but this is a very exceptional outcome. Some recoveries may be expected from operative interference in such cases. Extradural lipomata, fibromata, or fibro-sarcomata are the most favorable cases for operation. The outlook from operation is very good in vertebral exostoses. All tumors of the cauda equina that are not malignant are exceedingly favorable cases for operative treatment. In intradural meningeal tumors—which, like fibromata, lipomata, and some sarcomata, are of slow growth and compress rather than invade the cord—the outlook from operative treatment is fair. In multiple tumors the prognosis is bad even if the growths are benign. The outlook is hopeless in solitary tubercle of the cord and in glioma; that is, in strictly medullary tumors. This is true also of intensive sarcomata and carcinomata. Considerable damage to the cord by the compression of a syphilitic nodule—*i. e.* sufficient to justify the diagnosis of tumor of the cord—may be partially or wholly recovered from under antisyphilitic treatment, but even in such cases the prognosis is doubtful and is usually bad.

In the majority of tumors the damage to the cord is, on the whole, steadily progressive. In some cases the damage is rapidly increased, usually by adjacent myelitis, sometimes by extensive hemorrhage into the substance of the tumor. Occasionally there is a considerable period in which the symptoms are stationary, and in some instances there may even be temporary improvement in the symptoms. The duration of life in tumor of the spinal cord has already been spoken of.

Treatment.—There is little to be said in relation to the medical treatment of tumors of the spinal cord. In all cases where the symptoms even suggest tumor and there is a possibility of syphilitic infection, vigorous antisyphilitic treatment should be begun (see Syphilis of the Spinal Cord, p. 249). If there is reason to suspect that a tumor is tubercular, the use of cod-liver oil and of creasote is distinctly indicated, and, together with a life mainly out of doors, may do something to retard the progress of the lesion. The most important indication for medical treatment, at least from the patient's standpoint, is the pain, which is so often agonizing in character. If the diagnosis is reasonably

certain, there should be no hesitation about the use of morphine or codeine. Other narcotics and sedatives are of very little use, and the fatal nature of the disease disposes of the usual objection to the free use of narcotics. It is usually best to give it beneath the skin in the region of the pain. In every case of tumor it is important to take the usual precautions to prevent the occurrence of bedsores and of cystitis.

The triumphs of modern surgery in the successful removal of certain tumors of the spinal cord make it necessary to carefully consider the question of operation in every case of tumor. As yet the number of cases in which an operation has been attempted is too small to permit a statement either of the exact indications for surgical interference or of the results that may be expected to follow such interference in different classes of cases. It is claimed by some writers that every case of tumor of the spinal cord which is not specific in character may properly be subjected to operation, but it is clear from pathological considerations that there are a considerable number of tumors which damage the cord in such a way as to render impossible any restoration of cord functions after their removal. This is certainly the case with the chief medullary tumors, the massive tubercles which develop usually in the gray substance, and the gliomata which spring from the cell elements about the central canal.

There are also many intradural meningeal tumors which, though originating in the membranes, invade and destroy the nerve elements of the cord as extensively and irretrievably as though they originated in the cord itself. This is often the case with sarcomas, and sometimes it is true of syphilitic nodules and of carcinomatous growths. In other cases, although the destruction of cord elements by the tumor itself is not extensive, there is an adjacent focus of myelitis which extends transversely across the cord. All these cases are obviously unsuitable for operation. Of the extradural tumors there are few in which an operation is not indicated, excepting the malignant growths that spring from the vertebræ.

Taking into consideration all classes of tumors that damage the spinal cord, it may be said that the anatomical conditions are such as to encourage the hope of some benefit from operation in about half the cases. This is a rough estimate, but it is probably approximately true. In practice it is not possible always to distinguish the cases in which the pathological conditions warrant interference from those in which they do not. It is probably not far from the truth to say that an operation is indicated in all cases of tumor of the spinal cord in which there is no evidence either of malignancy or of a growth starting within the substance of the cord, or of multiple tumors, or of a complete transverse lesion of the cord.[1] If after careful consideration of the evidence there is a reasonable doubt as to the presence of the conditions mentioned, operative interference should be advised. A syphilitic growth which resists specific treatment for more than two months probably does not contraindicate operation.

It is too early to speak with confidence of the results which opera-

[1] In tumors above the lumbar enlargement a complete transverse lesion is indicated by complete motor and sensory paralysis below the level of the lesion associated with loss of knee jerks and a flaccid condition of the paralyzed muscles.

tion is likely to yield in the different classes of tumors in which surgical interference is justified.

Of the 21 cases collected by Starr[1] in which there was an operation for the removal of a tumor of the spinal cord, there were only 6 recoveries from paraplegia and 1 partial recovery. In 1 of the 6 cases the tumor compressed the cauda equina. In 3 of the remaining 5 cases the tumors are described as extradural connective-tissue tumors (Macewen's cases), but it is not clear whether they were sarcomata or fibromata or that they really were new growths. It is thus evident that the proportion of recoveries is small. In most of the cases that have been operated upon there has been little or no improvement in the symptoms, and death has occurred soon after the operation. It may reasonably be expected that with the greater accuracy in diagnosis and improvement in surgical technique to which we may confidently look forward the number of successful operations will be considerably increased. But at its best the operation must ever be regarded as a grave one. Nevertheless, its use is amply warranted by the results already obtained.

It is exceedingly important to emphasize the necessity of avoiding delay in operating after the diagnosis of tumor has been established with reasonable certainty. As already stated, the damage to the cord is necessarily considerable at the time when the diagnosis is first possible. To permit further delay is therefore most injudicious.

SYRINGOMYELIA;[2] SLOW GLIOSIS OF THE SPINAL CORD.

DEFINITION.—Syringomyelia literally means a cavity within the substance of the spinal cord, but the term is properly applied only to those cases where the cavity in the cord is due to the breaking down of certain proliferated neurogliar structures which infiltrate the substance of the cord (gliosis of the spinal cord), or to faulty closure of one of the divisions of the primary central canal of the cord associated with neurogliar hyperplasia. Such cavities, therefore, as depend on poliomyelitis or on abscess formation or on trauma to the cord are distinct from syringomyelia. Distinct also is that form of cavity within the cord which consists merely in a dilatation (usually congenital, sometimes due to pressure by tumor) of the central canal of the spinal cord. The term *hydromyelus* is used to designate this condition. Hydromyelus is to the spinal cord what internal hydrocephalus is to the brain as regards the position of the cavity and the nature of its lining. It may or may not be associated with hydrocephalus or other congenital deformity. It probably gives rise to no recognizable symptoms in itself if unassociated with neurogliar hyperplasia, and is accidentally discovered at autopsy.[3]

[1] Pfeiffer has reported a case in which, owing to an error in diagnosis, no tumor was found at operation.

[2] Schlesinger's recent monograph should be consulted by those who wish to make a study of the disease in detail.

[3] There is, of course, no sharp line to be drawn between simple hydromyelus and hydromyelus with neurogliar hyperplasia originating in superabundant residual epiblastic tissue, but the former condition probably causes no recognizable symptoms.

The essential feature of most cases of syringomyelia which give symptoms is the gliosis which precedes cavity formation, and, clinically, gliosis without cavity formation may be indistinguishable from gliosis without a cavity. The cavity is thus often of quite secondary importance.

There is a form of gliosis of rapid development which is closely related to the central gliomata of the spinal cord. Cavity formation may occur in these cases, but, owing to their distinct clinical features, they will not be included in the description of syringomyelia or (preferably) slow gliosis of the spinal cord.

Etiology.—Nothing is known of the conditions which stimulate the multiplication of the embryonic cells of and about the ependyma of the central canal of the spinal cord, which constitutes the first stage in the process that results in syringomyelia. It has been observed that syringomyelia not rarely begins to develop after a severe traumatism to the spine, and, although it is easy to exaggerate the importance of this factor, there is good reason to think that injury distinctly favors the development of the disease. A considerable number of cases occur after infectious diseases, especially after typhoid fever. Syphilis and alcoholism apparently have no influence upon the origin of the disease. The disease often occurs in persons in the best of health. It is doubtful if it is ever a family disease. The symptoms seldom come on before puberty or after the fortieth year. Nearly half the cases begin between the twentieth and thirtieth years. It is a little more common in males than in females, but the preponderance is not so great as some authors have claimed.

There is no satisfactory evidence in favor of the view that ascending neuritis may furnish the stimulus necessary for the initial proliferation of the residual ependymal cells. There is some reason to think that a hemorrhage into the central canal of the cord may occasionally be the starting-point of the disease (Minor), but no decided opinion can as yet be expressed on this point. Slow gliosis of the spinal cord is not an extremely rare disease. The writer believes it to be no more uncommon than cases of spinal progressive muscular atrophy, from which it was formerly not distinguished.

Pathological Anatomy.—In a large majority of cases, if not in all, the condition which lies at the basis of the pathological process is a congenital anomaly in the development of the spinal cord, as indicated by the presence of an enlarged central canal with an excessive number of lining epithelial cells, or of diverticula of the canal, or of one or more accessory central canals, or doubling of the canal, or of nests of residual embryonic cells of the ependymal type, or of several of these variations.[1] In consequence of some stimulus, as a traumatism, or spontaneously (that is, by the action of some unknown stimulus or by the removal of the normal inhibitory forces), certain of these residual ependymal or periependymal cells begin to proliferate, and to extend toward the periphery of the cord, producing as they infiltrate its substance a basement material (consisting of homogeneous material or of

[1] The imperfect closure of the posterior division of the primary central canal of the cord (which forms the posterior fissure) may be the chief basis of a future cavity in the cord.

fine parallel fibres or of a network of fine interlacing felted fibres) in which they lie. The newly-formed cells are in the beginning of the same type as the parent ependymal cells, but as they proliferate toward the periphery they grow smaller, and grow in time to be indistinguishable from fully-developed glia cells. This glia hyperplasia, or *gliosis*, is often followed by the narrowing or even entire obliteration of the central canal, from the pushing in of its epithelial lining in places. The gliosis has not the same characters in all parts: in places the cells are particularly numerous and the basement substance scanty; in other places the cells are scarce and lie in a compact network of fine fibres. The former structure breaks down readily; the latter is more resistant.

When the proliferative process is well advanced changes of a retrogressive nature make their appearance, similar to those which occur in the central portions of a glioma of the brain, and these changes are especially marked in parts near the starting-point of the still spreading gliosis. These changes consist in hyaline, gelatinous, mucoid, and allied metamorphoses in the glia cells and fibres, which soon lead to the breaking down of these and the adjacent nervous tissues, with the consequent formation of a pathological cavity in the cord. The immediate cause of the tissue alterations just mentioned is doubtless the degeneration which occurs in the bloodvessels of the glia-invaded territory. These vessels undergo hyaline degeneration; their lumina grow narrow from the thickening of the vascular walls, and in places thrombosis leads to complete occlusion. The starved tissues undergo necrosis and a cavity may result. The cavity is formed at the expense both of the new formation and of the nervous structures which are implicated by it. When, however, mere invasion occurs without cavity formation, the nervous elements invaded are not necessarily destroyed, and may be so far unaltered as to retain some functional activity. The nerve fibres are particularly resistant, and actual destruction of axis cylinders is rare in degenerated nerve fibres even when adjacent ganglion cells are greatly atrophied. Local hemorrhage and œdema of the cord are not rare occurrences.

The gliosis begins in a single segment of the cord or in a few adjacent segments, and not in the entire extent of the cord, in which a cavity may be afterward found. The cervical region is the part in which the process most often begins, but it may commence at any level. As the proliferative process usually begins in cells posterior to the central canal, it is not singular that the cavity usually extends into the posterior commissure, and thence into the posterior horn or into the white substance of the posterior column. Frequently the anterior horn is invaded. The lesion may remain limited to the part where it begins, or may extend up or down or in both directions until the cord is involved in its entire extent. When the extension is upward the fifth nerve is prone to suffer, and even the nuclei of the vagus and hypoglossal nerves may be damaged. In size and contour the cavity varies much in the same and in different cords. It is generally lined in some part of its extent, occasionally throughout, with cylindrical epithelium, which is often continuous with that of the central canal. Two cavities may be present. Owing to the predilection of the morbid process for the posterior horns, any invasion of the white substance is apt to

damage the posterior columns or the crossed pyramidal tracts, with resulting secondary degenerations. If the gliosis extends to the surface of the cord, there may be produced a local chronic leptomeningitis, and to this may be added in time a chronic fibrous pachymeningitis. It sometimes happens that the posterior nerve roots undergo partial degeneration, especially where they pass through the pia. A secondary ascending degeneration of the posterior median columns then occurs. When the ganglion cells of the anterior horns are destroyed the anterior nerve roots and the motor fibres of the peripheral nerves are degenerated, though the latter often suffer less than one might expect. The corresponding muscles undergo degenerative atrophy. It is very rare to find the sensory peripheral nerves altered. The lesions that have been described are not all present in every case of syringomyelia. In some cases the gliosis is confined to the region of the cord about the central canal, and there is no cavity formation. In other cases the neoplastic process is of all but universal extent, and the cavity may be so large as to convert the cord into a mere thin-walled tube. Between these extremes every imaginable transitional form has been observed. The existence of a cavity is not a necessary feature of slow gliosis of the spinal cord, although it is usual.

Cavity formation may occur in the course of a morbid process allied with, yet distinct from, slow gliosis—namely, rapid gliosis or gliomatosis of the spinal cord. In this condition the cord is increased in size and consistence by the development of a centrally situated glioma or glio-sarcoma or myxo-glioma or angio-glioma. Beginning in the cells of the ependyma of the cord, the gliomatous tumor extends with rapidity in all directions until it involves a considerable part of the length of the cord, and occupies, in section, all but a narrow strip of the periphery. Though this condition cannot be sharply separated from syringomyelia on pathological grounds, it differs from it in its greater cellular richness, in its more rapid and even extension, and in the slighter tendency to cavity formation. Clinically, this process runs a course quite different from that of syringomyelia, which will be described elsewhere (see Tumors of the Spinal Cord, p. 266).

The different conditions in which may be observed cavities of considerable longitudinal extent may be classified as follows:

1. *Hydromyelus* (congenital dilatation of the central canal), which runs a latent course.

2. *Slow gliosis of the spinal cord*, with or without hydromyelus: (*a*) without cavity formation; (*b*) with cavity formation (including most cases of syringomyelia).

3. *Rapid gliosis* or *gliomatosis*, with or without cavity formation (and with different clinical history from 2 *b*).

Symptoms.—The varied symptomatology of syringomyelia is readily understood if the facts of pathological anatomy are kept in view. It is essential to realize that the distribution and extent of the symptoms must vary with the localization and extent of the lesions, and that the character of the symptoms must depend in part on the position of the lesion and in part on its nature. As regards the nature of the lesion, it should be noted that the gliosis is competent to cause irritative symptoms, such as pain and hyperæsthesia, on the one hand, or symptoms

of a destructive character on the other, according as the nerve elements are merely infiltrated or destroyed.

Since the morbid process usually begins in the cervical region and extends longitudinally or transversely from the area about the central canal into the gray substance especially, it is evident that when the anterior horns are implicated there will be a resulting atrophic paralysis of one or both upper extremities, and that disturbances of sensibility with trophic and vasomotor affections of the skin should arise when the posterior horns are invaded. In distribution the sensory symptoms may correspond closely or only remotely with the motor symptoms. As the lesion is apt to be symmetrical, the symptoms are usually symmetrical, but either side of the body may be exclusively affected for a time.

The paralysis and wasting often begin in the muscles of the hand or shoulder (as in spinal progressive muscular atrophy), whence they may spread to adjacent parts as the lesion extends up or down the cord. The sensory symptoms may be ushered in with pain and paræsthesia in one or both arms. Then there develops a characteristic but readily overlooked symptom, the so-called dissociated anæsthesia. This consists in the partial or complete loss of sensibility to pain[1] and to temperature, with preservation or only very slight impairment of the tactile sense. The loss of sensibility usually begins in the fingers, and gradually extends centrally and may spread to the side of the chest. For a long time it may involve the skin in small irregular patches, but in time large areas are generally affected. Usually the sensory loss increases very slowly in intensity and extent, but at times the advance is rapid. Once established, there is seldom any retrogression in this symptom. The mucous membranes very often share in the disturbance (mouth, nose, eyes, even urethra, bladder, vagina). The various forms of muscular sense are not often lost, but are apt to be somewhat impaired. Owing to the loss of warning sensations of pain, patients are liable to injure themselves by contact with hot water, hot kettles, etc., which favors the appearance of trophic disorders of the skin. The dissociated anæsthesia is due to the interruption by the lesion of the fibres that convey the sensation of pain and temperature from the posterior nerve roots to the columns of Gowers. These fibres decussate in the gray matter about the central canal, and are thus easily damaged by the lesions of syringomyelia.

Of the vasomotor and trophic affections of the skin which occur, may be mentioned local hyperæmia, urticaria, glossy skin (neuritic dermatitis), ulcers, phlegmons, local gangrene, and various anomalies in the growth of the nails. Trophic affections of the bones and joints are not rare. Arthropathies occur which resemble those of tabes. They consist of effusions into one or more joints, with little or no pain, and with atrophic or hypertrophic change in the ends of the bones. About 80 per cent. of the arthropathies affect the upper extremities, especially the shoulder and elbow-joints. They are much more common in men than in women, and a considerable number (more than 10 per cent.) have followed local traumatism. Spontaneous fractures may occur. In many cases the vertebral spines become tender, and slight

[1] The loss of sensibility to pain usually extends to deeply lying structures.

scoliosis or kyphosis develops (especially in the dorsal region). Occasionally acromegaly is associated with syringomyelia. The various trophic affections of the skin and joints are to be referred to damage to the trophic fibres in the posterior horns or in the posterior nerve roots.

When the lesion extends only into the posterior horns paralysis and wasting are absent. If the process extends from the posterior horn into the pyramidal tract on one or both sides, one-sided or bilateral spastic symptoms arise—that is, there are partial paralysis of one or both legs, rigidity, increased knee jerks, ankle clonus, and sometimes violent spontaneous jerking of the limbs. But of course these spastic symptoms do not arise when the anterior horns are destroyed, no matter how seriously the pyramidal tracts are implicated. Extension of the lesion into the posterior columns causes loss of tactile sensibility in addition to the sensory symptoms already mentioned, and this loss may be insular or girdle-like when the posterior roots are involved. When the lesion extends back, not in the gray matter, but along the line of the posterior fissure, ataxia and loss of tactile sensibility may appear before the more characteristic symptoms, but this is rare. Pain in the back of the neck may be a prominent symptom when there is chronic meningitis. When the gliosis extends upward from the cervical region along one of the posterior horns, there is an extension of the dissociated anæsthesia to the occiput if the origin of the great occipital nerve is implicated, and to one side of the face if the sensory trigeminus root is damaged. The fifth nerve is thus damaged in about 10 per cent. of all cases of syringomyelia. Paralysis and atrophy of the tongue, laryngeal palsy, and facial paralysis arise if the corresponding nuclei are invaded, but these symptoms are rare. It occasionally happens that bulbar symptoms exist alone or before the arm or shoulder symptoms appear. This happens in the rare cases where the gliosis begins in the medulla. Bulbar symptoms are present at some period of the disease in about one third of the cases. Optic neuritis is a very rare occurrence, but symptoms due to damage to the cilio-spinal centre are not infrequent. They consist in contraction of the pupil, narrowing of the lid aperture, sinking of the eyeball, and disturbances of secretion upon the side of the lesion.

Sometimes the lumbar region of the cord is first implicated. Then the legs suffer first; there may be loss of knee jerks and the sphincters may be paralyzed—a very uncommon occurrence in syringomyelia. It has also happened that a diffuse lesion of the lower dorsal region has been associated with a lesion extending along the posterior horn on one side as high as the medulla; the symptoms were those of a diffuse lower dorsal myelitis with extensive one-sided dissociated anæsthesia.

Another important clinical variety of syringomyelia is that which presents the association of symptoms known as Morvan's disease. This condition (oftenest seen in Brittany, but not unknown in the United States) is characterized by atrophy of the muscles of the hand and forearm, analgesia, thermo-anæsthesia, and often some loss of tactile sensibility in the skin of the atrophic parts, together with the painless development of destructive whitlows and other trophic disturbances. It has been shown conclusively that the symptoms observed in Morvan's disease are usually due to syringomyelia, and it is probable that

the neuritic changes that have been observed are secondary in these cases to the spinal lesion. But it has also been shown that Morvan's disease may be caused by leprosy, and it is likely that where leprosy abounds most cases of the affection are due to the infection of leprosy.

For the sake of convenience we may summarize the symptoms of the chief types[1] of syringomyelia as follows:

Types.—(1) *Cervical Type* (*common form*).—Atrophy beginning in muscles of hand, paræsthesia, and dissociated sensation in fingers and hand; later, atrophy and paralysis of shoulder-girdle and upward extension of analgesia and thermo-anæsthesia, claw-hand, scoliosis, loss of reflexes in arms, exaggeration in legs, weakness and spasm in legs, sympathetic paralysis; finally, walking impossible, involvement of cranial nerves, wide extension of sensory disturbance, inflammation, and vesicular eruptions of skin.

(2) *Dorso-lumbar Type* (*rare form*).—Atrophy and weakness, beginning in the legs (especially adductors and extensors of knee), radiating lumbar pains, girdle sensation, dissociated sensation, tremor or convulsive jerkings of muscles; later, marked wasting and paralysis (sometimes one-sided), inco-ordination; finally, retention of urine, sphincteric paralysis, extreme paralysis and wasting, lumbar kyphosis, perforating ulcer of foot, perhaps spontaneous gangrene.

(3) *Cases with Preponderant Motor Symptoms.*—(*a*) Cases of the amyotrophic lateral sclerosis type, beginning often with rigidity of neck muscles or with jerking in one arm, followed by gradually extending weakness and atrophy, exaggerated reflexes, and early contractions. Sensory disturbances come on very late.

(*b*) Cases of the spastic paraplegia type, beginning with weakness and spasm in legs, which is progressive and extends to arms. Reflexes are exaggerated. Paræsthesiæ are common, but dissociated sensation is a late symptom. Muscular atrophy occurs late.

(*c*) Cases of the scapulo-humeral type, in which there are pronounced weakness and wasting of the muscles of the shoulder girdle, whilst arm and forearm muscles remain intact for a long period. Sensory symptoms late and slight. Bulbar symptoms common and early.

(4) *Cases with Preponderant Sensory Symptoms.*—(*a*) Hemianæsthetic type, in which there is one-sided dissociated anæsthesia, with little or no motor disturbance; trophic symptoms may become prominent, and in some cases there is considerable muscular atrophy.

(*b*) General anæsthetic type (very rare form), in which all forms of sensibility are lost below the neck.

(5) *Cases with Preponderant Trophic Symptoms.*—These comprise the cases of Morvan's disease.

(6) *Cases of the tabetic type*, in which the symptoms of tabes are combined with those of syringomyelia.

Complications.—The most important and frequent complication is hysteria. A variety of other nervous diseases has been found in association with syringomelia (epilepsy, chorea, tetany, Basedow's disease, cervical pachymeningitis, spinal syphilis, poliomyelitis, tumors of the spinal cord, hydrocephalus, general paralysis, etc.), and are of importance in so far as they affect the diagnosis and prognosis.

[1] These are the types recognized by Schlesinger.

Diagnosis.—With the recent increase in our knowledge of the symptoms of slow gliosis of the spinal cord it has become possible to make the diagnosis of syringomyelia in a large number of cases, probably in a very large proportion of cases. Those which have been described as producing no symptoms during life have probably been congenital hydromyelus, and not cases of slow gliosis.

The diagnosis of syringomyelia is based on the combination with dissociated anæsthesia of slow muscular atrophy, paralysis, and trophic symptoms. When these symptoms are present and the muscular atrophy is of the Aran-Duchenne type (cervical type) or of the scapulo-humeral type, the diagnosis presents few difficulties. The presence of slight kyphosis is a very important auxiliary symptom. When the disease is of the dorso-lumbar or tabetic type or of the type in which the motor symptoms preponderate over the sensory symptoms, the diagnosis may be difficult or impossible. It would be out of place to enumerate the special diagnostic difficulties which arise; they may be, for the most part, inferred from the description of the varied symptoms of syringomyelia.

As intimated above, the diagnosis of syringomelia should be based on the association of several characteristic symptoms, and not upon a single symptom, however characteristic. But it should not be inferred that the characteristic symptoms have equal diagnostic value. Of individual symptoms the presence of dissociated anæsthesia is by far the most important, and its existence in typical form (*i. e.* complete preservation of tactile sense with absolute loss of pain and temperature sense) is by itself highly suggestive of syringomyelia. So far as we now know, there is only one other condition in which this symptom occurs in typical form—namely, central hæmatomyelia. In this condition the sensory paths in the central gray matter are destroyed in considerable longitudinal extent by a hemorrhage, and dissociated anæsthesia is produced as in syringomyelia. In cases of central hæmatomyelia the symptoms are of rapid or sudden onset, and this fact sufficiently distinguishes these cases from syringomyelia.

The presence of a partially developed dissociated anæsthesia (considerable analgesia and thermo-anæsthesia, with disproportionately slight loss of tactile sense), while it has not the value of the fully-developed symptom, is nevertheless a highly significant symptom. In a case in which dissociated anæsthesia cannot be detected it is doubtful if a diagnosis of syringomyelia could be properly made, although other symptoms might justify a suspicion of its existence.

Prognosis.—Syringomyelia is a disease of exceedingly slow development, and the prognosis, so far as life is concerned, is usually a good one. The duration of cases of slow gliosis (without intercurrent affections) is often from twenty to forty years, and as the disease commonly begins in early adult life, the patient may reasonably hope to reach his sixtieth year if he possesses the means and intelligence to care properly for himself. As a rule, the prognosis is best when the disease is located in the cervical and upper dorsal regions. When it involves the lumbar region the outlook is less good, because of the danger of cystitis and pyelonephritis. When the process invades the medulla the prognosis is rendered distinctly less favorable, as regards the duration of life, than

if it remains confined to the cervico-dorsal region; but bulbar symptoms may be present many years without causing death. The significance of bulbar symptoms in syringomyelia is thus less grave than their presence in spinal progressive muscular atrophy. Death from a bulbar lesion is to be regarded as rare in syringomelia. Death oftenest results from phthisis, pneumonia, typhoid fever, or some intercurrent infectious disease, but death from the consequences of cystitis or from septic infection from local suppurative affections is by no means uncommon.

Remissions in certain symptoms of syringomyelia are not very rare. They may come on at any time in the course of the disease, and may last for months or even for years. The improvement may relate to paralysis, to wasting, to trophic disturbances, to bulbar symptoms, to sphincteric disturbance, and even to analgesia and thermo-anæsthesia. The cause of the remissions is obscure. Possibly it is referable to diminution of the œdema of the cord, which is no uncommon occurrence. However marked they may be, the remissions are not to be regarded as evidence of a reparative process in the cord.

In some cases certain symptoms of the disease grow acutely more pronounced or new symptoms appear rapidly or even suddenly. This is especially apt to be the case with bulbar symptoms. The exacerbations are thought to be due to vascular disturbances, especially spontaneous hemorrhage (which is not very rare), or to a rapidly increasing œdema.

TREATMENT.—It is evident from the nature of the lesions which form the basis of the disease that treatment directed to the removal of these lesions must be useless. There are, however, symptoms which may be modified by means of therapeutic measures.

For the relief of the severe pain which sometimes accompanies the disease it is best to use antipyrine, antifebrine, phenacetine, or ammonol. The use of lukewarm baths of ten or fifteen minutes' duration may greatly alleviate pain for a time. They may be given twice daily. Morphine should not be employed until all other measures have failed, for the disease is of such long duration that the establishment of the morphine habit is greatly to be regretted. Moreover, the period of severe pain is usually of short duration.

The early use of massage, passive movements, and of local hot-water baths (105° F.) for fifteen minutes, twice daily, does much to prevent serious contractures. Effusions into the joints may be treated by aspiration. The usual surgical means may be employed for the treatment of suppurative affections of the joints and exostoses.

Prophylactic measures are of the utmost importance. The patient must be guarded constantly from contact of the analgesic portions of the body with hot objects. Slight traumatismus and slight inflammatory affections should be treated promptly. Their neglect often leads to extensive trophic lesions, which may endanger the well-being of the patient. Treatment by suspension is probably dangerous on account of increasing the liability to hemorrhage into the spinal cord. All forms of electrical treatment are probably useless.

As in all varieties of chronic spinal-cord disease, it is essential to look carefully after the general nutrition, for it is likely that all influences which impair the general health tend to shorten the life of the patient.

MALFORMATIONS OF THE SPINAL CORD.

MALFORMATIONS of the spinal cord are usually classified as congenital or acquired, but no absolute separation of these two forms can be justified on biological grounds. Thus, syringomyelia, which has been regarded as an example of an acquired malformation of the spinal cord, is surely due to developmental errors which have their beginning in embryonic life. The only congenital malformation of the spinal cord which possesses clinical importance is spina bifida. It is true that a great variety of displacements of the gray and white matter have been described as examples of *heterotopia*, but it has been conclusively shown by Van Gieson that the majority of the cases described as heterotopias are the consequence of rude handling of the spinal cord in the course of its removal. True heterotopias occur, but are extremely rare. They are apt to consist in a separation of a bit of the gray matter or of the substantia gelatinosa from the rest of the gray substance. There is no reason to believe that the true heterotopias observed have given rise to symptoms. Deformities of the cord are sometimes observed in monstrosities. Thus there is a condition of *amyelia* in which the cord is congenitally absent (the brain usually being absent also). Sometimes the cord is greatly reduced in size (*atelomyelia*) or is divided into two halves longitudinally (*diastematomyelia*), each covered with its own pia, or there is a union of two cords (*diaplomyelia*), as in the case of some double monsters. In cases where there has been defective development or intra-uterine amputation of a limb the spinal cord presents a corresponding want of symmetry, especially in its gray substance.

Spina Bifida.

DEFINITION.—Spina bifida is a malformation of the vertebral column which permits the protrusion of some portion of the contents of the vertebral canal as an elastic compressible tumor containing cerebrospinal fluid, which is usually increased in size by respiratory spasm (as in crying), and may be increased by pressure on the anterior fontanelle. The disease is one of surgical interest chiefly, and only the barest mention of the more important facts will be undertaken here.

ETIOLOGY.—Spina bifida depends upon a congenital defect in the development of certain vertebral arches due to a deficiency in the growth of mesoblast from either side, which normally encloses the embryonic spinal cord and forms bone. The cause of this defect is to be sought in the germ-plasm, certain cell elements of which do not possess the capacity to proliferate fully along the normal lines of growth. The writer has noted that in the cases observed by him one or both of the parents have usually been immature or badly nourished. Very rarely one parent has spina bifida. Spina bifida is said to occur in about one child in every thousand born (Chaussier).

PATHOLOGY.—The essential element in spina bifida has just been referred to. In many cases the defect is not confined to the vertebral arches, but implicates the mesodermic tissues (corium, muscle) which normally cover the spine. The failure on the part of these tissues to grow gives the structures within the canal little or no support and allows their protrusion.

According to the nature of the contents of the protrusion there are several varieties of spina bifida. Of these the chief are (1) meningocele, (2) meningo-myelocele, and (3) syringo-myelocele.

(1) In a *meningocele* the sac contains only the spinal membranes; the tumor is sessile, sometimes pedunculated, and may attain a diameter of five or six inches. This variety is oftenest found in the cervical region.

(2) In a *meningo-myelocele* the sac contains the cord as well as the membranes, the tumor is sessile and never pedunculated, is only partly covered by skin (there being a central area covered only by a thin translucent epithelial membrane), is small in size (about the size of a small orange), and occurs in the lumbo-sacral region. The tumor often has a vertical furrow or umbilication which marks the attachment of the spinal cord. Ordinarily the cord or cauda equina runs horizontally backward across the upper portion of the tumor to the umbilicated spot, from which the nerves then pass forward to re-enter the spinal canal and pass to their distribution. Sometimes the nervous structures join the wall of the sac on entering the tumor, and the much attenuated nerves spread out all over the inner surface of the sac to converge below and re-enter the spinal canal. About two thirds of the cases of spina bifida are of this variety.

(3) In *syringo-myelocele* the central canal of the cord is distended with fluid and the sac is lined by the atrophied structures of the cord and cauda equina.

Other errors of development are not rarely associated with spina bifida (club-foot, hydrocephalus, occasionally encephalocele, cerebral meningocele, or hare-lip). Hydrocephalus is most often associated with syringo-myelocele. In what is known as spina bifida occulta there is no external tumor, but the defect in the arches can be felt on palpation, and, as in all forms of spina bifida, the spinal cord generally extends considerably lower than normally.

Symptoms.—Where the spinal cord is not damaged symptoms may be wholly absent. This is usually the case in meningocele. In meningo-myelocele and in syringo-myelocele there is commonly some damage to the cord or nerves, and paralysis in the lower extremities results. The paralysis and wasting are especially marked below the knee. The anterior tibial group often suffer less than the posterior, and talipes varus occurs. The sphincters may or may not be implicated. Rarely changes like those seen in tabes are found in the tarsal and metatarsal bones, or perforating ulcer of the foot may occur. Sometimes the knee jerk is absent on one or both sides.

Prognosis.—The prognosis is best in meningocele. In meningo-myelocele and in syringo-myelocele with well-marked paralysis the outlook is always bad, and if complicated with hydrocephalus it is hopeless. In Demme's cases there were 7 recoveries and 15 deaths out of 25 cases of spina bifida operated upon, and 28 deaths within the first month in a total of 32 cases which were not operated upon. The causes of death are marasmus, infection (with or without rupture), and convulsions following rupture of the sac and the rapid draining of fluid.

Diagnosis.—A pedunculated translucent tumor without palpable fissure of the spine is almost certainly a meningocele. An umbilicated

sessile tumor with palpable vertebral fissure and umbilication of the tumor indicates a meningo-myelocele, even if there is no paralysis. The distinction of meningo- from syringo-myelocele is not always possible. The presence of hydrocephalus renders the latter condition likely.

TREATMENT.—In every case the tumor should be guarded from pressure and injury, and this may best be done by means of a rubber ring-cushion which encircles the protrusion. The surface should be kept clean and aseptic, especially where the corium is deficient. Bismuth subnitrate or iodoform should be applied to the surface. The presence of a considerable degree of paralysis or of hydrocephalus or of advanced marasmus contraindicates any operative procedure. In other cases the question of operation is to be considered. Various procedures have been employed, the most satisfactory of these being aspiration and injection. Almost one half of the contents of the tumor is first evacuated. Then a drachm of Morton's fluid is injected without removal of the syringe. This fluid consists of iodine gr. x, iodide of potassium gr. xxx, glycerine ℥j.

The child should be kept in the recumbent position for several hours after the injection. After a period of inflammatory reaction lasting several days there is in successful cases a gradual contraction of the tissues of the tumor, with eventual obliteration of the sac. This method appears to have been successful in almost one half the cases in which it has been used. But even where the operation is surgically a success the symptoms due to damage of the cord (paralysis, incontinence) almost invariably increase and lead ultimately to death. It is best not to subject a child to this operation until it is six months of age, unless there is danger in waiting. Simple aspiration and compression are useless. Excision of the sac of ligation is of course permissible only in cases where the cord or nerves do not enter the tumor. Judging from the report of the London Clinical Society (1885), the cord or nerves enter the sac in almost 95 per cent. of all fatal cases of spina bifida.

DISEASES OF THE MEDULLA AND PONS.

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By CHRISTIAN A. HERTER, M. D.

PROGRESSIVE BULBAR PARALYSIS.

Synonyms.—Chronic bulbar paralysis; Labio-glosso-laryngeal paralysis.

Definition.—An afebrile disease of the second half of life, distinguished by gradual progressive impairment of speech, phonation, mastication, and deglutition, dependent on progressive symmetrical degeneration of the principal motor nuclei of the medulla. It is intimately related to progressive muscular atrophy.

Etiology.—Progressive bulbar paralysis is a very rare disease in most cases of which no distinct cause can be traced. It begins usually after the forty-fifth year, but has been known to occur in early life. Males suffer somewhat more often than females. Syphilis has not been shown to be a cause, but there is some reason to think that toxæmic states (lead-poisoning, diphtheria) may give rise to it. Over-use of the muscles, injury, exposure to cold, emotion, and debilitating influences generally have been suspected, but it is not clear what rôle, if any, these influences play. An acute nuclear inflammation has been followed by the chronic disease.

Pathology.—The primary lesion consists in a bilateral degenerative atrophy in the nuclei of origin of the hypoglossal, spinal accessory, and less constantly in the vagus nuclei, and only rarely in the nucleus ambiguus, the glosso-pharyngeal, the motor nucleus of the fifth, or in the facial nucleus. In general, the nuclei implicated are representatives of the continuation of the ganglion cells of the anterior horns. Some of the raphé fibres at the hypoglossal level may be degenerated and the posterior longitudinal bundle may be involved in part. The pyramidal tracts may or may not be implicated in the bulbar pontine region. When, as often happens, the disease is complicated with paralysis in the trunk and extremities, the anterior cornual cells and the motor paths in the spinal cord are the seat of degeneration. The histological changes are not always the same in character, and suggest that the pathological alterations are sometimes chiefly parenchymatous, sometimes chiefly interstitial. These changes are essentially the same in nature as those observed in the closely allied disease, progressive muscular atrophy.

The peripheral nerve fibres corresponding to the nuclei involved

undergo degeneration which extends to the intramuscular nerve endings and the muscle fibres. It must be admitted that we know nothing of the ultimate pathology of the disease, for we are ignorant of the conditions that induce those nutritional changes in the nerve elements that must underlie the atrophic process.

Symptoms.—The tongue is usually involved first. Speech gradually becomes indistinct, owing to the imperfect articulation of the linguals, *l*, *n*, *d*, and *t*. The tongue cannot be placed firmly against the roof of the mouth as in making *p* and *b*, and is in time protruded with difficulty. Very early, as a rule, the lips become weak and the patient cannot make the labials or whistle. The palate and pharyngeal muscles soon become weak and swallowing is impaired. As the paralysis advances it becomes impossible to close the lips, the saliva dribbles from the mouth, the lower part of the face becomes motionless and expressionless, articulate speech grows impossible, and it finally becomes all but impossible to swallow. In some cases there are paralysis and atrophy of muscles of of the upper part of the face and of the eyeball.

There is no change in sensibility in any of the parts implicated. The throat reflex is lost, sometimes early, and increases greatly the chance of food entering the larynx. The tongue is often considerably wasted, but may remain normal in size, when it is apt to be soft and flabby. The lips may or may not be wasted. The electrical reactions are usually little altered; sometimes a partial form of R. D. is found. In the cases where there is no wasting reflex action is apt to be preserved, and may even be increased. It is probable that in these cases the fibres of the pyramidal tracts are implicated out of proportion to the nuclei—a condition comparable to that found in the amyotrophic lateral sclerosis type of progressive muscular atrophy. Intellect is usually unimpaired, but there is apt to be considerable emotional instability.

The association of progressive bulbar paralysis with progressive muscular atrophy is not very uncommon. Sometimes the bulbar, sometimes the spinal, symptoms are the first to appear and dominate the clinical picture. Very rarely the muscles of the upper part of the face and of the eyeballs are implicated in bulbar paralysis, and the muscles of mastication have been known to be involved.

Although the disease is progressive, its advance is not uniform. In almost every case there are periods of weeks or months in which the symptoms are at a standstill. The entire duration of the disease is very variable (six months to ten years), but the majority of cases terminate fatally in from two to four years. Very rarely a case beginning in early life undergoes arrest after reaching a moderate degree of development. The chief causes of death are extreme debility from inanition, deglutition, broncho-pneumonia, failure of respiration from implication of the vagus centres, or atrophy of respiratory muscles, and intercurrent diseases (especially lobar pneumonia).

Diagnosis.—The cardinal features of progressive bulbar paralysis are the distribution of the paralysis, the gradual onset and development, the symmetry of the symptoms, and the age of the patient. The vascular lesions of the bulb are distinguished by their acute onset. The chronic inflammatory processes and tumors of the medulla are distin-

guished chiefly by the unsymmetrical symptoms they cause, their greater liability to implicate deglutition and respect the lips, and the presence of other cerebral symptoms (headache, optic neuritis). A chronic sclerotic process in each hemisphere has been known to cause symptoms similar to those of progressive bulbar paralysis. The symptoms in such a case are those of a double hemiplegia with involvement of the face and normal bulbar reflexes. The condition is known as "pseudo-bulbar paralysis." Ordinarily, the pharyngeal paralysis of diphtheritic multiple neuritis is readily distinguished from progressive bulbar paralysis, but cases of true bulbar paralysis have been known to succeed diphtheritic paralysis after several months, and the conditions have been confused. In general it may be said that so long as the muscles affected have undergone no atrophy, there is a possibility that the diagnosis of progressive bulbar paralysis is an error. It should also be remembered that there are cases of non-atrophic bulbar paralysis running a slow course and associated with weakness of the extremities and perhaps of the external ocular muscles (ptosis), which are not progressive and in which no lesions in the nervous system have been detected (see Asthenic Bulbar Paralysis, p. 284). A temporary functional bulbar paralysis of gradual development sometimes occurs in those who play wind instruments.

PROGNOSIS.—Progressive bulbar paralysis is a fatal disease in all but a few cases which begin in early life. When it forms part of an amyotrophic lateral sclerosis it terminates fatally in two or three years or less. In primary uncomplicated cases the duration is said to be usually one or two years longer.

TREATMENT.—The only rational indication is to do that which is most likely to improve the general nutrition of the patient, and with this the nutrition and functional capacity of the diseased portion of the nervous system. It is surprising to see how much improvement in voluntary power may follow the administration of an abundance of nutritious food where this has been lacking. Arsenic, strychnine, and other tonics are useful in so far as they improve nutrition. Electricity is useless. Care must be taken to avoid the entrance of food into the larynx, and the physician must be prepared for the use of feeding by the stomach tube. Atropine may reduce the salivation a little. Where the terminal respiratory disorders set in morphine should be freely used to relieve discomfort.

ASTHENIC BULBAR PARALYSIS.[1]

SYNONYMS.—Bulbar paralysis without anatomical basis (Oppenheim); Myasthenia gravis pseudo-paralytica (Jolly); Erb's disease (Murri).

DEFINITION.—A rare disease, characterized by rapid and often extreme exhaustion of muscular power, especially under the influence of

[1] The description of this condition is based mainly on that of Strümpell, in Bd. 8 of the *Deutsche Zeitschrift für Nervenheilkunde.*

functional activity, the muscles chiefly involved being those supplied by the motor bulbar nuclei, whilst any or all of the skeletal muscles are liable to suffer in lesser degree.

Etiology.—The condition usually makes its appearance during adolescence or early adult life. Of the 21 cases hitherto observed, only 5 occurred in persons over thirty years of age, and the 2 oldest of these 5 were forty-seven and fifty-five. The 4 youngest were from twelve to fifteen years of age. The two sexes were about equally represented. As yet no cause for the condition has been discovered. It has in several instances developed after an acute infectious disease (oftenest influenza), but it is not unlikely that the relation was accidental. Heredity and excessive fatigue do not appear to have played any rôle. It is indeed a striking feature of the disease that it usually appears in apparently healthy and robust persons.

Pathology.—Nothing is known of the pathology of the disease. In the five cases in which a careful examination of the central and peripheral nervous systems and of the muscles was made no pathological changes were found (Wilks, Oppenheim, Eisenlohr, Hoppe, Strümpell). One observer, indeed (C. Mayer), found changes (in preparations made by the Marchi method) in the intramedullary portions of the spinal and hypoglossal nerve roots, but it is not clear that his findings have any significance. The rapid recovery of power in the exhausted muscles when they are given rest is in harmony with the view that the symptoms do not depend on any structural change. It is not yet clear whether the functional exhaustion affects both the central and peripheral motor neurons, or whether, as seems likely, it affects especially the peripheral portion of the motor path.

Symptoms.—At first the essential feature of the disease is the rapidity with which one or more groups of muscles become fatigued on exertion. The exhaustion may be so complete that the muscles are apparently paralyzed, but in the course of a few hours or over night partial, if not entire, restoration of function occurs. After a time, however, the restoration of function becomes less complete, and a permanent paresis develops in the muscles most affected, whilst in others, which at first seemed normal, the characteristic rapid exhaustion from use appears. The order in which the different muscles are involved varies considerably in different cases, but, as a rule, ptosis is the first evidence of the disease. Very soon appears difficulty in swallowing, in mastication, and in articulation. These functions may be unequally affected. Only rarely are the eyeball muscles implicated. The implication of the facial muscles gives the face the same appearance of relaxation that is observed in nuclear bulbar paralysis. It is very rare for the paresis to be confined to the muscles innervated by the pons-medulla. As a rule, the muscles of the arms and legs are affected in the same manner as the bulbar muscles, and simultaneously with them, but in much less degree. In one case, indeed (Oppenheim), the extremities were involved before the bulbar muscles. The arm palsy is usually more marked than the leg palsy. All the voluntary muscles of the body may be affected. The degree of paresis varies considerably from day to day in some patients, quite aside from the influence of muscular exercise, and some patients speak of their "good days"

and "bad days." During the menstrual period "bad days" are apt to be numerous. Sometimes the loss of power grows very rapidly, almost suddenly, worse. Not rarely a patient is rapidly suffocated, owing apparently to paralysis of the pharyngeal and laryngeal muscles, and death has several times occurred from this condition. Sensibility is never disturbed, the sphincters are never implicated, and vasomotor changes are very unusual. At times the muscles undergo slight wasting, but the atrophy is never considerable. It has been found that when the affected muscles are stimulated indirectly by faradism—that is, through the nerve or directly—they are rapidly exhausted (Jolly).

Diagnosis.—The practitioner who is aware of the existence of this disease can have no difficulty in recognizing it and in distinguishing it from bulbar paralysis, the diagnosis being based especially on the characteristic rapid exhaustion of the affected muscles, the absence of R. D. and atrophy, the marked variations in the degree of the palsy, and on the early onset of the affection.

Prognosis.—The prognosis is necessarily always a matter of uncertainty. Some cases live only a few weeks or months; others last with more or less marked remission for many years. It may be that some cases entirely recover, but on this point nothing definite can yet be said. In the second case reported by Murri the disease had lasted twelve years, with numerous remissions and intermissions. Even in cases that are progressing favorably death may occur from suffocation without much warning.

Treatment.—There is no known means of influencing the course of the disease, but it is probable that the outlook is improved by all conditions that benefit the general health. Strümpell found strychnine and other nervine tonics quite useless. He suggests the use of a weak continuous galvanic current. It is best for the patient to be kept very quiet, with considerable rest in bed.

THROMBOSIS AND EMBOLISM OF THE BULBAR ARTERIES.

Definition.—The occlusion by thrombosis from syphilitic endarteritis or from atheroma, or by embolism of the arteries of the medulla (vertebral, anterior spinal), together with the softening of the substance of the medulla which such occlusion almost invariably entails.

Etiology.—The factors in the production of occlusion of the bulbar arteries are those which operate to cause the occlusion of vessels in other parts of the brain. Occlusion by embolism depends on the existence of endocarditis, and is essentially an occurrence of early life (childhood, adolescence, and early adult life). In thrombosis from syphilitic endarteritis the time which intervenes between the initial lesion and the onset of occlusion varies from a few months to many years, but, as the infection is apt to occur in early adult life, its effects in bringing about thrombosis of the bulbar arteries are usually felt in the first half of life.

Thrombosis from atheroma, on the other hand, is especially apt to occur in the second half of life, but its onset may be hastened through the influence of syphilis or of chronic nephritis. Blows upon the head may also hasten, or even determine, the development of atheroma of the bulbar arteries. Thrombosis of the bulbar arteries, though an infrequent condition, is probably ten times as common as embolism.

Pathology.—The arteries of the medulla are for the most part without anastomoses: their occlusion, therefore, leads to anæmic necrosis and reactionary inflammation of the bulbar substance, the extent of the softening being dependent on the size and number of the vessels involved. The arteries most often implicated by thrombosis or by embolism are the vertebrals, the left vertebral being oftener occluded than the right. As a rule, only one artery is plugged, and the result of such occlusion seems to be a spot of softening in the posterior part of the medulla of variable size, commonly extending somewhat across the median line. The individual branches of the vertebral are sometimes the seat of thrombosis, but usually the seat of embolism, owing to the angle at which they arise. Thrombosis of the basilar is not very uncommon, and when the clot extends from the vertebral there is softening of both medulla and pons—not an unusual condition. Sometimes the region of softening extends from the upper part of the pons to the lower part of the medulla, and takes in a large part of the transverse extent of the parts. On the other hand, there may be a single small spot of softening or a number of small spots. Very seldom no changes in the vessels can be found to account for the softening. Such cases are probably instances of poliomyelitis bulbi, and are doubtless of bacterial origin.

Symptoms.—As might be expected, the symptoms of occlusion of the bulbar arteries vary considerably according to the situation and extent of the lesion or lesions.

In a considerable proportion of cases, perhaps the majority, the onset of the symptoms is accompanied with sudden or rapid loss of consciousness. In this condition death may occur in the course of a few seconds or minutes, and in such cases the lesion has usually been large and has implicated the pons (Hoyer). In the cases where apoplexy has been absent the onset has generally been attended by vertigo, vomiting, headache, drowsiness, etc., after which the signs of bulbar paralysis have made their appearance. Weakness and disturbances of sensibility in the limbs, usually on both sides, are apt to attend the onset. These symptoms may entirely pass away in a few hours or days, leaving behind them only the distinctively medullary symptoms, or they may continue and form a permanent feature of the condition.

Chief among the medullary symptoms that result from occlusion of bulbar arteries are difficulty or loss of articulation, dysphagia, rigidity of the jaw muscles (perhaps followed by weakness), and laryngeal paralysis (uni- or bilateral), with resulting aphonia and hoarseness. Crossed hemianæsthesia, crossed tongue, and body paralysis, and unsteadiness in walking or falling to one side (from implication of the restiform body), are important but less common bulbar symptoms which possess a localizing value. The motor symptoms referable to implication of the general motor path differ much in different cases. The

paralysis may involve all four extremities: there may be merely paraplegia of the legs, there may be hemiplegia, especially on the side opposite the greatest bulbar involvement or most marked on the same side, or there may be paralysis in one arm and in both legs. Sometimes the motor weakness shifts from one side to the other. The muscles are usually rigid and the reflexes heightened. Paræsthesiæ are common; sometimes pain is a conspicuous symptom, especially in the arms (Oppenheim). Rarely there is typical hemianæsthesia below the neck. There may be some wasting of the tongue or face, but this is rare, for the corresponding tracts are implicated, as a rule, between the cortex and the nuclei (supranuclear paralysis). On the other hand, in acute polioencephalitis of the medulla the nuclei are implicated and wasting occurs. The mental state is clear, but, owing to the emotional disturbance which is usually present and the difficulty in articulation, the patient is apt to be thought weak-minded. When one vertebral is occluded there is usually one-sided sensory and motor defect, and perhaps hyperæsthesia in one or both legs, but the effects of such occlusion vary widely according to the variations in the extent of territory supplied by one vertebral. It occasionally happens that an acute lesion (either occlusion or hemorrhage) causes symptoms which are highly symmetrical, and which correspond clearly to those due to degeneration of the bulbar nuclei. Sudden deafness and vertigo have been known to arise from occlusion of the upper lateral branches of the basilar artery.

Diagnosis.—Occlusion of the bulbar arteries, and consequent softening, are liable to be confounded with two conditions especially—hemorrhage into the medulla and acute inflammation of the bulbar nuclei. The distinction from hemorrhage, usually difficult, sometimes impossible, has been elsewhere considered. Acute inflammatory bulbar paralysis (poliomyelitis) is of more gradual onset than the symptoms of occlusion, coming on as it does acutely in the course of a few days. In distribution the paralysis usually corresponds closely with that observed in degeneration of the bulbar nuclei; and in some cases of hemorrhage or softening it is more or less symmetrical and involves face, palate, and tongue, sometimes also pharynx and larynx. The diagnosis of occlusion from embolism is based on the occurrence of the symptoms already mentioned in a patient with acute or subacute endocarditis (especially in a patient under fifteen). Occasionally the diagnosis is further strengthened by the occurrence of embolism elsewhere, as in the spleen (suggested by pain and swelling) or retina (sudden blindness).

Prognosis.—In all cases of thrombosis or embolism of the bulbar arteries in which the onset is attended with apoplexy there is much danger to life for a time, especially the danger of respiratory and cardiac failure. Another source of danger during the first days or weeks is pneumonia from the entrance of food into the trachea or bronchi. Only a minority of the cases, however, die in the acute stage, and most of these are cases of bilateral vertebral thrombosis or of basilar thrombosis, which are almost invariably fatal. The majority of cases live through the acute stage and undergo gradual improvement. In a few cases the bulbar symptoms wear away altogether. Even cases with widespread paralysis (as of all four extremities) sometimes recover to a considerable extent. In many cases a condition like that of chronic

bulbar paralysis (with the important exception of being regressive in tendency and not progressive) remains over. In general, if after a month there is little improvement and paralysis remains considerable, there is little likelihood of material retrogression in the symptoms. Second attacks of similar nature sometimes occur.

TREATMENT.—Cases should be treated on the principles that apply to thrombosis and embolism elsewhere. When the symptoms depend on thrombosis from syphilitic endarteritis, energetic antisyphilitic treatment is indicated, and may be safely employed in doubtful cases in which the patient is under sixty and embolism can be excluded. During the acute stage the heart must be stimulated by means of strychnine, digitalis, and alcohol, according to the conditions, and prolonged artificial respiration may be necessary. The bowels should be kept open, but there should be no violent purging. The patient should lie flat. It is necessary to see that the patient does not die of inanition, and it may be necessary to feed through a tube. The increased susceptibility to lobar pneumonia must be kept in mind.

HEMORRHAGE INTO THE MEDULLA; HEMORRHAGE INTO THE PONS.

DEFINITION.—Extravasation into the substance of the medulla from rupture either of a bulbar vessel or of a vessel of the pons, with subsequent extension into the medulla. Usually, hemorrhage into the medulla is by extension from the pons, and on this account it is desirable to consider together the bulbar and the pontal extravasations.

ETIOLOGY.—The causes of bulbar and pontal hemorrhage are the causes of cerebral hemorrhage in general. The degenerative period of life, overwork, mental excitement, and sudden exposure to cold are the conditions that favor such hemorrhage. Occasionally multiple punctate hemorrhages into the pons have been caused by severe concussion of the head. The rigidity of catalepsy has been known to determine minute extravasations into the vagus nuclei of the medulla. Hemorrhage limited to the medulla is very rare. Hemorrhage into the pons is uncommon as compared with the frequency of hemorrhage into the internal capsule, and is of about the same actual frequency as hemorrhage into the cerebellum.

PATHOLOGY.—The hemorrhage is commonly from a small artery that has been weakened by atheroma, and in a certain number of cases (probably a minority) the bleeding has followed the symptoms of an aneurysm. Rarely the hemorrhage is capillary, and results from the vascular changes incidental to pernicious anæmia or leucocythæmia. The extravasation is then usually small. The vessels ordinarily involved in the pons are the median branches of the basilar artery, which explains the frequency of hemorrhage near the median line. The hemorrhages are often spheroidal in shape, and, owing to the resistance offered by the raphé, are generally limited to one side. When there is

rupture of a small lateral branch of a median artery the blood may extend transversely. Rupture of a radicular (lateral) branch is rare. When such rupture occurs the branch to the root of the fifth nerve is most often involved. A hemorrhage of considerable size is liable to extend into the medulla, into the middle peduncle, or to burst into the fourth ventricle, according to its situation. When the extravasation is into the medulla it is usually from a median artery, and is very apt to break into the fourth ventricle or into the meninges.

SYMPTOMS.—The majority of cases of hemorrhage into the medulla die in a few seconds or minutes from interference with the respiratory and cardiac nuclei. Usually there is sudden loss of consciousness, more often without than with convulsions. Weakness in the limbs is rare, because the lesion is generally posterior to the pyramidal tract. Sometimes death does not occur for several hours, and very rarely the apoplexy is recovered from and leaves behind it a bulbar paralysis. Very rarely, too, bulbar paralysis of acute (not sudden) onset, *without* loss of consciousness, results from a medullary hemorrhage (Senator). A hemorrhage into the pons is generally attended with initial loss of consciousness. In 48 of 78 cases of partial hemorrhage death occurred within twenty-four hours (Bode), and has been known to occur in seven minutes (Mickle). Convulsions at the onset are usual. They are general and epileptiform as a rule, but may affect the legs only—a very characteristic symptom. Paralysis is generally present, and is usually bilateral, but may be hemiplegic (crossed or common). Anæsthesia may be associated with the paralysis or exist alone. Commonly the pupils are pin-point, occasionally they are dilated. Vomiting is often present; the temperature is apt to be hyperpyrexial, and the respiration is usually stertorous or gasping or Cheyne-Stokes. In non-fatal cases there is either no loss of consciousness or the loss passes away in a few hours. The remaining motor and sensory symptoms differ considerably in different cases (see Cerebral Localization, p. 293). Hemiplegia, crossed or common, is frequent. Rarely there is crossed hemianæsthesia, which is apt to be associated with staggering to one side, owing to irritation of the middle peduncle of the cerebellum. Considerable mental loss is not rare.

DIAGNOSIS.—The diagnosis of bulbar hemorrhage is always uncertain and generally impossible. It may be confounded with almost any one of the causes of sudden death or apoplexy. In cases where the existence of a bulbar paralysis can be made out there is always the possibility of hemorrhage, even if the onset is acute and not sudden, and there has been no loss of consciousness, but the probabilities in such cases are always much in favor of acute softening of the medulla.

The diagnosis of partial hemorrhage in the stage of unconsciousness can only be made when some form of crossed paralysis is detected, but may be suspected in the absence of crossed paralysis if there is great embarrassment of respiration associated with repeated vomiting or pin-point pupils, or hyperpyrexia or convulsive movements limited to the legs. In the post-apoplectic stage the diagnosis is based on the presence of localizing symptoms, some of which have been mentioned in the preceding section (see also Cerebral Localization, p. 293).

PROGNOSIS.—The desperate nature of the prognosis in bulbar hemor-

rhage is evident from what has been said. Recoveries from bulbar hemorrhage are extremely rare, but do occur, especially where loss of consciousness is absent or lasts only a few minutes or hours. Probably four fifths of all partial hemorrhages lead to death within two days. If the initial loss of consciousness lasts more than six hours, the result is almost invariably fatal; if it lasts only a few minutes, recovery is probable, although a second large hemorrhage may succeed the first. The outlook is also very bad where the apoplexy is ingravescent. If there is recovery from a first seizure, there is always the possibility of recurrence, and the likelihood of this, which cannot be estimated, is always increased if the patient is unable to take the proper precautionary measures.

TREATMENT.—The problem of treating bulbar hemorrhage is one which very seldom arises. When it does, the treatment, like that of pontal hemorrhage, consists in quieting an overacting heart or of stimulating the flagging cardiac and respiratory functions by means of strychnine, alcohol, and digitalis. In pontal hemorrhage the respiration must be carefully watched, and artificial respiration may be necessary during an entire day. Where recovery has taken place recurrence is to be guarded against by means of rest, quiet, and the avoidance of mental excitement and alcoholic stimulation.

DISEASES OF THE BRAIN.

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By J. T. ESKRIDGE, M. D.

Cerebral Localization.

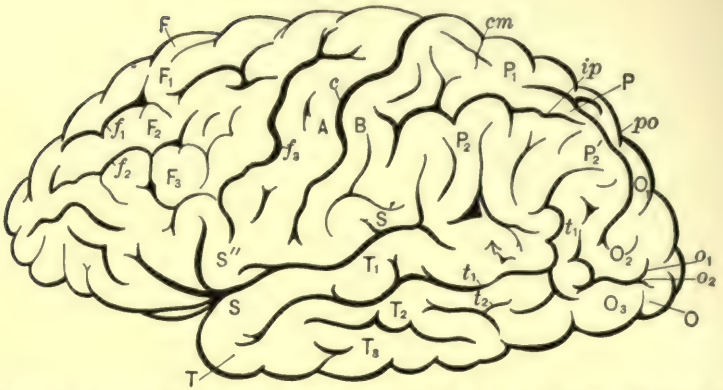
Experimental investigation and clinico-pathological observation, as well as the structural organization of the brain, all point conclusively to certain portions of the encephalic mass being endowed with special functions. While the study of limited portions of the brain has led to important results, we should ever bear in mind that the entire mass is capable of acting as a whole, and probably does so in the performance of its higher or psychologic functions. It is probable that all the functions of the human organism have their highest representatives in the cerebral cortex, and there may be a specialized centre for the immediate control of each function ; but every portion of the brain is so intimately connected, either directly or indirectly, with other portions that the action of the secondary or relative centres so mask the functional activity of the primary ones as to obscure their individuality, except for acts that are so highly specialized as to become partially reflex or automatic in their character. At all events, while our knowledge of limited portions of the brain is becoming each year more definite, but little progress has been made in determining the functions of the unknown or "latent regions."

A thorough knowledge of the gross and microscopic anatomy of the lobes, fissures, and convolutions is necessary to an intelligent and comprehensive study of the localizable functions of the cerebral cortex, but the space into which this article must be compressed is entirely inadequate for a discussion of this subject. For a detailed account of the description of the cerebral cortex the reader is referred to works on anatomy.[1] The lobes, fissures, and convolutions of the lateral, median, and basilar surfaces of the cerebrum are shown in Figs. 31–33, except a small lobe, the island of Reil, or insula, which is covered by the operculum. The latter is formed by the extension downward and junction of the ascending frontal and ascending parietal convolutions [2] below

[1] In *A Text-Book on Nervous Diseases by American Authors* will be found a carefully prepared article on the gross and minute anatomy of the cerebral cortex by C. K. Mills, in connection with the subject of cerebral localization.

[2] These are also known as the anterior and posterior central convolutions.

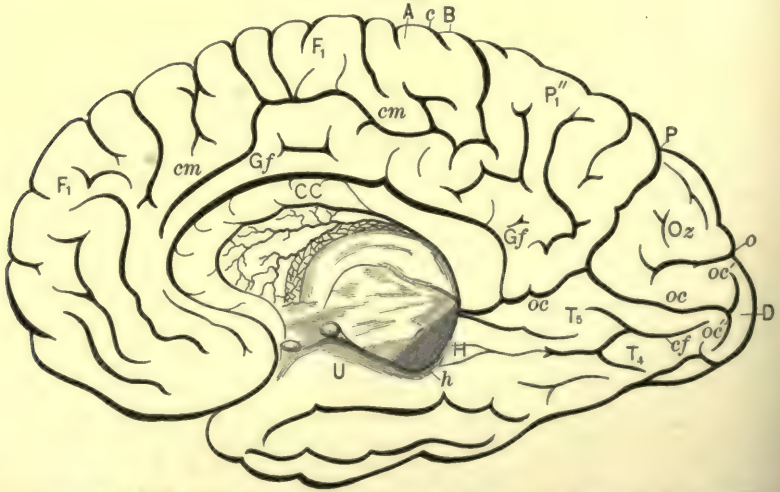
FIG. 31.



Lateral aspect of the left hemicerebrum: F, frontal lobe; P, parietal lobe; O, occipital lobe; T, temporo-sphenoidal lobe; S, fissure of Sylvius; S', horizontal; S'', ascending ramus of the same; c, sulcus centralis or fissure of Rolando; A, anterior central or ascending frontal convolution; B, posterior central or ascending parietal convolution; F_1, superior, F_2, middle, and F_3, inferior frontal convolutions; f_1, superior, and f_2, inferior frontal sulcus; f_3, sulcus præcentralis; P_1, superior parietal or postero-parietal lobule; P_2, inferior parietal lobule—viz. P_3, gyrus supramarginalis; P'_2, gyrus angularis; ip, sulcus intraparietalis; cm, termination of the calloso-marginal fissure; O_1, first, O_2, second, O_3, third occipital convolutions; po, parieto-occipital fissure; o_2, sulcus occipitalis transversus; o_2, sulcus occipitalis longitudinalis inferior; T_1, first, T_2, second, T_3, temporo-sphenoidal convolutions; t_1, first, t_2, second, temporo-sphenoidal fissures (lettering according to Ecker).

the lower end of the fissures of Rolando. In raising the operculum the main stem of the fissure of Sylvius is exposed, in which lies the

FIG. 32.



Median surface of the right hemicerebrum: CC, corpus callosum cut through the centre; Gf, gyrus fornicatus; H, gyrus hippocampi; h, sulcus hippocampi; U, uncinate gyrus; cm, sulcus calloso-marginalis; F_1, median surface of the first frontal convolution; c, terminal portion of the sulcus centralis fissure of Rolando; A, ascending frontal; B, ascending parietal convolution; P_3, precuneus; O_2, cuneus; Po, parieto-occipital fissure; o, sulcus-occipitalis transversus; oc, calcarine fissure; oc', superior; oc'', inferior ramus of the same; D, gyrus descendens; T_4, gyrus occipito-temporalis lateralis (lobulus fusiformis); T_5, gyrus occipito-temporalis medialis (lobulus lingualis); cf, collateral or occipito-temporal fissure.

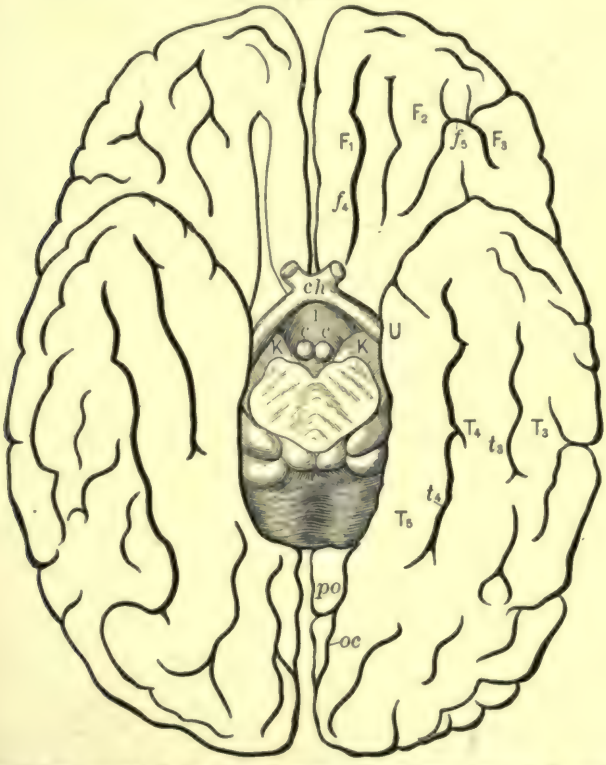
island of Reil, itself composed of several small convolutions. The parieto-occipital fissure which forms the anterior boundary of the occip-

ital lobe extends but a slight distance on the lateral surface of the brain, so that the convolutions of the temporal and parietal regions are apparently continuous with those of the occipital lobe.

Cortical Localization.

Few of the lesions produced by disease are strictly limited to the cortex, and those of an experimental character often involve the subcortical region. Even when electricity is employed to stimulate the

Fig. 33.



View of the brain from below: F_1, first frontal convolution or gyrus rectus; F_2, middle or second frontal convolution; F_3, inferior or third frontal convolution; f_4, sulcus olfactorius; f_5, sulcus orbitalis; T_3, third or inferior temporo-sphenoidal convolution; T_4, gyrus occipito-temporalis lateralis (lobulus fusiformis); T_5, gyrus occipito-temporalis medialis (lobulus lingualis); t_4, sulcus occipito-temporalis inferior; t_3, sulcus temporo-sphenoidalis inferior or third temporal fissure; *po*, parieto-occipital fissure; *oc*, calcarine fissure; *U*, gyrus uncinatus; *ch*, optic chiasma; *cc*, corpora albicantia; *KK*, crura cerebri (lettering according to Ecker).

cortex, it is impossible to limit the effects of the current to the gray matter. If greater care were used in trying to distinguish between the results of purely cortical irritation and destruction, and the phenomena that follow when the cortex and white substance immediately below are simultaneously involved, it is possible that we should soon be in possession of more accurate and reliable information regarding the function of the cerebral cortex.

Prefrontal Cortical Region.—All that portion of the cortex in front

of a line drawn on the scalp, 1 inch to 1¼ inches anterior to the fissure of Rolando and parallel to this fissure, may be designated the prefrontal cortical region. This is classed among the so-called latent regions of the brain, because its functions are not well known, and from the fact that lesions in it are often attended by indefinite symptoms. The conclusions of Bianchi[1] in an admirable paper in which he reviews the different theories in regard to the functions of the frontal lobe, and gives detailed results of his own carefully conducted experiments on twelve monkeys and six dogs, may be briefly summed up thus: Upon the integrity of these lobes depends the co-ordinated psychical personality of the animal, and their destruction is followed by the loss of that psychical tone common to the animal or individual. There are no known centres in the frontal lobes in which special functions are localizable, yet, somehow, in this portion of the brain are fused and co-ordinated the psychical results of all sensory motor impressions. The animal, the frontal lobes of which have been mutilated, ceases to show appreciation of friendship or kindness, and becomes unsociable, timid, sexually perverted, and filthy. The frontal lobes are bilateral in their action in man, and a non-irritative, slowly-developed lesion may almost completely destroy the function of one lobe without giving rise to appreciable symptoms. Sudden acute or irritative lesions, sufficiently extensive to interfere with function, cause a train of mental symptoms characterized by weakened power of concentrating attention, slowness of perception and appreciation, lessened will, judgment, and reason. Complex intellectual processes are no longer possible, and sustained attention is equally difficult. Not infrequently the individual becomes irritable, suspicious, and impatient; the moral tône may be weakened and ordinary neatness is often neglected. Inco-ordinate movements similar to those observed in lesions of the cerebellum have been observed in disease of the frontal lobes, but these are usually met with in certain cases of tumor of this region. (See Tumors of the Frontal Lobes, p. 468.)

Motor Localization.

It need not concern us here whether those portions of the cortex which are commonly known as motor are purely motor in their function, as Ferrier, Mills, and a number of others still maintain, are motor and sensory, as argued by a host of able observers, or are purely kinæsthetic, as first advocated by Bastian. The fact still remains for practical purposes—and the points discussed in this article will be limited to those of a practical character—that irritation or destruction of the cortical substance over certain areas of the brain will usually result in the manifestation of motor phenomena. It is probable that there is no cortical centre for a single muscle, except possibly for the diaphragm, and as yet none has been isolated for this, to my knowledge. Cortical centres seem to preside over the action of groups of muscles that act in association for the performance of definite movements. The "absolute" centres of Exner, those the irritation or destruction of which rarely fails to produce motor disturbance, are probably comparatively well defined and limited, and the "relative," those the irritation or destruction of which

[1] Bianchi: *Brain*, part lxxii., Winter, 1895, p. 497.

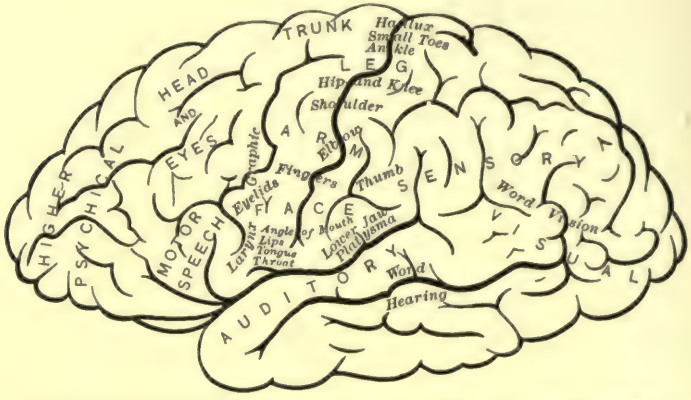
frequently, but by no means constantly, results in motor phenomena, are illy defined and extend over a greater area. While the "absolute" centres may not overlap each other to a great extent—but this is by no means certain—it is quite evident that the "relative" ones do. From the fact that centres do overlap each other the difficulties surrounding accurate cortical localization are greatly enhanced. Irritation of a cortical motor centre will give rise to spasm, and destruction to paralysis, in definite groups of muscles on the opposite side of the body; but it must be remembered, in the application of the principles of cerebral localization to the study of the seat of focal lesions of the brain, that it is not necessary that the morbid process must occur within the centre, and thus irritate and destroy it, to cause spasm or paralysis in a group of muscles. A lesion occurring some distance from a centre may affect it in various indirect ways—by pressure, by irritative inhibition, or by vascular disturbance. Sufficient time must be allowed to elapse after the development of a lesion to get its permanent effects upon a centre. Cases are almost valueless for the study of localization unless every necessary precaution to prevent erroneous conclusions has been employed. Sudden or acute extensive morbid processes do not occur in the motor centres without giving rise to localizing symptoms, provided the general cerebral functions are not so profoundly disturbed by shock that their manifestation is impossible; but chronic lesions, such as tumors or abscesses, may in rare instances develop in such centres without being attended by distinct symptoms of their seat. Such occurrences prove nothing beyond the fact that nerve tissue is much more tolerant of slowly developing lesions than of those that take them by storm. It is probable that in the former case many of the nerve fibres have been displaced rather than destroyed. Broadbent was the first to direct attention to the fact that muscles that act in association with similar muscles of the opposite side are controlled to a greater or less extent by the centres in both hemispheres, and the unilaterally acting muscles are largely represented by cortical centres in one hemisphere. The nearer the distal portion of the extremities the more highly specialized and the more unilateral in their actions are the muscles. The hand muscles are more unilateral in their action, because they are better educated to specialized movements than those of the foot. The muscles concerned in turning the eyes in different directions are probably the purest type of bilaterally acting muscles, but those of mastication, deglutition, the trunk, and many others belong to this class. The more highly specialized a group of muscles in their unilateral action, the more complete and permanent the paralysis from a unilateral cortical lesion, and the more intimately muscles are associated in action, with corresponding ones on the opposite side of the body, the less complete and the more transient the paralysis from a unilateral cerebral lesion.

Figs. 34, 35 give the probable motor area of the cortex. It includes on the lateral surface of the brain the two central convolutions, *A. F.*,[1] *A. P.*,[2] the base of the superior frontal and probably the posterior extremities of the second and third frontal convolutions. Some doubt that the second and third frontal convolutions are concerned in muscular movements in man, although it seems to be admitted that they are in the

[1] Ascending frontal. [2] Ascending parietal.

monkey. On the median surface of the hemisphere the paracentral lobule and the posterior portion of the marginal convolution, as first demonstrated by Horsley and Schäfer, are part of the motor area. On the median aspect of the hemisphere the following parts, in the order

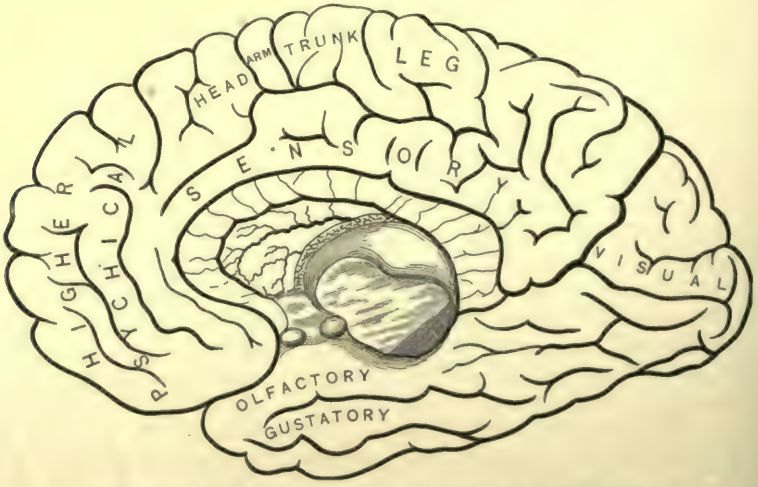
FIG. 34.



Lateral aspect of the left hemicerebrum, showing the cortical centres.

named, are represented from before backward: head, hand, arm, trunk, leg, and toes. On the lateral surface the leg is represented in the upper portion of the central convolutions, probably in about the upper fifth. The arm and hand occupy the middle two fifths, and the face the lower

FIG. 35.



Median aspect of the right hemicerebrum, showing cortical centres.

two fifths of these convolutions.[1] The arm centres extend higher on the ascending frontal convolution, reaching nearly to the longitudinal fissure, than on the ascending parietal.

[1] Mills: *A Text-Book on Nervous Diseases by American Authors*, p. 403.

Cortical Area for Leg Movements.—These centres seem to be limited to a narrow strip on the lateral and median surfaces of the hemisphere, extending anteriorly on the lateral surface into the foot of the superior frontal convolution, and posteriorly to the retrocentral fissure. As already stated, it does not occupy more than the upper one fifth of the Rolandic area. The centres for the hip and knee are forward and below those for the ankle and toes, those for the latter being the highest and most caudad of the cortical motor centres on the lateral surface. At the junction of the superior and ascending frontal convolutions there seems to be a centre for movements of the foot. The hallux or great toe is represented by a centre just in front of the highest part of the fissure of Rolando. The leg centre on the median surface is apparently confined to the paracentral lobule, with the parts above the knee represented forward of those below it. Only a few lesions limited to the leg centres have been recorded in man. Horsley[1] has reported a case of traumatic epilepsy in which the initial convulsive movements began in the great toe. On the removal of the lesion, a dense and cystic cicatrix, from the upper end of the ascending frontal convolution the fits ceased. In the same paper the author refers to another case, a tumor of the brain weighing four ounces, in the removal of which the cortex immediately in front of the upper extremity of the fissure of Rolando was excised. The only permanent paralysis following the operation was limited to the great toe. Mills[2] reports a case of gumma, involving the upper fourth of the precentral and a small segment of the postcentral convolutions, in which the patient had convulsions beginning with twitchings in the toe and foot. In a case recently observed by me the entire right leg was paralyzed, and ankle clonus was present in the left. A tumor involving the leg centre on the left lateral surface, but extending to the median surface and irritating the median surface of the leg area of the opposite hemisphere, was diagnosticated. E. J. A. Rogers successfully removed the growth, which proved to be a gumma. Centres for movements of the trunk muscles occupy a larger surface on the median than on the lateral surface of the brain, and are just forward of the area in which the centres for the leg are found.

Cortical Area for Arm Movements.—This area occupies the middle two fifths of the Rolandic region, spreading over both central convolutions, but extending higher in the ascending frontal than in the ascending parietal. A lesion in the upper portion of the ascending frontal convolution near the longitudinal fissure, especially if it extends forward, may paralyze the arm. Fig. 34 shows the relative positions of the subdivisions of the arm area. The shoulder is highest and forward, the elbow next below and behind, then come the centres for the wrist and fingers in the order named, in front, and the thumb centre is the lowest and behind. The carefully conducted experiments of Beevor and Horsley on the brains of monkeys, and numerous observations made by a host of neurologists by means of the faradic current on the human brain, leave little doubt concerning the accuracy of the subdivisions of the arm area as here given. Mills, Keen, and numerous others have made careful observations tending to establish the relative positions of

[1] *American Journal of the Medical Sciences*, April, 1887, p. 366.
[2] *A Text-Book on Nervous Diseases by American Authors*, p. 413.

these centres. During the past two years I have stimulated the area in question on eight different occasions in the human subject by means of the Keen double electrode, attached to a Flemming faradic battery. I will only refer to one of these cases, as the results were substantially the same in all: On the removal of a button of bone over what was apparently the middle portion of the fissure of Rolando, the electrode was applied posteriorly and the thumb flexed, then the index finger, followed a second or two later by flexion of the other fingers of the hand. By a slight movement of the electrode upward wrist movements occurred first, soon the arm flexed at the elbow, then the arm was moved at the shoulder and drawn across the chest.

Cortical Area for Face Movements.—The area for the face occupies the lower two fifths of the cortical motor area. On account of the bilateral action of most of these muscles the sub-areas have not been as well defined as those of the arm and hand. The lower portion of the ascending frontal convolution is more concerned in face movements than the corresponding portion of the ascending parietal. Gowers[1] thinks that it is probable that the face centres extend to the ascending parietal convolution, but says that it is not yet proved. The sub-area for the upper portion of the face occupies the highest portion of the face area, and is probably situated anterior to the fissure of Rolando, mainly, if not entirely, in the ascending frontal convolution. On two occasions, while I was endeavoring to define the face area in man, the platysma contracted, with depression of the lower jaw, when the electrodes were applied below and posterior to the lower end of the fissure of Rolando. On a repetition of the experiment in November, 1895, in a case of epilepsy in which I desired to have the face area removed, the platysma and pterygoid muscles contracted and depression and lateral movements of the lower jaw occurred. Horsley has obtained the same movements on the human subject from stimulating the ascending frontal convolution at the junction of the middle and upper thirds of the area for the face. There are at least two sources of fallacy in these experiments: When the button of bone removed is small, as it was in my own case, it is difficult, and sometimes impossible, to be certain of the exact area excited; secondly, if the electric current is strong, adjacent centres as well as immediate ones are excited into activity. In a case of convulsions beginning in the platysma the source of irritation was found to be a spicula of bone in the lowest portion of the ascending parietal convolution.[2] A case of twitching of the left angle of the mouth, due to a spasm of the zygomatic muscles for a period of thirty months, was observed by Berkley. At the autopsy a nodule of calcareous degeneration, half as thick as the cortex, was found on the surface of the right anterior central convolution in its lower third.[3] The centre for the lips and tongue is placed by Mills in the lowest portion of the ascending frontal convolution, posterior to the sub-area for the larynx. It probably extends to the third frontal convolution, as suggested by Gowers. Nothnagel places the tongue centre at the junction of the third frontal and anterior central convolutions. Hirt[4] believes that the

[1] Gowers: *Diseases of the Nervous System*, vol. ii. p. 18.
[2] Byron Bramwell: *Brit. Med. Journ.*, Aug. 28, 1875.
[3] *Med. News*, July 15, 1882. [4] Hirt: *Diseases of the Nervous System*, p. 55.

cortical motor area of the trigeminus is situated in the lower third of the ascending frontal and adjoining portions of the second and third frontal convolutions. The clinical observations of Seguin and Gorel and the experimental investigations of Krause, Semon and Horsley, and others seem to establish the existence of a laryngeal centre in the posterior portion of the third frontal and adjacent portion of the anterior part of the ascending frontal convolutions.

Cortical Area for the Movements of the Head and Eyes.—While the centres for these movements have been pretty definitely located in the monkey by Ferrier, Horsley, and others, it is by no means certain that they occupy the same position (the posterior portion of the second and third frontal convolutions) in man. It is probable, however, that conjugate deviation of the head and eyes to the opposite side occurs from irritation of the frontal lobe immediately in front, cephalad, of the prefrontal fissure, and that this centre is most highly differentiated in parts adjacent to the horizontal branch of this fissure. In the monkey elevation of the upper eyelids is caused by exciting the cortex at the foot of the second frontal convolution and also in the region of the angular gyrus. As the latter area is connected with sight, and the upper lids are raised in intent vision, the movement may be reflex from the stimulation of a sensory centre, as Ferrier contends. The observations of Landouzy, Grasset, Rendu, and others, who have reported ptosis from isolated lesions in the lower parietal lobule, are offset by the experience of a host of observers who have carefully studied numerous cases with lesions in the same region without finding any affection of the muscle that elevates the upper lid. As the cortical and ocular motor centres are usually bilateral in their action, it is probable that persistent oculomotor paralysis is due to a nuclear or a peripheral lesion, and not to a cortical one.

Sensory Localization.

The weight of evidence, experimental, clinical, and pathological, seems to point to less definitely localizable and more extensive areas in the cerebral cortex for the general sensory functions than for the motor. Those who contend that the central convolutions are purely motor and do not directly preside over sensation have well-attested observations to support them in their position, but the cases with lesions in the cortical motor area attended by sensory disturbances are far more numerous than those in which the interference has been limited to motility. From careful experimental investigation and numerous clinico-pathological observations Victor Horsley was led to believe that the motor area of the cortex is endowed with slight tactile sense, a degree of muscular sense, and great motor representation.[1] In an unreported case of mine in which Clayton Parkhill removed the hand center for the relief of spasm beginning in the hand and usually limited to the muscles of the hand and arm, the resulting paralysis was attended by nearly absolute anæsthesia. In another case,[2] in which E. J. A. Rogers removed for me the cortical center of the right hand for the relief of epilepsy of eighteen years' duration, the paralysis, which was nearly complete in the hand,

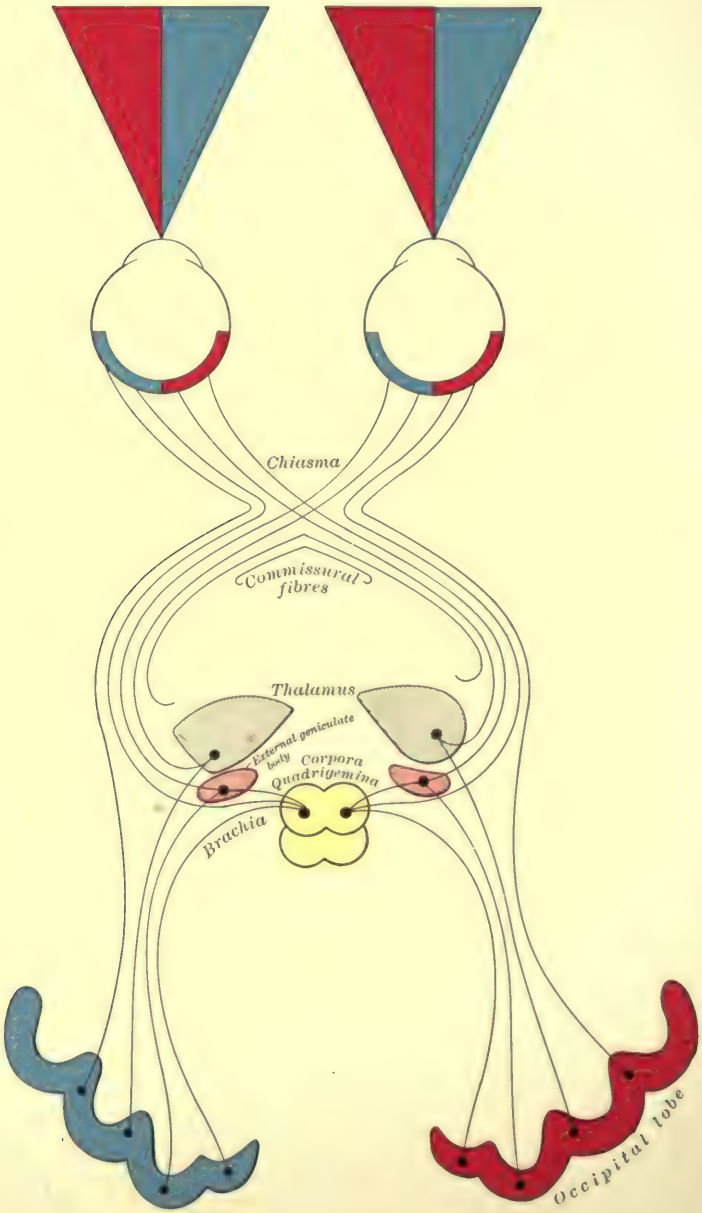
[1] *Trans. Cong. Amer. Phys. and Surg.*, 1888, p. 341. [2] *Med. News*, Oct. 13, 1894.

was not attended by any sensory disturbance until the sixth day after the operation, and then it was limited to the tips of the ring and little fingers, and only lasted two days, notwithstanding that the hand continued paralyzed for several months. In this case great care was observed in testing tactile, localization, posture, pain, and temperature sensations. A feather or camel's-hair pencil, as well as an anæsthesiometer, was used in testing tactile sense. In a third case, in which E. J. A. Rogers recently removed for me the right face centre, no sensory disturbance followed, although the lower side of the face was partially paralyzed. With reference to the cortical motor area in its relation to sensibility, it may be said to be intimately associated with tactile sense, especially in its higher and more differentiated forms, and probably also to a slight degree with muscular sense. Injury of the central convolutions is more likely to be attended with sensory disturbance when the principal lesion is caudad to the fissure of Rolando than when it is cephalad of this fissure. While within the central convolutions the existence of motor centres for different portions of the body has been demonstrated, these also seem to be the seat to some extent for cutaneous sensory centres, so that muscular spasms or paralysis from a cortical lesion will be attended by subjective sensory disturbance or partial anæsthesia of the skin covering the affected muscles. Still, we have to admit that lesions of the central convolutions in which no apparent sensory disturbance follows are too numerous to enable us to say positively in what way the cortical motor centres are associated with sensation. Sudden and complete destruction of areas in the central convolutions does not cause absolute anæsthesia in the paralyzed parts, such as follows a lesion in the posterior portion of the interior capsule; therefore other portions of the cerebral cortex than the motor must possess sensory function. Numerous observations apparently establish the fact that in the cortex of the parietal region, more probably in the supramarginal convolution, the sensory centres in part are represented. The clinical records are few in which a limited lesion on the median surface of the brain in the region of the limbic lobe has been attended with anæsthesia on the opposite side of the body. The experimental investigations of Ferrier, Schäfer, and Horsley seem to justify them in attributing a sensory function to this lobe, but that it is the predominant centre for general sensation, as Ferrier still contends, cannot be accepted. Charles K. Mills,[1] who still maintains that the central convolutions are for motor representation only, and that separate sensory localization is in the gyrus fornicatus and hypocampal region, lays considerable stress on the clinico-pathological observations of Savill in confirming the doctrine of separate sensory localization. One of the conclusions arrived at by a study of the first case reported by Savill is that the gyrus fornicatus is the centre for common sensation on the opposite side of the body. The second case points to the posterior portion of the gyrus fornicatus as the cortical centre for tactile sensation of the arm.[2] It seems to me, until further proof justifies a change of opinion, that we must assign general sensory function, to a considerable extent of the cerebral cortex, to the

[1] *A Text-book on Nervous Diseases by American Authors*, p. 419.

[2] These cases, from which Mills quotes at length, are reported in full in *Brain*, vol. xiv., 1891, p. 274, and vol. xv., 1892, p. 448.

PLATE IV.



Schematic Representation of the Optic Visual Centres, Optic Radiations, Basial Visual Ganglia, Optic Tracts, Nerves, Retinæ and Fields.

central convolutions, especially the posterior—to the parietal region, to the gyrus fornicatus, and to the hippocampal region. Muscular sense may be situated in the portions of the cerebral cortex in which common cutaneous sensibility is located, although several observations point to a separate centre for this in the parietal lobe, and Charles L. Dana places it in the inferior parietal lobule.[1] An observation made by Starr and McCosh points to a separate centre for the memory of muscular movements in the parietal lobe.[2] In commenting upon this case they say : " It was evident that the effect of the operation upon a spot in the brain about at the junction of the superior and inferior parietal convolutions, clearly posterior to the posterior central convolution, had resulted in a loss of muscular sense in the opposite hand and forearm, without any disturbance of other sensations or of the power of movement."

Olfactory Localization.

Experimental investigation on the lower animals and clinico-pathological observations on man connect the median surface of the uncinate gyrus with the olfactory bulb of the same side.[3] That this is not the only centre for smell in the brain is proved from the fact that a lesion of the posterior or sensory portion of the internal capsule, abolishing most of the other special senses on the opposite side of the body, often similarly affects smell.[4] So that while the uncinate gyrus is connected with the sense of smell on the same side, there must be a centre in the opposite hemisphere for each olfactory bulb.

Gustatory Localization.

The fourth temporo-sphenoidal convolution is supposed by some to contain the cerebral centre for taste, but experimental evidence and clinico-pathological observations are too meagre to justify such a conclusion. It seems better to admit that we know almost nothing definite of the cortical centre for taste.

Visual Localization.

Plate IV. shows the cortical visual centre and the course of the fibres from the retinæ to this centre. Clinical and pathological evidences justify the conclusion that the principal, and probably the only, cortical centre for sight, in man at least, is in the occipital lobe. Further, the visual fibres from one occipital lobe go to the like-named sides of each retina, those from the right occipital lobe supplying the right half of each retina, and *vice versâ*. A lesion, therefore, in one occipital lobe destroying the centre of sight gives rise to bilateral homonymous hemianopsia, the blind fields being on the side of each eye opposite to that of the brain lesion. The exact location in the occipital lobe of the cortical visual centre cannot be said to be entirely settled, although most

[1] Dana : *Text-book on Nervous Diseases*, p. 321.
[2] *Amer. Journ. of the Med. Sci.*, Nov., 1894, p. 517.
[3] Ferrier, A. McLane Hamilton : *N. Y. Med. Journ.*, June, 1892 ; Hughlings-Jackson and Beevor : *Brain*, vol. xii. p. 304, quoted by Gowers.
[4] Gowers and the writers quoted by him, and several personal observations.

observers are agreed that it is on the median surface in the cuneus.[1] Henschen[2] contends that it is limited to the parts immediately surrounding the calcarine fissure. Hemianopsia has followed lesions in the white substance in the apical region and on the lateral and ventral aspects of the occipital lobe, but it is probable that a lesion not too limited in any of these situations may involve the visual fibres on their way to the cuneus. Henschen[3] in an analysis of about 160 cases of hemianopsia gives 11 in which the lesions were limited to the lateral surface of the occipital lobe, and there existed no defect of vision or only a transitory one in all of them. He reports a number of cases with lesions limited to the lateral or ventral surface without hemianopsia. He gives 25 cases (all that he could find in literature) with lesions limited to the median surface, and of these he remarks: "A methodical analysis of them leads to the following results: Firstly, a lesion on the median surface causes hemianopsia only if the cortex of the calcarine fissure or the fibres derived from it are affected. Secondly, a lesion limited to the calcarine cortex can induce complete hemianopsia." The truth of the latter statement he believes a case published in his book[4] fully substantiates. The lesion was stationary and uncomplicated, and a careful clinical study was crowned by an accurate post-mortem examination. The conclusions of Henschen were not hastily reached, but are the results of a most careful analysis of 160 cases of hemianopsia. Much credit is due Starr[5] and Seguin[6] in this country for their work in collecting and analyzing similar cases. Henschen's researches led him to the conclusion that the fibres from the dorsal retinal quadrant lie dorsally both in the frontal and occipital visual path, and he refers to Hun's interesting case in which the same arrangement was found to exist in the calcarine cortex, and adds: "Thus the upper lip represents the upper retinal quadrant, and other cases strengthen this opinion." In Hun's[7] case the defect in the fields of vision occurred in the lower left quadrant of each eye, with atrophy of the lower half of the right cuneus. It must be remembered that Henschen places the cortical representation for the lower quadrant for each retina in the lower lip of the calcarine fissure, which is just below the cuneus. In regard to whether the same cells in the calcarine lips represent homologous parts of the retina, or whether one half of each retina is represented by different cells, Henschen has made some interesting observations. He says: "After destruction of both eyes in a patient suffering from leprosy there ensued a complete atrophy of the calcarine cortex, but in a case of destruction of one eye only I found in that situation a number of pigment-changed cells alongside of perfectly normal ones. These facts seem to prove that the elements of both retinal halves are represented in the calcarine cortex by different cells, which lie beside each other."

Of the organization of macular or central vision little is definitely known. Wildbrand believes that the macula lutea is innervated by both

[1] Vialet believes the cortical centre of vision occupies the entire median surface of the occipital lobe (*Les Centres cerebraux de la Vision et l'Appareil*, 1893), quoted by Mills.

[2] *Brain*, vol. xvi., 1893, p. 177. [3] *Ibid.* [4] *Pathologie des Gehirns.*

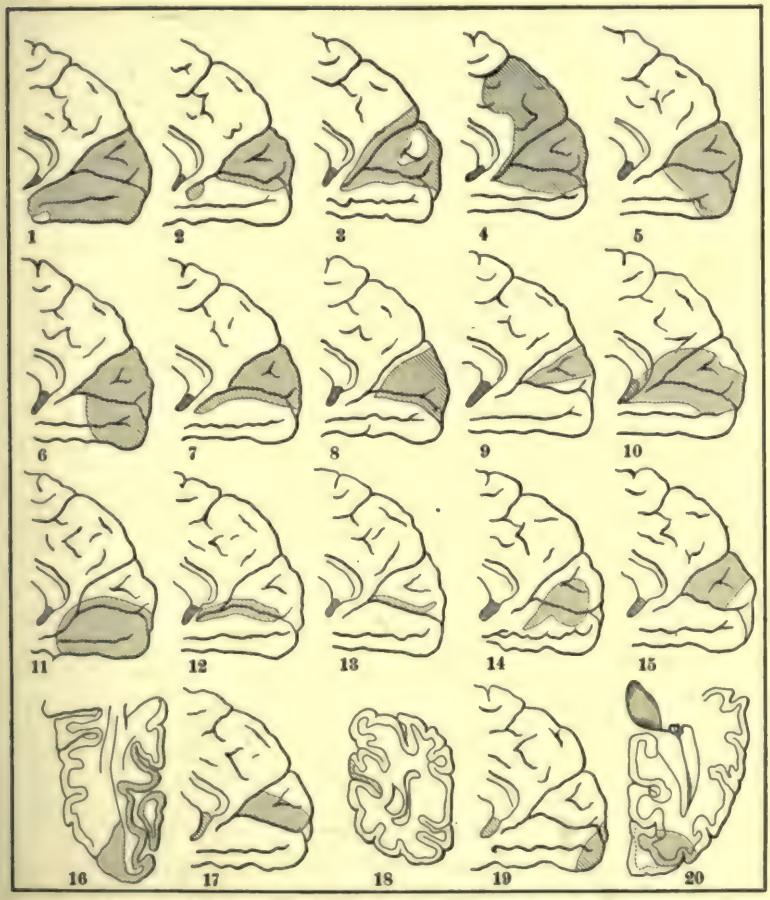
[5] M. Allen Starr: "The Visual Area in the Brain determined by a Study of Hemianopsia," *Amer. Journ. Med. Sci.*, Jan., 1894, p. 65.

[6] E. C. Seguin: *Journ. of Nerv. and Ment. Dis.* vol. xiii., Jan., 1896, pp. 1–38.

[7] Henry Hun: *Amer. Journ. Med. Sci.*, Jan., 1887, p. 140.

hemispheres in a variable manner. Henschen, while he agrees to the bilateral representation in the cortex of each macula lutea, is inclined to locate the centre in the calcarine fissure anterior to the peripheral fields. Charles K. Mills[1] dissents from the latter view, believing the macula is represented in the occipito-angular region on the lateral surface of the

FIG. 36.



Lesions of the occipital lobe with hemianopsia (from Henschen).

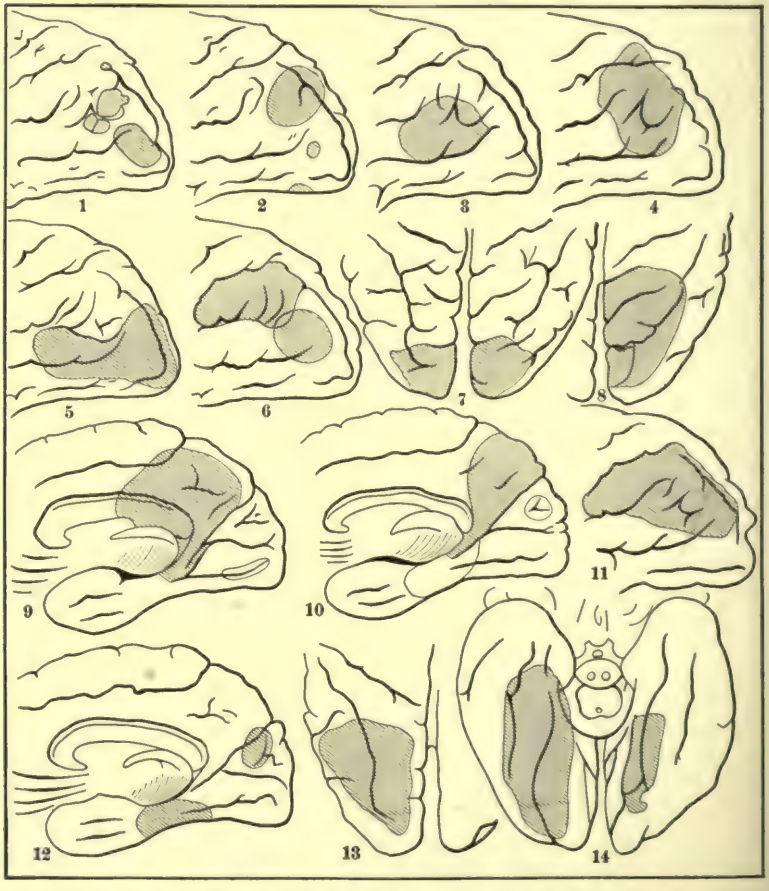
hemisphere, in which are probably stored visual images of words, letters, and probably of objects.[2] Color-perception is undoubtedly a faculty of the cerebral cortex, and probably is situated in the occipital lobe, but as to its exact location we are still in the dark. Henschen, however, contends that some of his cases prove positively that color-perception is situated in the calcarine fissure. Gowers thinks it may possibly be in the anterior portion of the occipital lobe.

Whether the cortical centre for visual recognition, or what is termed

[1] Mills: *A Text-book on Nervous Diseases by American Authors*, p. 422.
[2] *Ibid*, p. 439.

mind or soul vision, is the same as for perception has not been determined. Hun,[1] after referring to a report of a case recorded by Monakow in which visual recognition was lost after the occurrence of a lesion on the left side, giving rise to atrophy of the superior and middle temporal convolution, slight atrophy of the middle and inferior temporal con-

FIG. 37.



Lesions of occipital lobe without hemianopsia (from Henschen).

volutions, and softening of the white matter beneath all of these convolutions, concluded that "on the convex surface of the (left?) occipital lobe take place those actions which are associated with complete visual perception and recognition." In view of the fact that the patient was suffering from a more extensive lesion on the right side than on the left, although the former was of older date than the latter, and the extensive nature of the lesion on the left side, it does not seem to me that such a conclusion is justified.

Both word-blindness and mind-blindness occur from lesions in the

[1] *Amer. Journ. of Med. Sci.*, Jan., 1887, vol. xciii. p. 150.

inferior parietal lobule and angular region, but these symptoms may be the result of the lesion extending to the visual fibres as they go to the occipital lobe. They will be discussed in connection with the mechanism and disturbances of speech.

The optic tract winds around the outer side of the crus on its way to the central ganglia, the pulvinar, the corpora quadrigemina, and external geniculate body. According to Henschen, the fibres from the upper retinal quadrant lie dorsally in this tract. He believes that only those fibres of the tract that go to the external geniculate body are visual, while those connected with the pulvinar and tubercular quadrigemina are reflex. None of the visual fibres, according to this observer, pass through the internal capsule. In this opinion he is supported in one case reported by Mills.[1] The visual path is situated in the lower portion of the optic radiations through the centrum ovale of the parietal and angular region, and there forms a bundle less than a centimetre thick which lies at the level of the second temporal gyrus and second temporal sulcus.

Results of Disease in the Occipital Lobe.—As the only function of the occipital lobe known is that of vision, a lesion in it will only result in disturbance of vision as a localizing symptom, usually causing bilateral homonymous hemianopsia, either for the entire half fields or sections of those of the opposite side. Sometimes an irritative lesion in one occipital lobe will give rise to conjugate deviation of the eyes from the side of the disease.[2] A lesion in the temporal or parietal lobe or in the angular region, if it extends deep enough to affect either directly or indirectly the optic radiations, will cause bilateral homonymous hemianopsia. But such lesions in this situation attended by hemianopsia are also associated with some form of sensory aphasia when they occur on the left side, and on either side some general sensory disturbance will be found on the opposite side of the body if the sensory fibres to the inferior parietal lobe have been affected. In hemianopsia resulting from lesions posterior to the external geniculate body the iris will react normally to light, even when the light is thrown into the eyes from the side of the blind fields only. On the other hand, in hemianopsia from a lesion in the external geniculate body[3] or optic tract, the iris will not respond to light on the side of the blind fields, but will when the rays of light penetrate the eye from the side of the preserved fields, thus giving rise to the hemianopic pupillary reflex of Wernicke. It must be borne in mind that diffuse lesions in or near the basilar ganglia producing hemianopsia, although they may not directly involve the external geniculate body, may give rise to hemianopic pupillary reflex from their indirect effect upon the reflex optic ganglion of that side. "Crossed amblyopia," dimness of sight of the eye on the side opposite to the lesion, with concentric narrowing of the fields, has been observed by Gowers and others. The lesions giving rise to this condition have been found in the region of the angular gyrus.[4] The fields of the eye of the same

[1] Mills: *A Text-book on Nervous Diseases by American Authors*, p. 422.

[2] Gowers: *q. v.*

[3] This probably results from injury to the corpora quadrigemina, the reflex centre of sight.

[4] Gowers: *Diseases of the Nervous System*, 2d ed., vol. ii. p. 22, and Seymour J. Sharkey's observations: *The Lancet*, May 12, 1883, June 14, 1884, and May 22, 1897.

side are often narrowed. I have carefully observed a number of extensive lesions involving the angular gyrus, and have not met with a single case of "crossed amblyopia."

Auditory Centre and Temporo-sphenoidal Lobe.

Professor Schäfer[1] has vigorously opposed Ferrier's conclusion that in the temporal lobe of the monkey, especially in the first convolution, is located the auditory cortical centre. Ferrier[2] in an elaborate review of Schäfer's experiments on the temporal and occipital lobes condemned his conclusions, and on carefully reinvestigating the whole subject of cerebral localization he reaffirmed his own statements in regard to the location of the auditory centre in the first temporal convolution.[3] Careful clinical observations followed by accurate post-mortem examinations seem to prove conclusively that in man the auditory centre is in the temporal lobe, and largely limited to the posterior half of the first and probably to the second convolutions. These convolutions on one side innervate the auditory nerve of the opposite side, but that each nerve has a cortical representation in both hemispheres seems to be established from the fact that sudden or acute destruction of the temporal lobe on one side results in only temporary disturbance in hearing,[4] and almost complete destruction of these convolutions may occur, slowly on one side without any perceptible diminution in hearing.[5] In one case of large tumor beneath the first temporal convolution the convulsions were preceded by an auditory aura referred to the opposite ear, and in another of tumor in the supramarginal gyrus the unilateral convulsions were preceded by a loud noise, as of machinery.[6] That bilateral lesions completely destroying the integrity of the first and second temporal convolutions will permanently destroy hearing is conclusively proved by cases reported by Shaw,[7] Wernicke and Friedländer,[8] and Mills.[9] Ferguson has reported an interesting and curious case in which the patient, a young man, had almost completely lost the power of hearing with the right ear from otitis media of eight years' duration. Two years before death symptoms of tumor in the right temporal region developed, with convulsive movements and auditory aura, both referred to the left side. Hearing was completely although gradually lost at the end of eighteen months, but the auræ continued until death, which occurred six months later. The autopsy revealed a tumor in the right temporal lobe, with complete destruction of the first temporal convolution and partial of the second.[10] We have already seen that the centre for smell of the same

[1] E. A. Schäfer: *Brain*, vol. x., 1887, p. 362.

[2] David Ferrier: *Brain*, vol. xi., 1888, p. 7.

[3] *The Croonian Lectures on Cerebral Localization*, June, 1890, by David Ferrier.

[4] Sharkey: *Medical and Chirurgical Transactions*, 1884, p. 265, quoted by Gowers: a personal observation.

[5] Case of large fibro-glioma observed by H. C. Wood, and referred to by Charles K. Mills, *Trans. Amer. Cong. Phys. and Surg.*, vol. i. p. 277, and another case of softening recorded by Hall, *Arch. Med.*, April, 1881.

[6] Gowers: *Diseases of the Nervous System*, 1st ed.

[7] Shaw: *Arch. Med.*, Feb., 1882.

[8] Wernicke and Friedländer: *Brain*, vol. xi., 1888, p. 19.

[9] Mills, two cases: first case, *Univ. Med. Mag.*, Nov., 1889, vol. ii. p. 69; second case, *Ibid.*, Nov., 1891, vol. iv. p. 105.

[10] John Ferguson: *Journ. Anat. and Phys.*, vol. v., 1891, quoted by Gowers and Mills.

side is probably located in the anterior portion of the temporo-sphenoidal lobe on its median surface in the uncinate gyrus. In the posterior portion of the superior temporal convolution on the left side in right-handed persons, and on the right side in left-handed individuals, there seem to be cells that aid us in appreciating differences between the sounds of words and the differences between various sounds other than words. The loss of the first faculty results in word-deafness, and of the second in mind-deafness. That these centres are not as extensive as the auditory is evident from the fact that a person may be word- and mind-deaf and still be able to hear. This subject will be considered at greater length under the following head:

Affections of Speech as Localizing Symptoms.

The nearer cerebral localization approaches a science the more the various affections of speech as localizing symptoms are appreciated. During the year just past I met with three cases of brain lesions in which the only reliable localizing symptoms were the disturbances of speech, and in all, guided alone by these symptoms, surgical interference proved successful in averting apparently fatal terminations. The first case was that of a man who had been knocked senseless three months before by being struck with a chair on the right forehead just above the supraorbital ridge. He soon regained consciousness, but for two or three days was troubled with dizziness and headache. After this he returned to work as waiter for eleven weeks, when, after suffering from headache for a few hours, he became confused and passed into a semi-conscious and delirious condition. When I saw him, two days later, in St. Luke's Hospital a thrust of a pin into the right side of the body caused no movements, but the left leg and arm were withdrawn on being irritated. There was no paresis or paralysis of any muscles. The patient, although stupid and apparently delirious, would respond to questions, but his answers were usually irrelevant. When asked his name, he said, "Beer." When asked if he had pain, he replied, "Mamma, why don't you?" By a series of questions, continued for nearly half an hour, it became evident from his answers that he recognized in a dazed manner what was said to him, but that he was paraphasic. A full report of this interesting case has been published.[1] On E. A. J. Rogers removing a button of bone, at my suggestion, from the left parietal region, just below the parietal eminence, and exploring the brain, a considerable quantity of clotted dark blood and softened brain tissue was found in the centrum ovale at some depth below the angular gyrus. A complete recovery resulted. The second case was that of a Swede who had his skull fractured above the left ear. On E. J. A. Rogers removing a quadrangular piece of depressed bone, which was compressing the brain substance, consciousness was partially restored, but the patient was left completely word- and mind-deaf. One week after the first operation the wound was opened by Clayton Parkhill, the surgeon then on duty at the County Hospital, and the external surface of the dura was found covered with a reddish granular exudate which was composed of partially organized lymph. This was scraped off, and the surgeon felt

[1] *The Medical News*, June 6, 1896.

inclined to explore no farther, as the meningeal inflammation seemed sufficient to account for the convulsions from which the patient had suffered since the first operation. On my insisting that the inflammation on the external surface of the dura would not give rise to word- and mind-deafness, the dura was opened, but the internal surface of the dura, the pia, and cortex seemed normal. It was now evident that there must be a subcortical lesion. On exploring the first temporal convolution considerable partially coagulated blood was removed from the white substance of the brain just below the cortex. The mind-deafness began to lessen within a few days and soon passed away. The word-deafness remained for several weeks, but a most satisfactory recovery finally took place.[1] The third case was one of almost pure motor agraphia, and by this symptom alone the foot of the second frontal convolution was exposed, a cyst found, and removed for me by Clayton Parkhill. It had involved the cortex and white substance of this convolution. This case will be referred to again in considering Motor Agraphia.

The various affections of speech from brain lesions that are usually included under the term aphasia constitute one of the most difficult subjects in neuro-pathology, and for a comprehensive appreciation of the various speech disturbances more time is required than the general practitioner of medicine finds at his disposal. Not infrequently an attempt to simplify the study of such a subject results in obscuring it. Speech and language—for language is more than speech—are so intimately connected with the various functions of the brain that only lesions giving rise to certain disorders of speech can be accurately localized. There are probably numerous cortical centres for memories of speech, but at present not more than five are localizable. Of these, two are sensory, the auditory and visual, and the remainder motor—the centre for the muscular memories of the necessary movements concerned in speech utterance, the oro-lingual, and the graphic[2] centres. One or all of these centres may be partially or completely destroyed. The symptoms will vary according to the number of cortical centres involved and in proportion to the extent of the affection of the different centres. Subcortical lesions by interfering with the fibres that connect the various cortical centres with one another may produce very complex and profound disturbances in speech.

It is requiring too much to expect that every physician will master all the important details of the various forms of aphasia and understandingly investigate all the cases that come under his care. If one succeeds in presenting the most important localizing symptoms of the subject in such a manner as to be easily comprehended by all, it must be done at the risk of disregarding theories and suppressing many facts that are intensely interesting.

While Charles K. Mills, B. Sachs, and others of this country have written excellent articles on aphasia, M. Allen Starr has succeeded beyond all others in attracting general attention to the subject by his terse, smooth, and easy style of writing, and by his valuable suggestions for the clinical investigation of aphasia.

I shall make no attempt to write an article on aphasia, but shall

[1] *The Medical News*, June 20, 1896.

[2] It is questionable whether there is a separate and distinct centre for writing alone.

simply endeavor to arrange the principal positive and negative symptoms occurring from brain lesions in certain situations from which defects of speech are marked, so that they may be of service for ready reference in determining the location of the lesions in the various principal forms of aphasia.

To save the necessity of continual repetition it may as well be stated that lesions giving rise to disturbances of speech are, for all practical purposes located on the left side of the brain in right-handed persons and on the right side in left-handed individuals. The exceptions to this statement are sufficiently infrequent to prove the rule.

Motor Aphasia.—There are probably three cortical centres concerned in motor or emissive speech; hence motor disturbances of speech from limited cortical lesions may assume one of three forms. It is the rule, however, in motor aphasia to have all the cortical motor centres affected, and a mixture of symptoms in proportion to the extent of the lesion. The most common isolated variety of this form of aphasia is the one produced by involvement of the foot of the left third frontal convolution. If the lesion is strictly limited to the cortex, but more or less completely destroys this centre, the patient will be unable to speak voluntarily, to repeat words after another, to read aloud, to think in words, and in the majority of cases to write voluntarily or from dictation so as to be understood, but he will be able to understand speech, at times printing and writing, and he may be able to write from copy and to transcribe printing into script. If the wrong word is used, he is aware of it, although he may not be able to correct his mistake.

Dysarthria or defect in speech utterance from oro-lingual paralysis or paresis occurring from a lesion limited to the cortical centre for the movements of the muscles directly concerned in speech. This centre is located in the lower portion of both central convolutions. Mills[1] refers to an interesting case observed by him in which the lesion involved the lower portions of both central convolutions on the left side, the foot of the third frontal escaping almost entirely. Articulation was imperfect, but the patient could talk and propositionize. A case[2] corroborative of this observation is under my care at present. The patient is fifty years old, and gives a history of syphilitic infection a number of years ago. He was attacked with pain in the left side of the head and clonic spasms of the muscles of the right side of the face one day while he was on the street. Soon after this he found that he could not articulate distinctly, and that the right side of the face was partially paralyzed and the right hand was weak. He improved slightly except in the power of articulation, but three or four days later he noticed that the parts first affected were becoming weaker. When he first came under my care the right lower side of the face was completely paralyzed, the tongue in protrusion deviated to the right, the right arm and hand were paretic, and the right leg was weaker than the left. Articulation was indistinct, but with an effort he could make himself understood except for certain words. It was observed that the greater the effort he made to utter a word the more nearly he succeeded, although the articulation was accomplished with an explosive effort. He could think in words, proposition-

[1] *A Text-Book on Nervous Diseases by American Authors*, pp. 409, 410.

[2] *Medical News*, Aug. 15, 1896.

ize, read aloud, write spontaneously at dictation and copy, but he had never been a good penman, and it is impossible to determine to what extent his power of writing has been lessened. There is no transposition of letters or words nor insertion of wrong words. On E. J. A. Rogers trephining over the lower portion of the fissure of Rolando and exposing the lower portion of both central convolutions a softened or cystic degeneration of the cortex of these convolutions was found. The softened area was about three fourths of an inch in diameter and was fairly well defined.[1]

The symptoms of this form of speech-defect are quite distinct, as may be gathered from the case observed by Mills and the one under my own care. The patient may be able to utter short and simple words quite distinctly, and by repeated voluntary efforts pronounce words which at first trial are impossible for him to utter, provided the centre is not completely destroyed. In this respect the defect of speech is in marked contrast to that which occurs from a lesion in Broca's convolution, in which the greater the voluntary effort the more pronounced the failure of speech-utterance. The patient can write, read aloud simple words, and think in speech-symbols (propositionize).

In regard to the occurrence of dysarthria from oro-lingual paresis or paralysis due to a lesion in the extreme lower portions of the central convolutions (ascending frontal and parietal), we may conclude that this centre is bilateral and the paralysis is never complete from a unilateral lesion. The defect of speech from such a unilateral lesion is a dysarthria rather than an anarthria. It is distinguished from the disjointed articulation resulting from a lesion in the pons or medulla by the slight mental defect, by the symptoms of encroachment on Broca's centre, by the character of the paralysis, and the normal response of the affected muscles to the faradic current.

Anorthography, or So-called Motor Agraphia.—While the possibility of the occurrence of motor agraphia has been recognized by nearly all systematic writers on aphasia, yet very few are inclined to the view that there is a graphic centre distinct from that which controls the muscles of the hand. Most cases of motor agraphia have occurred in connection with a lesion of the foot of the third frontal convolution. Many, however, are of the opinion that in the foot of the second frontal convolution, which is just in front of the centre for the movements of the hand, is situated a centre in which the relation to writing is similar to that of Broca's centre with reference to speech utterance. Mills[2] says that no clinico-pathological case fully corroborative of this statement has been put on record. A case, caused by a cyst of the brain in the foot of the left second frontal convolution, has recently been observed by me, which as nearly fills the gap as it is possible for one case to do: A vigorous man, a farmer and a stock-raiser by occupation for the last few years, began about a year ago to suffer from irregular attacks of a spasmodic nature, during which he would be dazed and temporarily unable to speak. Nine months later, when I made a careful

[1] This patient died some months later from a thrombotic occlusion of an artery of the pons. It was found at the autopsy that the lesion, for which he had been operated upon, was limited to the lower portions of the ascending convolutions.

[2] *A Text-book on Nervous Diseases by American Authors*, p. 436.

examination of his condition, I found all sensory phenomena normal and no paresis or paralysis of any muscles. With the right hand he registered on the dynamometer 230; left, 220. He complained of some headache, which was intermittent and located in the front of the head, more on the left side than on the right. There was no disturbance of the special senses. The fundus and papilla of each eye were normal. There was no sensory aphasia. The power of articulation was quite good, except that it was slow and long words were difficult for him to utter distinctly. He could talk, read printing and writing to himself or aloud, and understood what he read. There was no difficulty in propositionizing, and the movements of the lips and tongue were well preserved except in uttering long and hard words; otherwise it was impossible to discover any defect in speech. In writing he formed his letters, as a rule, perfectly, but he transposed letters, words, phrases, and occasionally sentences, so that it was impossible to read what he had written. He had formerly been a good penman, and for two terms served as bill clerk in the house of representatives of the State legislature. I had the patient under observation for about two months, and during this time he wrote for me a letter daily. The letters usually consisted of about eight or ten lines, and each day he spent two hours or more in writing and erasing words before he could complete his assigned task. At times he seemed to recognize his mistakes in spelling, and at others he did not recognize them or was indifferent to them. On realizing a mistake in spelling he would often make a greater one in trying to correct it. Finally, I decided to recommend a surgical operation for the removal of a supposed growth or cyst in the left frontal lobe. On December 5, 1895, Clayton Parkhill, at my request, trephined over the foot of the second left frontal convolution, found and removed a cyst containing about half an ounce of a straw-colored fluid. The cyst had destroyed the cortex over an area about half an inch in diameter, and extended into the white substance of the brain to the depth of nearly an inch. For a full account of this interesting case and its subsequent course the reader is referred to the detailed report.[1] The patient did well for some time, and his subsequent efforts at writing and the gradual improvement in his letters are most interesting.

If any conclusion is justifiable in the observation of one case, it would be that a lesion in the foot of the second frontal convolution on the left side in right-handed persons, and probably on the right side in left-handed individuals, will give rise to a defect in speech in which the principal symptom will be the loss of the power to spell[2] or arrange letters in words. It should be borne in mind that a lesion, such as a tumor, abscess, cyst, and probably thrombotic softening, occurring in the foot of the second frontal convolution will interfere to some extent with articulation, with the mentality of the patient, and in many cases with the function of Broca's convolution. It is probable that the syntactic disturbances in speech depend upon the impaired mental condition of the patient. Unlike words, sentences are not stored up in memory ready for use.[3]

[1] *Medical News*, Aug. 1, 1896.
[2] It is possible that the primary defect is in the inability to pronounce.
[3] Kussmaul.

Does a centre for writing exist? According to Bastian and others, cases of agraphia have been observed in which the patient was unable to make more than meaningless strokes. These patients have been unable to read or spell correctly. We can write with a pen held with the teeth, hand, or foot. It is very improbable that the same centre in the cerebral cortex would serve to guide these various widely-separated groups of muscles in writing. On the other hand, no matter what groups of muscles we employ in the formation of letters, the arrangement of the letters in a given word is always the same. It seems to me more probable that the memory for the arrangement of letters in words is stored up in the cerebral cortex. This process is learned by tedious and repeated repetition. We should naturally expect to find the cortical centre in which is registered the memory pictures for the arrangement of letters in a word to be located near the centre which governs the muscles usually employed in writing. It will be interesting to observe whether a lesion in the second frontal convolution on the right side of the brain in right-handed persons is attended with any loss of the power in spelling. So far as I can see at present, it is impossible to investigate the orthographic centre in a very illiterate person.

The centre in which is stored the memory pictures for the arrangement of letters in words, etc. is probably situated in the foot of the left second frontal convolution in right-handed persons, and presumably in the corresponding convolution on the right side in left-handed individuals.

Cases of so-called pure motor agraphia, apart from the involvement of the muscles concerned in writing, have never been demonstrated, and, reasoning from analogy, it seems improbable for them to occur.

Whether the loss of the power to write intelligently the letters of the alphabet, still being formed perfectly, from the loss of the ability to spell or arrange letters in words according to usage, should be termed agraphia or anorthographia is questionable. I have met with one case in which the only defect connected with speech-mechanism is the inability to pronounce. The patient is well educated and bright, writes a good hand voluntarily at dictation, and copies well and rapidly, transcribing both printing and writing into script. When I pronounce a word for him he spells it without hesitation, but he cannot pronounce such simple words as dog, car, rat without showing some hesitancy and uncertainty. Longer words he cannot pronounce at all or very rarely, and then only after a considerable effort. If I pronounce similar words for him, he spells them readily. The lesion in this case was an old injury to the skull over the cortex, just in front of the face centre on the right side. To what extent the brain has been injured I am not able to say.

A few words here will be sufficient to call attention to the differential diagnosis between motor and sensory anorthographia or agraphia. In pure motor anorthographia or agraphia there is neither auditory nor visual disturbance of speech. The patient does not copy mechanically as in the sensory form, but both printing and writing are copied into script.

Combined Motor Aphasia.—The most common form of motor aphasia is where all the cortical motor centres are affected by the lesion, either by partial destruction of all three cortical areas or indirectly by

involving the commissural fibres that connect the several cortical centres. In such cases, if the lesion is sufficiently extensive or situated so as to affect the fibres to the corpus callosum, the aphasia will be permanent.[1] The patient may be speechless, not able to utter a single word or intelligent sound, although he is rarely wordless,[2] as he can understand the speech of another. Emotional speech is affected to a less extent than voluntary speech. While the patient may at first be unable to utter a single word voluntarily, oaths, interjections of surprise, delight, anger, etc. may be uttered suddenly; but if the patient attempts to repeat them voluntarily he will often fail. Gesture or pantomimic expression, next to emotional speech, is best retained, although this is often greatly impaired at first. It must be remembered that loss of the power of expression by gesture may be due to a lesion either on the motor or sensory side of speech mechanism. A motor aphasic, when the lesion is a cortical one, if able to write at all, will usually copy both printing and writing into script, while a sensory aphasic will copy mechanically each letter just as it appears to him; thus writing will be copied into script and printing will be slowly and laboriously printed, each letter receiving the degree of shading found in the original. Whether this method of copying is invariably followed out by the sensory and motor aphasic, as Déjerine believes, is doubtful.[3]

Sensory Aphasia.—Under this head most of the auditory and visual disturbances of speech, including apraxia,[4] may be studied in reference to localization. That a lesion in the posterior portions of the first and second temporal convolutions will cause word-deafness and mind-deafness, and that the lesion necessary to produce mind-deafness must be more extensive than one capable of causing word-deafness, seem to be demonstrated by a case recently seen and studied by me:[5] A vigorous young man was struck above the left ear by the butt of a pistol, fracturing the skull and driving a piece of bone against the first and second temporal convolutions. After the depressed bone had been removed the patient soon regained consciousness, but he was mind- and word-deaf. There were other disturbances of speech, but they do not concern us at present. The patient could hear, as was evidenced by his looking in the direction from which a sound proceeded. He showed no gleam of intelligence when a watch was held to either ear, when a bell was tapped, or when coins or keys were jingled. A few days later the first and second temporal convolutions were explored, and a small quantity of dark, semifluid blood was found just below the cortex and removed. Improvement began soon after the second operation, and a few days later he was able to understand all sounds except those of words. When a watch was held against his ear he said, "Going pretty well," but when I spoke to him he stared at me and seemed puzzled. In the course of a few weeks he made a complete recovery. The depressed bone was $2\frac{1}{2}$ inches long by $1\frac{1}{4}$ wide, with the long axis extending from before backward. The centre of the fracture was crossed by the auric-

[1] M. R. Gowers: *Diseases of the Nervous System*, 2d ed., vol ii. p. 114.
[2] Hughlings-Jackson.
[3] Déjerine: "Sensory Aphasia," *Internat. Med. Mag.*, Aug., 1895, pp. 524–528.
[4] Apraxia is due primarily to a mental defect, but gives rise to speech disturbance.
[5] *Med. News*, June 20, 1896.

ulo-bregmatic line, and the lowest portion of the fractured bone was about half an inch above the base of the skull. The hemorrhagic extravasation had taken place subcortically in the posterior half of the exposed area of the brain. If the ability to interpret the meaning of sounds in general resides in the same cortical area in which the memory of the sound of words is stored, the former must extend over a larger surface than the latter, because mind-deafness began to lessen as soon as pressure from the extravasated blood was relieved, although the word-deafness did not show much improvement for two weeks later. It is just possible that the cortical centre for the interpretation of sounds in general is situated posteriorly to the cortical area in which word-memory resides.

Auditory Affections of Speech and Language.—Two forms of word-deafness are recognized by some. One is termed pure word-deafness, and the only symptoms are inability to understand spoken speech and consequently failure to write at dictation. The person so affected shows no defect in reading aloud, writing voluntarily or copying, or understanding what he has read or written. Déjerine refers to a typical clinical case of this form of word-deafness observed by Serieux.[1] Déjerine thinks that the anatomical localization of pure word-deafness is not established, but is inclined to place it in the left temporal lobe. This form of word-deafness is probably due to a lesion in the left temporal lobe, and involves the auditory entering tracts from both hemispheres, as suggested by Lichtheim.[2] The latter observed a clinical case of pure word-deafness in which there was no disturbance in volitional speech and writing or in reading aloud. M. Allen Starr has collected seven cases, most of which are examples of pure word-deafness, and in all of these the lesion was limited to the first and second left temporal convolutions.[3]

The second form of word-deafness is the sensory aphasia described by Wernicke, in which there is not only loss of the power to understand spoken speech, with inability to write at dictation, but reading aloud is impaired or abolished, and, as the patient cannot verify by audition what he says or means to say because the centre for word-hearing is affected, paraphasia is present, including paragraphia and paralexia. It is questionable whether two forms of word-deafness should be recognized. In both the lesion is in the posterior two thirds of the first and probably the second temporal convolution, but in pure word-deafness it is limited to the cortex in all probability, while in word-deafness with paraphasia it is more extensive and the subcortical association tracts are undoubtedly affected.

While we may clinically distinguish three forms of auditory disturbances of speech, pure word-deafness, mind-deafness, and word-deafness with paraphasia, the lesion in all is in the posterior portion of the first and second temporal convolutions, being least extensive in the first variety, and increasing in the order named in the other two, in both of which it extends to the subcortical association tracts.

Visual Affections of Speech and Language.—Two forms of word-

[1] *Internat. Med. Mag.*, 1895, Aug., p. 529.
[2] Quoted by Mills in *A Text-Book on Nervous Diseases by American Authors*, p. 438.
[3] M. Allen Starr: *Brain*, vol. xii., 1889, p. 86.

blindness are described by Déjerine :[1] (1) The ordinary form, in which the patient is usually completely or partially agraphic, paraphasic, and alexic. This condition is due to a lesion in the cortex of the gyrus angularis. When the lesion is deep and involves the optic radiations on their way to the cuneus, right lateral hemianopsia will also be present. The patient is usually unable to recognize words or letters, but he may or may not be able to read figures and to calculate. (2) The second form of word-blindness has been described by Déjerine and termed *pure word-blindness:* "The patient writes spontaneously and at dictation, as in the normal condition, naturally, without being able to read it; but a copy is altered and made mechanically. Speech and intelligence are intact." In a case observed by him the lesion was found at the autopsy to involve the point of the occipital lobe, the base of the cuneus, and the lingual and fusiform lobules. The patient, very intelligent and cultivated, was totally word-blind and hemianopsic, but he could write spontaneously and at dictation. He was also note-blind. Formerly a good musician, he could not decipher a single note. He could read figures and calculate, and speech and intelligence were intact. Wyllie and Redlich, according to Déjerine, have reported similar cases which presented the same anatomical localization. Cases 42, 43, and 44 in the table compiled by Starr bear a striking resemblance to Déjerine's case.[2]

Mind-blindness, inability to determine an object by the visual organs, is a variety of apraxia in which the patient has lost the power to recall the use of an object. Such a defect is due to a lesion in the occipital lobe, situated in the cortex or deep in the white substance, involving the association fibres which connect the occipital cortical centres of sight with the other regions of the brain. In the 50 cases of sensory aphasia collected by Starr, 12 presented a condition of psychical blindness, and in all the occipital lobe was diseased. In 6 of the 12 cases hemianopsia was also present.

Not infrequently word-deafness and word-blindness, with various disturbances of speech, occur from extensive cortical or subcortical lesions, so that while word-blindness is produced by a lesion in the cortex of the angular gyrus, it may also occur, as Starr has pointed out, from one extending from this centre either anteriorly into the temporal region or posteriorly into the occipital. In the former instance it would probably be associated with word-deafness, and in the latter with mind-blindness.

In testing the temporo-occipital association tracts, inability to distinguish a familiar object by its sound would indicate that the temporal end is affected; failure to recognize an object seen, a break in the occipital end; and loss of power to recall the name of an object recognized, a lesion in the course of the tracts between the temporal and occipital lobes.[3]

Professor Déjerine was among the first to call attention to the fact that in some cases of word-blindness there occurs a defect of speech similar to that observed from a lesion in Broca's convolution. Of 27 cases tabulated by Starr in which word-blindness and word-deafness existed, a motor defect of speech was observed in only 2.

[1] *Internat. Med. Mag.*, Aug., 1895, p. 528.
[2] *Brain*, vol. xii., 1889, p. 100.
[3] Starr.

Speech disturbances from a lesion in the internal capsule scarcely require a notice here, as the unilateral motor or sensory symptoms sufficiently determine the seat of the disease.

Recapitulation of some of the principal localizing symptoms of speech-defect from cortical lesions.

If the lesion is in the foot of the third frontal or Broca's convolution, the patient will be unable to speak voluntarily, to repeat words after another, to read aloud, to think in words, and in the majority of cases to write voluntarily or from dictation so as to be understood, but he can understand speech.

A lesion in the oro-lingual centre (lower portion of the central convolutions) will cause paresis or paralysis of the oro-lingual muscles, including the lower side of the face on the side corresponding to the most dextrous hand normally, and imperfect articulation, but the patient will be able with an effort to repeat words, to talk voluntarily, to think in speech, and to write, although the last may be done imperfectly.

A lesion in the foot of the second frontal convolution (the probable anorthographic or graphic centre) will be attended by disturbances in writing. In the only case in which a limited lesion has been found in this centre the patient could articulate, propositionize, and form his letters perfectly in writing, but there was usually such a general transposition of letters, words, and at time phrases, that no sense could be gotten out of what he had written, and he himself could not read it much better than another, especially his wife.

A limited lesion in the posterior two thirds of the first and second temporal convolutions will be attended by word-deafness and inability to write at dictation ; in more extensive cortical lesions in this region mind-deafness with paraphasia and some disturbances in reading and writing will be added. The greater the extent of the cortical area involved the more marked the paraphasia and other symptoms of sensory aphasia. If the lesion involves the subcortical white substance, the disturbances in speaking, reading, and writing become very pronounced.

A lesion involving the angular gyrus and adjacent parts will cause word-blindness and inability to read, and defects in writing, copying, and speech (paraphasia) will usually be present. If the lesion involves the parts posterior to the angular gyrus, mind-blindness will be added.

It is impossible to condense the symptoms of lesions in the different associating tracts between the various cortical areas concerned in speech, and the attempt that has been made to represent in a few words the symptoms of speech-defect from cortical lesions is imperfect in that it takes no account of many varying conditions.

There is much to be said in favor of recognizing a centre for the memory of nouns, a naming centre (Broadbent and Mills), but further observations are necessary before ascribing to this faculty a distinct and separate cortical centre.

In studying cases of aphasia it is often just as important to note what symptoms are absent as to recognize and describe those that are present. Very few can systematically examine cases of aphasia, especially of the sensory variety, for all possible symptoms, both negative and positive, unassisted by some plan in which the different headings are represented.

It is needless to remind the reader that a most thorough and exhaustive examination of the general physical and mental condition of the person suffering from aphasia should be made at the time of recording the symptoms.

Of all plans suggested for investigating and recording cases of aphasia, I have found none simpler than the one suggested by M. Allen Starr.[1] I have employed this for a number of years, and added to it from time to time. I am in the habit of having the suggestions printed on a page of legal paper, and allow sufficient space between each division of the subject for recording changes at different examinations. At the end two or more blank sheets are bound with the printed ones, on which general observations of the case can be recorded.

The plan, as modified, is as follows:

1. The power to recognize objects seen, heard, felt, tasted, smelt, and their uses.
2. The power to recall the spoken names of objects seen, heard, felt, tasted, smelt.
3. The power to understand sounds other than speech and music.
4. The power to understand speech and music.
5. The power to recall to mind objects named, and point them out at request.
6. If word-deaf, can the patient recognize his own name when spoken?
7. The power to recognize a word spelled aloud.
8. The power to pronounce inaudibly or aloud on hearing the word spelled.
9. The power to spell a word inaudibly or aloud on hearing it pronounced.
10. The power to call up mentally the sound of a note, figure, letter, word.

The examination, thus far, will test the various sensory areas, but more especially the auditory and the association tracts between the different sensory areas connected with speech.

11. The power to recognize letters, figures, notes, and colors seen.
12. The power to understand printed and written words seen.
13. The power to read printing, writing, and music aloud or inaudibly, and to understand what he reads.
14. The power to recall objects the names of which are seen.
15. The power to write voluntarily.
16. The power to write at dictation.
17. The power to copy and the manner of copying, printing, and writing.
18. The power to write the names of objects seen, heard, felt, tasted, smelt.
19. The power to read aloud or inaudibly, and to understand what has been written.
20. The power to write his name, and the ability to read it when written by himself, by another person, or when it is printed.
21. The power to recognize a letter by tracing it with the index finger or with a pencil, the movements being guided by another.

[1] Starr: *Med. Record*, Oct. 27, 1888, p. 497; and *Brain*, vol. xii., 1889, p. 95.

22. The power to call up mentally the appearance of an object, a figure, note, letter, or word, when word-blind.

These additional tests will aid in determining the condition of the visual word-memories in the angular gyrus, and the connections between this area and the surrounding sensory and motor areas:

23. The power to speak voluntarily, and, if it is impaired or lost, the character of the defect.

24. The power to repeat words after another.

25. Does the patient recognize his mistakes in speaking, reading, writing, and spelling, and can he correct them?

26. Can the patient think in speech?

27. Is there any special difficulty in the use of nouns, verbs, or other parts of speech.

28. The power to understand and use pantomimic or gesture expression.

29. The power to read figures and to calculate.

30. The power to count both money and in numbers.

31. The power to play a game of cards or other games.

Non-cortical Centres.

Centrum Ovale.—The white substance of the brain contains at least three distinct sets of connecting fibres. One set connects the different convolutions in the same hemisphere, and it is by these fibres that the frontal and occipital lobes may be brought into close relation with each other. The second is composed of the callosal fibres, which probably connect corresponding portions of each hemisphere. The third set contains fibres that pass from the cortex to the great ganglia and crus. Most of the fibres from the cortex that reach the crus pass through the internal capsule, but some pass without the capsule. Among the latter may be mentioned fibres from one occipital lobe to the cerebellum of the opposite side. The function of the fibres of the centrum ovale seems to be to carry impressions that reach the sensorium to the cerebral cortex, to conduct impulses from the latter to the motor apparatus, and to connect and harmonize the various portions of the brain.

A lesion in the centrum ovale, if it involves the motor or sensory fibres, produces symptoms that are somewhat similar to those that follow a lesion in the cortex or internal capsule. Whatever may be the opinion in regard to the function of the cortical motor area, the sensory and motor fibres are separated in the greater portion of the centrum ovale and in the internal capsule. A lesion in the centrum ovale just below the cortex of the motor region may cause a monoplegia, but convulsions will not likely occur unless the lesion is an irritative one. In cortical lesions local spasms usually precede paralysis, while in subcortical ones the order of the development of these symptoms is commonly reversed. General convulsions from a lesion in the white substance are rare unless the intracerebral pressure is considerably increased. If the lesion in the white substance of one occipital lobe involves the optic radiations, hemianopsia will be present; if in the parietal region, partial anæsthesia of the entire opposite side of the body may occur; and when the centrum ovale in the left hemisphere is affected in the temporal and

parietal regions, or in such a position as to invade the fibres from Broca's convolution to the internal capsule, aphasia will be present. A lesion in the centrum ovale will probably not cause permanent motor aphasia unless the fibres immediately subcortical to Broca's convolution are destroyed. Lesions in the centrum ovale near the internal capsule usually invade the latter, and are rarely distinguishable from a primary capsular lesion.

Corpus Callosum.—While corresponding portions of the two hemispheres are probably connected by fibres passing through the corpus callosum, it is doubtful whether the lesions limited to this structure can be localized with any degree of accuracy. The principal lesion that has occurred in the corpus callosum is tumor, and the symptoms have been so variable as to leave little doubt that they are due to extension of the growth to adjacent structures and to the increase of general intracerebral pressure. (See Tumor of the Corpus Callosum, p. 472.)

Central Ganglia and Internal Capsule.—In the study of cerebral localization from a practical standpoint little more need be said of the great ganglia than that their exact functions are too imperfectly understood to be of much value in this connection. Lesions in these ganglia are mainly localizable by their effects upon the internal capsule. The importance of the fibres composing the internal capsule anterior to its angle is not known, and in consequence a lesion in this situation may be attended by no definite symptoms. The fibres from the cortical areas for the face, tongue, and larynx are found at the angle or knee of the internal capsule and in the part immediately posterior to it; those from the arm centre come next, and the fibres from the leg centre lie immediately behind these. The anterior two thirds of the internal capsule posterior to the angle contain the fibres from the cortical motor areas, the relative position of the fibres from each area being represented in the same order from before backward in the capsule as they are in the cortex from below upward. The posterior third of the hinder limb of the internal capsule contains the sensory fibres to the opposite side of the body. The fibres of the organs of special sense, except probably those of vision, find a place in the "cross-way" of Charcot, or sensory portion of the internal capsule. A lesion may affect the motor or sensory fibres alone. Hemorrhagic lesions involving the motor portion, with only temporary disturbance of sensation, are very common. It is possible to have a lesion so limited in the motor portion of the internal capsule as to produce monoplegia, but this is of rare occurrence. Complete destruction of the motor fibres gives rise to typical hemiplegic symptoms of cerebral origin, in which the muscles of the lower side of the face, of the tongue, arm, and leg, are paralyzed on the opposite side, and the trunk muscles are weakened on both sides. If the lesion occurs on the side in which the faculty of speech is located, temporary motor aphasia may result. General convulsions often occur from irritative lesions in the motor segment of the internal capsule. Partial destructive lesions, especially if attended by symptoms of irritation, may cause troublesome contractures in the affected muscles. A sudden lesion involving all the fibres in the sensory portion of the internal capsule gives rise to complete anæsthesia of the entire opposite side of the body, including the scalp up to the median line, with loss of hearing, taste,

and smell of the anæsthetic side. If the lesion and its effects are strictly limited to the internal capsule, vision, according to Henschen and Charles K. Mills,[1] is not affected. These authors contend that none of the visual fibres pass through the internal capsule. The affection of the special senses is usually temporary in character. Incomplete destructive lesions of the sensory portion of the internal capsule, especially if they are attended by irritation, as sometimes happens from thrombotic softening, may give rise to most harassing and distressing symptoms of a sensory character. These were so intolerable in a case recently seen by me in consultation with J. W. O'Connor as to drive the patient to commit suicide.

A lesion strictly limited to the thalamus does not affect sensation, but a condition of athetosis or mobile spasm, or one of tremor similar to that found in multiple sclerosis, except it is always unilateral.

Corpora Quadrigemina.—The connections of these bodies with various structures are quite numerous, but their exact function is unknown. They seem to be intimately associated with the reflex acts of vision, apparently in this manner regulating the size of the pupils. Unilateral destruction of these bodies causes loss of the reflex action of the pupil to light on the opposite side, the hemiopic pupillary reflex of Wernicke. (See Tumor of the Corpora Quadrigemina, p. 473.)

Crus Cerebri.—In the anterior or lower portion of the crus, in the middle third of the crusta, the motor fibres for the opposite side of the body, including the face, lie; the sensory fibres to the entire opposite side of the body are found in the tegmentum which occupies a position between the crusta and the parts beneath the floor of the aqueduct of Sylvius; and the third cranial nerve from its nuclear origin in the latter situation emerges on the inner border of the crus to reach the muscles of the eye of the same side. The most characteristic symptom of a lesion in the crus is paralysis of the lower side of the face and of the arm and leg of the opposite side, and paralysis of the third nerve on the side corresponding to the lesion. Sensory disturbances are common, and are found on the same side as the paralyzed limbs. The paralysis of the third nerve may be partial, but it is more commonly complete. Sometimes the internal ocular muscles escape entirely. When the lesion is near the median line and involves the nerve nuclei near the floor of the aqueduct of Sylvius, ophthalmoplegia of nuclear origin will occur. Hemianopsia from crus lesions is rare, except in the case of a tumor. A superficial lesion on the inner side of the crus may cause partial paralysis of one third nerve and be unattended by sensory or hemiplegic symptoms. Under such circumstances giddiness, probably from the affection of the ocular muscles, may be troublesome and persistent.

Pons.—The sensory fibres of the pons occupy a position posterior, caudad, to the pyramidal tracts. The fibres of the fifth cranial nerve are in the outer third of the formatio reticularis, both above and below the junction of the ascending and descending roots of this nerve.[2] Above this junction, which is near the vertical middle of the pons, a lesion limited to the outer portion of the reticular formation produces anæsthesia in the distribution of the fifth nerve on the opposite side. If the lesion is equally limited to the outer third of the reticular formation

[1] *A Text-Book on Nervous Diseases by American Authors*, p. 422.
[2] It is probable that the "descending" root is really ascending.

below the junction of the two roots, the sensory disturbance occurs in the distribution of the fifth nerve on the side corresponding to that of the lesion. A lesion limited to the inner two thirds of the reticular formation in any portion of the pons causes anæsthesia in the limbs, trunk, neck, and back of head nearly up to the vertex on the opposite side. A lesion limited to, but involving, the entire formatio reticularis above the middle of the pons produces anæsthesia on the opposite side of the body, including the face; below the middle of the pons, crossed hemianæsthesia, the face on the same side, and the limbs, body, neck, and back of head on the side opposite to that of the lesion. A few cases of lesion of the pons have been attended with loss of temperature and pain sensations in the limbs, tactile sense being nearly normal. Such a lesion is situated deep in the substance of the pons. It is probable that dissociation of sensory symptoms is most complete when due to a lesion of the pons, next when caused by lesion of the cord, and least when it results from a lesion of the spinal nerve roots. If the lesion involves the motor tracts above the middle of the pons, and is limited to them, the limbs and the lower side of the face will be paralyzed on the opposite side; and if none of the cranial nerves or their nuclei other than the facial are affected, the symptoms may be indistinguishable from those of a lesion in the motor portion of the internal capsule. A lesion affecting the motor tracts below the middle of the pons causes crossed paralysis, the face, both in its upper and lower portions, on the side of the lesion, and the body on the opposite side. Several of the cranial nerves and their nuclei may be affected by pontile lesions. The third, fourth, fifth, and sixth are the ones that are most commonly included in such lesions, but any from the third to the eighth inclusive may be involved, depending upon the character, seat, and extent of the lesion. A unilateral lesion in the extreme upper portion of the pons may affect the third nerve or its nuclear origin, but such a lesion could scarcely involve the fifth nerve without giving rise to hemianæsthesia and hemiplegia. The fibres of the fourth cranial nerve from their origin in the floor of the fourth ventricle pass over to the opposite side in the valve of Vieussens. A unilateral lesion affecting the middle vertical third of the posterior portion of the pons may paralyze the fourth nerve on the same side as that of the lesion or on the opposite side. If the nerve is affected by a vascular lesion in the lateral substance of the pons, it is usually the one on the side corresponding to the lesion. The seventh and eighth nerves are occasionally affected by lesions of the middle and lower portions of the pons. Owing to the associated action of the external rectus of one eye, supplied by the sixth nerve, with the internal rectus of the other eye, supplied by a branch of the third nerve, in conjugate deviation of the eyes, portions of these two nerves are frequently affected by a common lesion.

A paralyzing lesion of the pons above the nuclear origin of the sixth nerve often causes loss of conjugate deviation of the eyes toward the affected side, while an irritating lesion may give rise to conjugate spasm of the eyes toward the same side. The motor root of the fifth nerve may be involved and the sensory may escape. Bilateral lesions of the pons are not uncommon, and may cause, besides the bilateral motor and sensory symptoms in the face and limbs, great disturbance in deglutition

and articulation, especially when the lesion affects the lower portion. Irritative lesions of the pons may give rise to rigidity of the muscles of the limbs or of those supplied by the motor root of the fifth nerve. Choreoid movements or rhythmical spasms have been observed in rare instances. Trophic disturbances, especially in the eye, and sometimes pain, are well-marked symptoms on the side of the lesion in the distribution of the fifth nerve when the trunk of this nerve is involved. Unilateral sweating has been observed in a number of cases from lesions of the pons. A sense of unsteadiness amounting to actual giddiness is not infrequent from pontile lesions when the middle peduncle is affected. Such cases are usually attended by vomiting. A marked rise of temperature is common immediately after the occurrence of an acute lesion of the pons.[1]

Medulla.—Besides the sensory and motor tracts connected with both sides of the body the medulla contains nuclei of the pneumogastric, glosso-pharyngeal, hypoglossal, and spinal accessory nerves. A lesion destroying the cardiac and respiratory centre is rapidly fatal. Symptoms of bulbar lesions are usually bilateral. The lesion may be limited to the nuclei of the motor nerves, as in acute or chronic bulbar paralysis, or it may affect the glosso-pharyngeal, hypoglossal, and spinal accessory nerves and the motor and sensory tracts. The muscles of the face will escape, except the orbicularis oris, supplied by the fibres that take their origin near the hypoglossal nucleus. The tongue, pharynx, and larynx are affected, so that the power of deglutition and articulation may be greatly impaired or entirely lost.

Cerebellum.—The cerebellum, by means of its middle lobe and its peduncles, has numerous and important connections. The inferior peduncles are the paths by which nerve fibres reach the cerebellum from the auditory nerves and their nuclei, and from the posterior portions of the cord, especially the direct cerebellar tracts. The middle peduncles serve to establish a connection between the middle pons and cerebellum. The superior peduncles contain many fibres that connect various portions of the cerebrum, probably the tubercular quadrigemina, the thalamus, corpus callosum, and the cortex of the hemispheres, with the cortex and middle lobe of the cerebellum. Each cerebellar hemisphere is connected with the cerebral hemisphere of the opposite side. Clinical and pathological observations point to a close relation existing between each cerebellar hemisphere and the frontal lobe of the opposite side.

Whatever the functions of the cerebellar hemispheres are, their individuality is lost in the middle lobe. The connections of the cerebellum with parts, the direct cerebellar tracts and the semicircular canals, that are concerned in maintaining the equilibrium of the body, favor the generally accepted opinion that the cerebellum, especially its middle lobe, is intimately associated with co-ordination of muscular movements. Many of the symptoms following lesions of the cerebellum result from the pressure upon or irritation of adjacent structures. Tumors of the middle lobe or of the peduncles not infrequently interfere with the functions of the pons or medulla. A lesion in one cerebellar hemisphere unattended by pressure upon the middle lobe may

[1] A number of exceptions to this rule have been observed.

not give rise to any appreciable symptoms. The most common symptoms of a lesion in the middle lobe of the cerebellum are the disturbed muscular movements, limited largely to the legs when the patient is in the upright posture, seen to a less extent in the trunk, and occasionally observed as a tremor in the arms. The patient may have a sense of insecurity even when in the recumbent posture. When he is on his feet the gait is uncertain or zigzag, and resembles that of a drunken person. Both in standing and walking the feet are kept well separated. If standing is attempted with the feet close together, the body sways to and fro. Closing the eyes increases the difficulty of maintaining the equilibrium. In some cases there is a tendency to forced movements in one direction, probably less commonly backward than forward or to one side. The irregular movements of the legs, even when the patient is in the erect posture, are rarely as great as in locomotor ataxia, and on lying down the inco-ordinate movements of the legs are scarcely apparent, differing in this respect from the ataxia from a spinal lesion. Vertigo and vomiting are common in irritative lesions of the cerebellum. General convulsions may occur from a vascular lesion, but they are more frequently associated with tumors of the cerebellum. (For a fuller description of the lesions of the cerebellum and its peduncles, see Tumors of the Cerebellum, p. 474.)

DISEASES OF THE MENINGES OF THE BRAIN; THROMBOSIS OF THE SINUSES OF THE DURA MATER.

By F. T. MILES, M. D.

The brain is usually described as being enveloped by three membranes—viz. the dura mater, the arachnoid, and the pia mater. The two latter there is good reason for considering as a single membrane, and we may designate it as the pia-arachnoid. This is the more justifiable, practically, as the arachnoid is not affected by disease as a separate membrane.

The dura is composed chiefly of white fibrous tissue, is tough, inelastic, and of low vascularity. It is supplied with nerves, principally from the fifth pair, from which it derives a sensibility which may be exaggerated in disease so as to cause exquisite pain. It must be remembered, however (and this has an important bearing on the symptoms when it is diseased), that, like other internal organs, its sense of localization cannot be depended upon, as the pain radiates widely from the point of causation (Hugenin). Its outer surface is the internal periosteum of the cranial bones, and is intimately connected to them by fibres and blood-vessels which further communicate with the vessels of the scalp. Its inner surface is smooth and glistening, and covered with a layer of epithelium. It is made up of two layers intimately united in the adult, except where at certain points the inner layer leaves the outer and forms lamellar projections between divisions of the brain. Between and formed by the two layers are the sinuses of the dura mater, channels for carrying the venous blood from the brain, which are lined by a continuation of the internal coat of the veins. The dura mater is supplied with blood principally by the middle meningeal artery, which ramifies in its periosteal or outer layer, giving branches to the bones and to the inner layer.

INFLAMMATION OF THE EXTERNAL LAYER OF THE DURA MATER.

External Pachymengitis.

The inflammation of the outer layer of the dura mater can scarcely be classified as a disease, so seldom does it occur as an idiopathic affection. It is almost always observed as the result of head injuries,

such as concussion or lesions of the cranial bones or diseases or inflammations of the soft parts covering them. Concussions of the head, as from blows which cause no apparent lesions, may so jar the cranial bones as to loosen the dura mater from their inner surface, either at the spot of concussion or by contre-coup at a point opposite to it. With this there may be extravasation of blood from the torn vessels between the bone and membrane. Sometimes it is but a thin layer of blood, which is readily absorbed, leaving only a thickening of the membrane; or the blood may be in such quantity as to cause symptoms of general cerebral compression, and if the compression occurs at the motor region (beneath the parietal bone, a common seat of injury), there may be contractions, paresis, or paralysis of the opposite limbs or of the muscles of the face. In this way it may be possible to make a diagnosis of the lesion. It is rare to have pus formed between the dura mater and the bone, but if this occurs to any great extent, there will most likely be localizing symptoms, or those of compression. But even in cases in which a blow has not been of force sufficient to detach the dura mater an inflammatory condition may be set up in a circumscribed area of it corresponding to the point of violence. The vessels of the membrane are distended, and it becomes swollen with exudation and thickly crowded with leucocytes. This morbid process seldom ends in the formation of pus; oftenest there is the production of fibrous tissue, which thickens the membrane permanently, and unites it firmly to the bone, which itself probably undergoes changes, making it thicker and more dense. Such changes often exist unrevealed by symptoms, and are unexpectedly found after death. Not a few cases, however, occur in which we have good reason to believe that persistent headache and other cerebral symptoms depend on a change and thickening of the dura mater resulting from some almost or quite forgotten injury. The diagnosis of such cases is obviously very difficult, and often impossible, but it may sometimes be assisted by a sensitiveness of the cranium to manipulation, as upon percussion. In this connection may be mentioned the general thickening, with induration and adherence to the bones of the dura mater, in old people, the chronic insane, etc. These changes are often found in individuals who during life presented no symptoms to cause suspicion of such a condition. This alteration of the dura is possibly due to degenerative changes rather than to any process that can properly be called inflammation.

The extension of an inflammation to the dura mater from wounds which have penetrated through the bones of the skull (fractures, perforating wounds, etc.) is so obvious as only to require mention. The symptoms of such an inflammation (which is circumscribed, not diffuse) are lost amidst those of the primary injury. Their consideration may be properly referred to the surgeon.

Caries of the bones of the skull, even where the inner table is not involved, and especially where there is a syphilitic infection of the system, may involve the outer layer of the dura through the vessels common to the bone and the membrane. Especially is this the case when the petrous portion of the temporal is necrosed. Here the attachment of the dura to the bone is so close at certain points, so thin a lamella of bone separates the cranial from the tympanic cavity, the

vessels pass so directly from one to the other, serving as bridges to a morbid process, that the frequent involvement of the dura at this point is easily explained. Even in cases where the cranial bones themselves are not diseased the inflammation of the soft tissues covering them may extend to the outer layer of the dura. This we see in erysipelas, in inflammation of the mucous membrane of the ethmoid bone, the frontal sinus, etc.

Inflammation of the outer layer of the dura mater may cause simply a chronic thickening of the membrane at the spot involved. In these thickened portions there is sometimes set up a formation of bone, as of spiculæ, laminæ, or nodules.

Sometimes, unfortunately, either primarily or succeeding to the process of thickening, there is formation of pus. This is rarely confined to the outer layer of the dura, but involves also the inner, and upon the surface of this layer pus may be formed. Such an extension of the inflammation may, and very often does, involve the pia-arachnoid, and we then have a leptomeningitis with its accompanying symptoms.

TREATMENT.—As has been indicated, it is only in exceptional cases that the diagnosis of external pachymeningitis can be made with any degree of certainty. If a collection of pus is diagnosed and located, the trephine must of course be resorted to, and the sooner the better. If the conclusion is arrived at that the symptoms of the case (headache, etc.) depend upon an inflammatory thickening of the membrane, with hypertrophy of the adjacent bone, internal medication should be tried. Mercury may be used, preferably by inunction, to the verge of salivation. Iodide of potassium pushed to large doses and continued for some time, has seemed to the writer to give good results in some cases. Counter-irritation by means of the Paquelin cautery is to be strongly advised. The point of the cautery, brought to a high heat, should be scored lightly and repeatedly over the skin of the back of the neck or mastoid processes, causing a slight scorching of the epidermis without the after-production of sores. This should be repeated from time to time. The trephine should not be regarded exclusively as "a last resort." There is little danger in its application: it helps to clear up, the diagnosis, and sometimes the removal of a button of bone in these cases is followed by great relief to persistent headache, and would appear to exercise a salutary influence on the progress of the morbid process.

We know but little of an inflammation of the inner layer of the dura mater, except where it has extended from the outer layer, as in cases such as have been described above. If it is involved in a purulent inflammation, this will extend to the pia-arachnoid, and we will have a leptomeningitis. Or if there is no formation of pus, the inner layer may form adhesions to the pia-arachnoid, the membranes being glued together, and they may become adherent to the brain. Not very infrequently there is a formation of spiculæ or plates of bone in the inner layer of the dura: this is seen very often in the falx cerebri.

INTERNAL PACHYMENINGITIS.

Pachymeningitis Hæmorrhagica Interna; Hæmatoma Duræ Matris.

This affection consists in the formation between the dura mater and pia-arachnoid of fibrous tissue arranged as membranes of various thickness and extent. Sometimes there is but a thin cobweb of fibres, easily stripped in shreds from the inner surface of the dura. This imperfectly organized membrane shows many minute hemorrhages and rust-colored points, the result of changes in the coloring matter of older blood extravasations. In a more advanced stage there are layers of thicker and more definite membrane superposed and connected with each other, firmly attached to the dura, and sometimes it may be to the pia-arachnoid. Of these layers the older ones next the dura are the densest and best organized. Imperfectly formed vessels with very thin walls, of greater calibre than capillaries, are seen throughout the new tissue, and hemorrhagic points of greater or less size are scattered through it. Sometimes hemorrhages occur between the membranous layers, and these may acquire such dimensions as to give to the new formation the appearance of a blood-filled cyst, a hæmatoma.

The name internal pachymeningitis implies an inflammation of the inner layer of the dura mater, and formerly it was the almost universally accepted view of the nature and origin of this affection. Many, however, amongst them notably Hugenin, have advanced another explanation, and one which appears to be generally adopted. According to this latter view, the starting-point of the new tissue formation is the occurrence of a hemorrhage between the dura and the pia-arachnoid. Observations which have followed step by step the organization of the blood clot, the subsequent hemorrhages leading to the formation of new layers, and also the behavior of blood injected into the subarachnoid space of animals (rabbits), would seem to give the question a definite settlement. The initial hemorrhage comes almost always from the dura, which, however, shows no congestion or inflammation, its lining epithelium keeping its natural appearance up to an advanced stage of the affection. The conditions leading to the hemorrhage are not always obvious, but in the majority of cases there is probably a degenerated condition of the veins and arteries, combined with such an atrophic condition of the hemispheres as leaves these vessels without their natural support from the surrounding tissue, thus facilitating their congestion. Thus, pachymeningitis is frequently found in dementia paralytica, dementia senilis, chronic alcoholism, the brains of old people, in all of which there is a diminution of the bulk of the hemispheres and an œdematous condition of the pia-arachnoid—hydrocephalus ex vacuo.

The location of the pachymeningitic formation is generally under the parietal bone, oftenest on both sides of the brain. Sometimes it occupies only a limited extent at the vertex, sometimes it is spread cap-like over both hemispheres, and sometimes it is found in the middle and posterior fossæ of the skull.

It is found in very varying morbid conditions. Thus it has been

observed in pernicious anæmia, leucæmia, phthisis, chronic venereal disease, disease of the heart, etc. Sometimes it is the result of an injury, and sometimes, it must be admitted, it occurs without any ostensible exciting cause. Although more especially a condition accompanying senile changes in the tissues, many cases have been recorded where it existed in children. The view has been advanced that the affection is one of trophic disturbance (Dercum).

A diseased condition occurring under such different circumstances and running its course in so varied a manner must exhibit a very varied display of symptoms. Very often no symptoms at all reveal its existence, and its presence is discovered only by a post-mortem examination.

The essential nature of the affection being a subdural hemorrhage with subsequent organization of the clot, concomitant circumstances will have a great effect in modifying its course and symptomatology. In cases of chronic insanity or chronic alcoholism, where the intracranial space is partially occupied by fluid which has taken the place left by the shrunken brain, considerable hemorrhage may occur or numerous layers of membrane be successively formed without exciting such pressure on the brain as to cause recognizable symptoms. There may be headache, dizziness, etc., indicating intracranial trouble, but these have no distinctive reference to pachymeningitis. When pachymeningitis gives rise to symptoms, they will be those of compression or irritation, or a combination of both. A sudden hemorrhage may occur at the inner surface or in the midst of the pachymeningitic tissue, and rapidly cause death by compression. In this case it may be impossible to distinguish the condition from an intracerebral hemorrhage. When an attack of convulsive movements, especially if they are one-sided, is followed by coma that may last "days and weeks," emerging from which the patient shows paralysis or weakness of one side, the symptoms point strongly to pachymeningitis. Inasmuch as the morbid process takes place with greater frequency over the motor region of the brain, hemiparetic, or hemispasmodic, or aphasic symptoms will be most likely to be present when sudden hemorrhage or the successively formed membranes cause pressure on the cortex. Sometimes the picture is complicated by paresis of the limbs on one side and spasmodic contractions on the other. The condition of the pupil varies, being sometimes contracted at first, and afterward dilated or of unequal size on the two sides. There may be choked disk from effusion of blood in the sheath of the optic nerve. Generally the nerves at the base of the brain are not affected. The disease, as stated, may prove rapidly fatal, but it may again run a chronic course for years, the symptoms, as one would expect from the nature of the affection, showing rapid and frequent exacerbations, with subsequent remissions, as hemorrhages occur and are subsequently absorbed or the brain accommodates itself to its narrower limits. Occasionally pachymeningitis is cured—that is, the morbid process comes to a standstill, the effused blood is absorbed, and the membranes already formed become thin and shrunken.

Little can be expected from treatment except in cases where the diagnosis and location of a hemorrhage have been made out (*e. g.* from focal symptoms), when the aid of the surgeon may be called in with

some hope of relief. Counter-irritation, as advised in pachymeningitis externa, should be tried. Internal medication gives little chance of relief, and we must content ourselves with enforcing a quiet routine of life, free from excitement, and a non-stimulating diet, as the best means of diminishing the tendency to renewed hemorrhages.

Diffuse inflammation of the dura from syphilitic infection belongs to the account of that disease (see Vol. I. p. 884).

AFFECTIONS OF THE PIA MATER AND ARACHNOID.

Structure.—Beneath the dura mater the brain and spinal cord are invested by the pia-arachnoid membrane. This consists of a membrane the two surfaces of which are condensed into laminæ, these laminæ being connected together by bundles of fine fibrous tissue, thus leaving spaces of various sizes which communicate with each other, and which contain the cerebro-spinal fluid; this is the subarachnoid space. The outer condensed layer, usually described as a separate membrane under the name of "arachnoid membrane," invests the brain without closely following its inequalities, but passing from convolution to convolution, from hemispheres to cerebellum and medulla oblongata, without dipping into the sulci and fissures separating these different parts. It is made up of a meshwork of fibres and is non-vascular. The glandulæ Pachioni are projections from the arachnoid situated on each side of the falx cerebri. They push up to the inner surface of the cranial bones, not penetrating the dura mater, but carrying it before them, and make pits more or less deep in these bones, sometimes indeed penetrating through their thickness. They invade the dura at points where it is divided into two layers, between which are the "blood lakes" which empty directly into the sinus. They are important agents in transmitting the cerebro-spinal fluid from the subarachnoid space into the lymphatic circulation. The inner layer of the pia-arachnoid, commonly called the "pia mater," is closely applied to the surface of the brain, following all its inequalities, dipping into the sulci between the convolutions of the hemispheres, and adhering intimately to the cerebellum, pons, and medulla oblongata. These two layers of the pia-arachnoid do not maintain an equal distance from each other at all points. In some situations they are widely separated, and thus leave large spaces, "cisternæ arachnoidales," which are filled with cerebro-spinal fluid. The largest of these is at the base of the brain. Thus we may regard the pia-arachnoid as a spongy membrane filled with fluid. The spaces between its surfaces are lymph-spaces lined with endothelial cells, probably exercising a function connected with the secretion and absorption of the fluid in its meshes, the cerebro-spinal fluid, which may be regarded as lymph. Moreover, this internal layer of the pia-arachnoid (pia mater) sends a process through the great transverse fissure of the brain which roofs over the third ventricle (tela choroidea) and is prolonged into the descending horns of the lateral ventricle. Along the margin of this involuted portion are the choroid plexuses, villous projections which are highly vascular. They are made up of a fringe of convoluted vessels, the free surfaces

of which are covered with a layer of cubical epithelium, reminding us of the arrangement of vessels and cells in the glomerulus of the kidney. They have doubtless important functions connected with the secretion of the ventricular fluid, and may serve as regulators of the cerebral circulation, balancing the amount of intracranial blood-pressure by the amount of fluid in the ventricles. The fluid in the ventricles communicates with the fluid in the subarachnoid space by a small opening (the foramen of Magendie) at the point and two slits at the sides of the fourth ventricle. This communication is of great importance in view of certain diseased conditions of the brain and its membranes in which these openings may be occluded. The pia mater (inner layer of the pia-arachnoid) is very vascular, but by far the greater portion of the blood in its vessels is destined for the brain. The arteries are supported in the meshes of its tissue, where they divide and subdivide until they are diminished almost to the size of capillaries ("terminal arteries"), when they plunge directly into the cerebral tissue.

Anæmia and Hyperæmia of Pia Mater.

From the description just given we see that the vascular connections of the pia mater and brain are so intimate that the blood supply of one must closely correspond with that of the other, and that when we speak of the anæmia or hyperæmia of the pia mater it is but another name for the same conditions of the brain; nevertheless, they will be treated of, rather to make complete the pathology of the membranes of the brain than because we look upon such states as pathological entities.

That an increase or diminution in the size and fulness of the vessels of the pia mater takes place under various influences, such as modifications of heart action, changes in the respiratory activity, vasomotor influences, etc., of course is known. That these variations of the blood supply should sometimes transcend the normal bounds and become pathological the analogy from other organs would certainly lead one to suppose. But one encounters great difficulty in obtaining evidence of such pathological conditions after death, so as to intelligently connect them with symptoms occurring during life. In short, one cannot get at the pathological anatomy of anæmia and hyperæmia of the pia mater, or, at best, it is understood in a very imperfect manner. This depends on several circumstances, principally on the fact that the weakened propelling force of the heart and its final cessation allow the minute vessels to be compressed and emptied by the recoil of the swollen, elastic tissues surrounding them, thus entirely changing the vascular condition from what it was intra vitam. One cannot but suppose, however, that a condition of anæmia of the pia mater (the brain) results from certain conditions, and that certain symptoms which appear have their origin in it. Anæmia of the pia mater (and brain) may be caused by great loss of blood; by the unequal distribution of the blood, as when through vasomotor influence a large amount is diverted into the abdominal cavity; by weakened action of the heart, as in fainting; by contraction of the arterioles when the vasoconstrictor nerves are stimulated, etc. It may be also that in cachexiæ, when there is a diminution of the volume of the blood, there exists a chronic anæmia of the pia, with a corresponding diminution of

blood in the cerebrum. The symptoms connected with anæmia of the pia mater are those of diminution or loss of cerebral functions. Vision fails; "everything looks black before the eyes;" there are ringing in the ears, nausea, confusion of thought, and finally unconsciousness. These symptoms are combated by whatever tends to send the blood to the brain with increased force, as lowered position of the head, stimulation of the heart's action, etc. Those remedies are also to be employed which, acting on the vasomotor nerves, inhibit the vasoconstrictor centre and allow dilation of the vessels, such as nitroglycerine and nitrite of amyl. This is probably the mode of action of the popular means to prevent fainting, as when the skin is sharply stimulated or ammonia is applied to the nose the impression inhibits the vasoconstrictor centre. Cerebral anæmia from general diseased conditions will be best treated by the remedies appropriate for such conditions. The complex of symptoms called "hydrocephaloid" (Marshall Hall) observed in young children as the result of profuse discharge from the bowels, in which there are symptoms of profound cerebral depression, would seem from the sunken fontanelles and general condition of exhaustion to be one of anæmia of the brain and its membranes.

We recognize two conditions of hyperæmia of the pia mater—(1) the active and (2) the passive.

(1) **Active or Fluxionary Hyperæmia.**—It has long been a familiar idea that the brain and its membranes may be in a state of congestion which is pathological, and to this state many cerebral symptoms have been referred without sufficient grounds. At present probably such a congestion is too much ignored as a pathological factor. It is true that the symptoms of cerebral congestion are somewhat deceptive, and, while impressing the patient and others with the conviction of "too much blood in the head," a careful consideration of them leaves considerable doubt as to a real pathological over-fulness of the arteries. Thus the usual signs, a flushed face and throbbing arteries, are not conclusive proof of an over-distention of the intracranial vessels, but such a condition of the external circulation may be in absolute contrast to that within the cranium. All allowance for objections, however, being made, it would seem from observation and experience that there may be an active hyperæmia of the pia and brain so exaggerated as to give rise to symptoms of more or less gravity.[1] Such a condition is probably oftenest the effect of vasomotor disturbance, and, as there are individuals notably predisposed and liable to it, so we may suppose that these have some defect by which the balance of the vascular nervous system is readily disturbed, giving rise to abnormal fulness of the intracranial vessels.

Although certain individuals, the so-called "plethoric" (and the condition of "plethora" is doubtful), are more liable to cerebral congestion than others, theirs is not a constant condition of hyperæmia. Indeed, permanent arterial hyperæmia probably never exists, such hyperæmia being in its nature a transient affection. There are various causes as-

[1] A number of cases of "pseudo-meningitis" (Krannhals) have been recorded in which symptoms closely resembling meningitis terminated in death, and in which a post-mortem examination showed only great hyperæmia, œdema, and hemorrhages in the pia-arachnoid, without a trace of inflammation.

signed as giving rise to this condition. Over-action of the heart is one, but it would seem incompetent unless there is at the same time a paralysis of the vessels of the pia; sudden and extensive contraction of arteries elsewhere in the body; violent emotional excitement, the arrest of the menstrual flow, sunstroke, high temperature of fever, certain poisons,—all of these probably act upon the vessels through the vasomotor centres. Idiopathic cases of fluxionary hyperæmia very rarely, if ever, occur.

The symptoms most reasonably connected with cerebral hyperæmia or congestion are headache, photophobia, abnormal sensitiveness to sounds, singing in the ears, giddiness, torpor or confusion of mind, which may increase to delirium. The patient's face and head are flushed and hot, the eyes are suffused, the pupils contracted, and the carotids and temporals throb. After a longer or shorter time these symptoms pass off and appear to leave no injurious after-effects. In young children, in whom the conditions are more favorable for the occurrence of arterial hyperæmia, and in whom the central nervous system (with the vasomotor centres) is much more impressionable, there is oftener recognizable fluxionary hyperæmia, the symptoms of which closely resemble those of beginning meningitis. Rigidity and tremor of the limbs and convulsions may occur. Such a condition, with all its apparent gravity, may pass off in a few hours, leaving no permanent injury. One thing seems almost certain—that cases of acute congestion are transient and cause no organic lesion of the brain, such as would occasion paralysis, although this was at one time the opinion. It is obvious how readily such transitory conditions of abnormal cerebral circulation may be associated with the unstable condition of the nervous system in neurasthenics and the hysterical.

(2) **Passive hyperæmia** has more or less of a chronic character, and consists in the continued over-fulness of the veins of the pia mater. This is brought about by everything that interferes with the return of blood to the heart through the large veins of the neck. Tumors of all kinds which interfere by compression with these veins, habitual depending position of the head, compression of the contents of the thorax as in straining and coughing, emphysema, capillary bronchitis, disease of the heart preventing free return of blood from the lungs,—all can, in a greater or less degree, cause a passive hyperæmia. If the passive congestion is slight or its cause not continuous, the condition is marked by headache, sluggishness of intellect, drowsiness, giddiness, etc.—symptoms which are aggravated by everything that increases the obstacle to the blood flow. If the conditions causing the congestion are more pronounced and continuous, the symptoms increase in gravity. There is delirium, there may be epileptiform convulsions, and the stupor deepens into coma. The gravity of the prognosis is in proportion to the gravity and the persistency of the cause.

TREATMENT.—For the so-called "plethoric" and those who are liable to sudden flushes of cerebral hyperæmia a constant guard is to be kept against excitement, over-exertion, stimulating ingesta, overloading the stomach, constipation, and everything which unduly excites the heart's action and disturbs the nervous balance of the system. At the same time, an equalization of the circulation by regular and judicious

exercise is very important. Of internal remedies the bromides are of prime value, probably for their power of correcting the irregular or ataxic condition of the vasomotor centres. Ergot seems to have a good effect, although its action is not satisfactorily explained. During an attack of fluxionary hyperæmia all therapeutic efforts must be made in the direction of causing a contraction of the intracranial arteries. For this purpose are used cold to the head and neck, hot baths for the feet, sinapisms and cups to the back of the neck, leeches to the temples and mastoid processes, purgation; all of which remedies probably influence the hyperæmia, not by "drawing away blood from the brain," but by acting on the vasomotor centres and so causing contraction of vessels. Passive hyperæmia can be successfully treated only by removing or alleviating its cause.

Leptomeningitis.

Inflammation of the Pia-arachnoid; Meningitis.

Cerebro-spinal meningitis, an infectious disease in which an inflammation involves the pia mater of both the brain and spinal cord, and which occurs as an epidemic, and tuberculous meningitis, an inflammation of the membrane caused by the invasion of the tubercle bacillus, are treated of elsewhere in this work. (See Vol. I. pp. 425 and 755.) Leptomeningitis is most frequently caused by an extension to the pia-arachnoid of an inflammation from adjoining parts. Thus in wounds of the soft parts and bones of the cranium infectious agents (streptococcus pyogenes) from without cause an inflammation which makes its way through the vessels of the diploë to the membranes of the brain within, giving rise to a local or diffused leptomeningitis. In caries of the bones of the cranium there is a frequent cause of meningitis, the inflammation extending to the pia, as has just been said, through vessels (vasa emmisarii) common to the exterior and interior of the skull and through the veins of the diploë. Of all of the cranial bones, the temporal is the one the disease of which most readily and frequently causes meningitis; and this is readily enough explained, first, by the fact that the petrous portion is so frequently the seat of caries, and, secondly, when one recalls the close connection of the dura and its sinuses with it by means of vessels, prolongations of tissue, and the passage of nerves. The tympanic cavity (so frequently the seat of disease) is separated from the dura by but a thin lamina of bone, and the vessels passing through the petro-squamous fissure present another ready way for the passage of inflammation from without to the interior of the cranium. Without caries of the bone an otitis media may set up a leptomeningitis by causing a sinus thrombus in which may occur purulent changes which infect the pia mater. In this case the extension of the inflammation to the sinus is through the diploic veins of the petrous portion of the temporal. Inflammation may extend to the pia-arachnoid from the lining membrane of the mastoid cells, from the frontal sinuses, rarely from the nasal cavities, and more rarely still a leptomeningitis may result from a panophthalmitis. The bursting of an abscess of the brain upon the cerebral surface will light up a meningitis. In all of these conditions there is

but little difficulty in understanding how the inflammation takes place—that is, how pus-forming organisms reach the points within the cranial box where the inflammation is set up. In meningitis from other causes this is not so obvious. We will return to this point later on. Blows upon the head, especially in children, may give rise to a hyperæmia which, according to Henoch, sometimes develops into leptomeningitis. Erysipelas of the face and scalp may be the cause of meningitis, although the steps of the causation are difficult to see. Sunstroke is affirmed by some to be a cause of meningitis. In insolation there is certainly every evidence of great arterial hyperæmia, and it is somewhat difficult to draw a sharp line between such hyperæmia and inflammation. One cannot suppose that mental excitement and exertion, although claimed by some to be a cause of meningitis, can do more than act as a predisposing factor.

As a secondary affection leptomeningitis occurs in the course of a number of acute infectious diseases. It is seen probably oftenest in pneumonia. It has also occurred in smallpox, typhoid fever, scarlet fever, measles, influenza, "grippe," ulcerative endocarditis, acute rheumatism, Bright's disease, and other diseases. When meningitis develops in the course of a disease, it is caused by the invasion of the pia mater by the infecting agent of the disease (micro-organisms and toxins) by way of the blood and lymph. Besides these, cases of leptomeningitis occur without discoverable cause, which may provisionally be called "idiopathic." They have been observed most frequently in children, the male sex being oftenest affected. One cannot but suppose an infectious cause for such cases, probably similar to that which causes epidemic cerebro-spinal meningitis. This seems the more likely as such cases of "idiopathic" meningitis have been seen to occur in close relation of time and place to each other—"miniature epidemics."

Leptomeningitis may be diffuse, involving the whole surface of the brain, or it may be confined to a particular locality. Names have been applied to these varieties which probably give an erroneous idea of a fundamental distinction amongst them which does not exist. Thus there are meningitis of the convexity or vertical meningitis, meningitis of the base or basal meningitis, but these run into each other, and, although their symptoms necessarily differ, the diseased condition is essentially the same, and the location is, as it were, an accident. In some cases the location depends upon the cause, as in limited meningitis originating in otorrhœa or caries of the petrous portion of the temporal bone. There is a variety of meningitis confined to the ventricles. This is mostly seen in children.

In cerebral meningitis the inflammation often extends partially to the membranes of the cord.

Pathology and Pathological Anatomy.—The varied conditions under which meningitis occurs modify in a not inconsiderable degree the character and amount of the pathological changes found after death. In cases of extension of the inflammation from wounds, necrotic bones, or neighboring purulent processes, there is usually found a well-characterized purulent meningitis. The subarachnoid fluid is turbid, pus may appear only in whitish streaks on the borders of the veins in lighter cases, but in the more virulent it accumulates in the sulci between the

convolutions and in the subarachnoid spaces at the base of the brain, and may be in such amount that the vessels are hidden from view by a greenish-yellow layer or fibro-purulent deposit. The meshes of the pia-arachnoid may be infiltrated and stiffened with the sero-purulent effusion, the membrane being greatly thickened. The intracranial portions of the nerves may be involved in the inflammatory process and show points of hemorrhage. This is especially seen in the acoustic and facial nerves in cases where the petrous portion of the temporal bone is involved in caries. In meningitis occurring as secondary to general infectious diseases (pneumonia, etc.) the macroscopic changes may be much less marked, and there may be only a slightly turbid subarachnoid effusion, with hyperæmia and cloudiness of the pia mater, and, it may be, here and there flecks of pus.

With the inflamed condition of the cortical pia mater there is often associated an effusion in the ventricles, moderate in amount, usually turbid, sometimes purulent, and rarely serous in appearance. This effusion occasionally occurs in considerable amount. The walls of the ventricles exhibit a rough surface, the result of an inflammation of the ependyma, and the choroid plexuses show evidence of being also involved in the morbid process. The brain, as one might expect from its close vascular connection with the pia, does not escape without injury in leptomeningitis. When there is a great effusion in the subarachnoid space the gyri are flattened from its pressure, and the substance of the cortex shows various alterations—points of hemorrhage, softening, and pus. Sometimes a layer of pus covers the inner surface of the dura mater. The cortex of the hemispheres sometimes looks pale and anæmic from the pressure made upon it by a large ventricular effusion. There is a variety of leptomeningitis called serous meningitis (Quincke) in which there is little inflammatory exudation in the subarachnoid space, the effusion of the serous fluid in the ventricles being the distinctive pathological change found. This ventricular effusion is free from pus, clear, and like the cerebro-spinal fluid in appearance, and is in much greater quantity than the fluid ordinarily found in the ventricles in meningitis. In serous meningitis the pia mater presents simply a hyperæmia or, more rarely, a cloudiness.

Another form of meningitis is "primary chronic meningitis." In this are found thickening and adherence together of the membranes, and an attachment of them to the brain. The cranial nerves are involved with the thickened membranes, and hydrocephalus may result from the occlusion of the ventricular outlets caused by the inflammatory process. In children a chronic basilar meningitis occupying the posterior fossa of the skull sometimes occurs. There is great thickening of the membranes, which are agglutinated together and adherent to the cerebellum, pons, and medulla oblongata. These two last forms of meningitis are very frequently, if not always, the result of syphilitic infection.

Lastly, cases occur with all the symptoms of acute leptomeningitis, terminating, too, in death, in which the post-mortem examination reveals no pus, no effusion, or other signs of inflammation, nor, indeed, any changes that can be reasonably considered as having caused the fatal termination —"pseudo-meningitis." The changes in the meninges that occur in

cases of chronic alcoholism, and which are sometimes designated chronic meningitis, can hardly be considered as the result of an inflammation, but rather as the degenerative tissue changes brought about by the toxic effects of alcohol.

Purulent meningitis is an infectious disease, and the channels through which the infecting organisms reach the cerebral meninges are, in most cases, obvious. Thus, in meningitis following injuries of the head, caries of the temporal and other cranial bones, inflammation of the cavities about the head (the antrum, frontal and sphenoidal sinuses, etc.), the invasion of pyogenic organisms (streptococcus, staphylococcus pyogenes) may readily be accomplished from these contiguous structures; and, indeed, these microbes have frequently been found in the meningitic exudation. In the meningitis occurring in the course of general diseases the toxic micro-organisms peculiar to such diseases, or it may be soluble poisons derived from them, find their way to the pia-arachnoid through the blood and lymph streams. The microbe that has been found most frequently in the inflammatory products of meningitis is the pneumococcus (micrococcus lanceolatus), and this not only in the meningitis occurring in the course of a pneumonia, but also in the majority of cases of associated meningitis. The typhoid bacillus, the bacterium coli communis (Osler), and many other micro-organisms have been found in meningitis. Nevertheless, it is probably going too far to say, with our present knowledge of the subject, that all cases of meningitis are parasitic in origin. While it is indeed true that "cortical meningitis is predominantly parasitic," it would appear that an inflammatory ventricular effusion may occur without the toxic action of micro-organisms. This is claimed by Quincke for the ventricular fluid in serous meningitis. Even in such cases, however, the possibility of the influence of a general infection is not excluded.

Symptoms.—Almost all of the organs of the body are to a greater or less extent the subjects of direct observation, but the brain and its membranes are withdrawn almost entirely from such investigation of their condition. Percussion of the cranial walls may sometimes elicit pain, as in inflammation of the dura mater, and the fundus of the eye may present changes connected closely with intracranial pressure; but, for the most part, with exception of tumors and focal lesions, a knowledge of the condition of the organs and tissues within the skull must be gathered from general symptoms. But the symptoms which are caused by organic pathological changes in the brain and its membranes may be so simulated by general or local diseased conditions which excite merely functional brain disturbance without tissue change that much difficulty and confusion may be encountered in a diagnosis. It not unfrequently happens that in the cases of meningitis occurring in the course of other diseases the symptoms of the primary disease so mask those of the meningitis that the latter escape observation, and the proof of an existing meningitis is only discovered at the post-mortem examination. Moreover, cases of extensive purulent meningitis have been known to run their course almost without symptoms. Meningitis is a disease so varied in its cerebral location, its intensity, duration, and pathological changes, that the complex of symptoms connected with it varies within very wide limits. Thus, there are no one

or two symptoms so characteristic that their presence can fix the diagnosis of meningitis. One must carefully consider the general bearing of all of them that go to make up the picture, and endeavor to eliminate those which are caused by other morbid conditions than those of the disease. There are, however, certain symptoms which are very constant, and, although under certain circumstances they may be modified or simulated, they can be considered as the distinguishing indications of meningitis.

A very constant and prominent symptom of meningitis is headache. It is usually very severe, continued, and increasing with the progress of the disease. Evidence of it is given by the patient even in sleep and in delirium by the pained and frowning facial expression, by the movement of the hands to the head, and in young children by cries and an almost automatic movement of the head from side to side. Often there are giddiness and confusion of mind; and delirium, sometimes violent, makes its appearance early in the disease; it may alternate with somnolence. Painful sensitiveness to light and sound is commonly observed, the patient starting and shrinking at loud noises even in sleep or partial stupor. There is hyperæsthesia of the skin and muscles. These symptoms of nervous irritation and excitement yield to those of depression, and stupor supervenes. Among the earlier symptoms, but by no means a constant one, is cerebral vomiting—that is, vomiting which occurs with little nausea and independent of irritating substances or ingesta in the stomach. A sudden invasion of purulent meningitis is often ushered in by a chill, and indeed chills may in some cases recur during the progress of the disease. To this succeeds fever, which has nothing characteristic in its course except it may be its irregularity. It is by no means necessarily high, though it sometimes rises to 104–105°, probably corresponding with the extent and violence of the inflammation. In rare instances the temperature has not risen above normal, and has even been recorded as subnormal in some cases. The higher temperatures generally prevail in young children, as is usual in their diseases. In the course of diseases in which meningitis occurs secondarily a sudden high rise of temperature, unaccounted for by the course of the primary disease, may mark its commencement. The agonal temperature may rise very high.

The pulse rate may be rapid throughout the disease, but often it is notably not in accord with the temperature. With high temperature the pulse may be remarkably slow, sometimes being reduced to 50 or 40 beats per minute; not unfrequently it is irregular, slowness and rapidity alternating.

The motor system shows great variety and different degrees of derangement. Sometimes there are general convulsive seizures of a violent character, like epilepsy. These occur more commonly in young children. The convulsive movements may be confined to spasmodic movements of the muscles of the face and mouth or of the extremities, grinding the teeth, and at times a tremulous rigidity of the limbs. A characteristic contraction may be observed in the lower limbs when the patient is placed in the sitting posture and a movement of flexion on the pelvis is impressed on the thigh. If while doing this one endeavors to extend the leg on the thigh, the motion is resisted by a contraction

of the flexors on the back of the thigh; but if the patient be placed in the prone position, this contraction disappears and there is no resistance to extending the leg. Stiffness and contraction of the muscles of the back of the neck, made evident when present by an attempt to bend the head forward, are the most characteristic symptoms of the disease, and especially of basilar meningitis; indeed, this retraction of the head may in some cases be the principal and almost the only sign of meningitis. Sometimes the retraction is to one side (Hennoch). The abdominal walls are rigid and retracted. When the meningitis is mostly at the base of the brain, the cranial nerves, as one would anticipate, are involved in the inflammation or compressed by the exudation, and thus give rise to ocular and other symptoms. Strabismus is a frequent symptom. There may be ptosis. The pupils are variously affected. At first, from irritation of the third nerves, they are contracted; afterward, as the nerves are paralyzed by compression, they are expanded and immovable. The pupils may be of unequal size, and this condition may alternate in the two eyes from time to time. If the seventh nerve becomes involved, twitching of the facial muscles, as mentioned above, occurs. If the involvement of this nerve trunk is more complete, as in meningitis with caries of the temporal bone, the face is paralyzed, and if the patient survives long enough the muscles will show the degenerative reaction, as in ordinary Bell's paralysis. A facial paralysis may also occur with meningitis of the convexity, but in such cases it is (as in hemiplegia) only the muscles below the orbit that are paralyzed, and they do not exhibit the reaction of degeneration.

Optic neuritis and venous congestion of the optic papilla do not always occur, but when present are very distinctive symptoms of a meningitis involving the optic nerve and making pressure upon its vessels. Nevertheless, optic neuritis may be present in meningitis where there is no pressure on the optic nerve. Blindness may supervene. The effect of the inflammation of the pia-arachnoid on the subjacent motor centres of the cortex is either to excite them, causing spasm or contraction of one or both limbs, or of the face on the opposite side, or, in case their function is destroyed, hemi- or monoplegia. The respiration is hurried, especially in children, irregular, sighing, with long pauses. Cheyne-Stokes respiration is not seldom observed, although it is less frequent than in tubercular meningitis. It always ceases before death. As a rule, the bowels are constipated. In the end deep coma, with loss of the reflexes, precedes death.

It is obvious that individual symptoms may be absent or combined in great variety according to the intensity of intracranial location of the meningitic inflammation, thus giving rise to very unlike symptom complexes.

In *serous meningitis* (Quincke) the above symptoms are more or less modified. Its invasion is more gradual. The fever does not run so high, seldom rising over 102° F., and indeed, in some cases it may be absent. The headache and stiffness of the neck are not so pronounced as in purulent meningitis, nor does delirium make its appearance so early nor is it so distinctly marked. The symptom of choked disks is more frequent in serous meningitis, and in some cases it would appear to be the only noticeable one. Irregularity in appearance and variation in

the severity of the symptoms which occur from time to time in the course of serous meningitis probably depend upon the alternate increase and absorption of the ventricular fluid, and the well-known tolerance to pressure which the brain acquires within a short time.

Diagnosis.—So many of the general symptoms of meningitis are present in other diseased conditions that we must be careful in making a diagnosis not to lay too exclusive a stress on any one (as headache or convulsions, for example), but to study the picture made up by them all, with a careful examination of the causes to which they may owe their origin. Again, too, we must avoid keeping in view too typical a picture of the disease, remembering that, as has been already said, the combinations of its symptoms are very varied, and that there is no one symptom the absence of which would exclude a diagnosis of meningitis. These considerations refer to the diagnosis of the disease in its earlier stages, for of course toward its end symptoms dependent on the local lesions it has inflicted are generally too obvious to be mistaken. In the course of many general diseases (typhoid and the eruptive fevers, pneumonia, etc.) the symptoms caused by the disease, such as headache, temperature, vomiting, delirium, may resemble with confusing closeness those of meningitis. Convulsions and a condition of stupor are often seen in certain nervously irritable children to accompany almost every pyrexia, but passing off without after-effects. When, however, the headache persists during sleep (as may be evidenced by the expression of the face and the movements of the patient) and during delirium, it is probable that it is a symptom produced by meningitis. If with such a headache the temperature is irregular, it strengthens the evidence for meningitis. If with a high temperature the pulse does not show a corresponding rapidity, but is slow, it is a further and strong proof in favor of the diagnosis. If the convulsion is unilateral, if there is spasm, stiffness, or tremor confined to the muscles of one limb or one side of the face, it speaks in favor of meningitis. If with this the muscles of the back of the neck are rigid, it would make the diagnosis fairly complete. But in the endeavor to make a differential diagnosis between meningitis and a general disease it is important that we should not confine our attention too exclusively to the meningitic symptoms, but closely investigate those which belong to the lesions of other organs and which are indicative of other diseases. Thus in typhoid the regular variations of temperature, the local abdominal tenderness, the rose spots, etc. will serve to distinguish it. In pneumonia an examination of the lungs will probably clear up the diagnosis.

For septicæmia we must look carefully into the history of the case, examine for deposits of pus or for some point from which septic poison may have invaded the system. Uræmic poisoning may sometimes simulate closely meningitis. Indeed, we might expect similar manifestations when the so-called "uræmic poison" invades the very part, the brain cortex, which is involved in meningitis. Examination of the urine or the existence of dropsical effusions will assist us in coming to a conclusion.

An acute purulent otitis, especially in children, may present all the more prominent indications of meningitis. There may be violent headache, delirium, giddiness, convulsions, and, in some cases, even strabis-

mus and optic neuritis, and yet upon a free discharge of pus from the ear all the symptoms pass away, leaving us to solve the somewhat difficult problem of their causation. In such cases, if there are local symptoms, as weakness or paralysis of the lower muscles of the face, spasm or paralysis of a limb, or retraction of the head, we may conclude there is meningitis; but sometimes the diagnosis is impossible. In cases other than those of caries involving the temporal bone, in which the symptom has been occasionally observed, congestion of the optic papilla and optic neuritis speak decidedly for meningitis; so also does inequality of the pupils and strabismus. In fact, the general symptoms are far behind the local in importance in making the diagnosis of leptomeningitis. Hysteria, again, is sometimes liable to be mistaken for meningitis, or the reverse error may be made. In this simulator of diseases the symptoms may present a puzzling resemblance to meningitis. We must, moreover, bear in mind that the existence of the graver disease does not preclude the coexistence of hysteria, especially in the beginning of the attack. In hysteria the patient complains bitterly of headache, but it is scarcely as oppressive as in meningitis; and if we observe carefully, we may notice that at times the patients' attention may be diverted from it; it does not claim their attention as imperatively as in meningitis. In hysteria the vomiting will probably want the true cerebral character, being accompanied with more effort. Stiffness of the neck may be present, but is likely to vary in degree at different times, or, it may be, during the same examination. If there are convulsions due to hysteria, they will most probably not be accompanied with unconsciousness, although it may require close observation and careful examination to make sure of the fact. The symptoms which speak for meningitis as against hysteria are increased temperature—this being rare in hysteria—unequal pupils, divergent strabismus (convergent strabismus often occurs in hysteria), weakness or paralysis of one side of the face, and incontinence of urine. Delirium tremens may sometimes appear at first to simulate meningitis of the convexity, but the previous history and the course of the affection should within a very short time make the diagnosis clear. The "hydrocephaloid" condition, which presents certain symptoms of meningitis, may readily be distinguished by the sunken instead of the projecting fontanelle of meningitis and the history of exhaustive discharges and depressed vitality.

Diagnosis of the Various Forms of Meningitis.—We distinguish various forms of meningitis, which are characterized by their special symptoms, the distinctive course they run, and their termination. Although in meningitis the inflammation very often affects at the same time both the pia-arachnoid of the base and of the convexity, cases occur in which it is confined more or less exclusively to one or other of these regions; and this difference of the location of the disease causes a decided modification of the symptoms. In meningitis of the convexity delirium is likely to begin earlier, and to be more active and pronounced, and from the proximity of the inflammation to the motor region monoplegias and spasm, stiffness or tremor of a single limb are more commonly seen. Occasionally there may be aphasia. A paralysis or weakness which only affects the lower portion of one side of the face is strongly presumptive of meningitis of the convexity of the opposite side. In meningitis of

the base, if there is a facial paralysis, it is complete, the whole trunk of the seventh nerve being involved. The ocular symptoms are prominent. Stiffness of the neck and retraction of the head are marked. Vomiting is more common than in meningitis of the convexity. Limited meningitis may occur from local injuries or disease, as caries of bone, especially the temporal. Severe localized pain is felt at the point affected, and according to its position there may be focal symptoms or those arising from the implication of the adjacent cranial nerves, as, for example, the seventh. An inflammatory effusion at the base may be so limited (as in caries of the temporal bone) as to compress one cerebral peduncle together with the cranial nerves of the same side, thus causing a crossed hemiplegia.

Serous meningitis is most commonly found in young children. It is distinguished from purulent meningitis by a more gradual invasion (though there are exceptions to this) and a slower and more lingering course. The temperature may not be above normal or show but a slight rise. The general symptoms are mainly those of compression and hebetude, rather than those of cerebral irritation, as headache, delirium, etc. Choked disk, owing probably to the pressure of the distended ventricles, is more common than in purulent meningitis. In young children the ventricular effusion is revealed by the enlargement of the head.

To distinguish between *purulent meningitis* and *epidemic cerebro-spinal meningitis* is not always possible. Indeed, we sometimes see minor epidemics of meningitis confined to a household or, it may be, single cases, which in all probability are caused by the specific toxic agent of the epidemic disease. When an epidemic of cerebro-spinal meningitis is prevailing the probability is all in favor that an individual case occurring will belong to that form, or when, on the other hand, there are suppurative lesions of the head, as caries, otitis, etc., we may in making a diagnosis of meningitis exclude the epidemic disease. Otherwise the differential diagnosis will rest on the presence of spinal symptoms, opisthotonos, paraplegia, etc., which, however, are not invariably present in the epidemic disease. Marked herpetic eruptions are indicative of cerebro-spinal meningitis. Sometimes the extreme severity of the symptoms, the fulgurant nature of the attack, and the rapidly fatal course make it probable that the case is one of epidemic meningitis.

A diagnosis between *tuberculous*, *purulent*, or *serous meningitis* cannot always be satisfactorily or certainly made, although it is a matter of great importance, in view of the prognosis, that it should be. First of all, it will be necessary to make a careful examination of all of the organs for indications of tuberculosis and to inquire judiciously into the antecedents of the patient. Tuberculous meningitis occurs most frequently in children, and is more gradual in its invasion, prodromal symptoms generally exhibiting themselves to an observant eye for days or may be weeks, during which time the patient is dull, fretful, and out of health. As the most frequent seat of tuberculous meningitis is the base of the brain, vomiting and ocular symptoms may be expected to occur earlier and be more marked than in the other varieties. Aphasia occurs oftener in tubercular meningitis, as might be looked for from the tendency in that disease for the pus to occupy the Sylvian fissure. With all this, however, it may be impossible to make a diagnosis between a

tubercular and a simple basilar meningitis. As a means of differential diagnosis an examination of the cerebro-spinal fluid has been made. The fluid is obtained by puncture made between the laminæ of the lumbar vertebræ below the end of the cord, such an operation being attended with no danger (Quincke, Lichtheim). If the tubercle bacillus is found, the diagnosis of tubercular meningitis is certain, or if pus and streptococci alone are found, it is extremely probable that we have to do with a purulent meningitis. Unfortunately, in both of these affections the examination may give negative results. Syphilitic meningitis may sometimes run the acute course of the purulent. Its diagnosis must be made by examining the history of the case, and sometimes by the rapidity with which the symptoms clear up by the use of iodide of potassium.

Prognosis.—The prognosis of purulent meningitis is always very grave, although it may be somewhat modified by the particular conditions producing the inflammation. In meningitis resulting from the extension of an infection from carious bone the prognosis may be pronounced all but hopeless, cases of apparent recovery being probably mistakes in diagnosis, such as may be made in middle-ear disease. Of course great violence of the invasion, the rapid advance and prominence of the graver symptoms indicating extensive purulent effusion and consequent disorder, and depression of brain function make the diagnosis almost as grave as possible. On the other hand, a slowness in the advance of the disease, mildness or absence of the graver symptoms (paralysis, convulsions, etc.), a prolonged course without coma, permits a doubtful hope of recovery or of arrest and a chronic continuation of the disease. In judging of the patient's condition from the symptoms we must remember that in the very varied forms of this disease some cases run a course of remarkable latency, with few and unpronounced symptoms, and in which the post-mortem examination reveals extensive pus-formation and hopeless lesions.

The prognosis in serous meningitis, which is almost exclusively an infantile disease, is less grave. The ventricular effusion which is the essential feature of the disease may come to a standstill, or even diminish and disappear. This would seem the only explanation of the cases in which recovery takes place after all the symptoms of meningitis are developed. An important point to keep in mind is, that cases of serous meningitis may be arrested and become chronic, or, indeed, leave the patient free of nearly every indication of the disease, and yet acute exacerbations are apt to occur which prove fatal. Even when the patient lives through an attack of serous meningitis it often leaves disastrous results, and blindness, mental enfeeblement, etc. mark the ineffaceable injuries it has caused to the brain.

Treatment.—If, as too often happens, we are helpless to avert the fatal lesions of meningitis, we can at least use remedies in a reasonable manner to alleviate the sufferings accompanying them. Moreover, when we remember how many deaths occur with every symptom of meningitis, and in which the post-mortem examination discloses no lesion of a necessarily fatal character, we may feel encouraged to employ all the therapeutical agents at our command with careful perseverance. Rest and perfect quiet are to be enforced from the very beginning, even while the prodromal symptoms leave us in doubt as to the real nature

of the disease. The necessity for this is readily appreciated when we reflect that every mental excitement or irritation is accompanied by a flush of blood to the membranes whose hyperæmia is the initial step of the pathological processes we dread.

General bloodletting has always received favorable consideration, especially from the laity. Its indiscriminate use and routine employment are to be deprecated, and its employment confined to robust adults with strongly acting hearts and high arterial tension, and when the disease invades with violence. When used under such circumstances it may stave off the disastrous effects of sudden intracranial pressure and effusion. Local bloodletting by means of cups and leeches admits of more general application. But here also we must be guarded. A weak child may not be able to spare even a little blood in its battle with a depressing disease, and in cases where the hemispheres are compressed against the skull by a ventricular effusion we should not seek to diminish the amount of blood circulating in the brain, for we would but increase the cortical anæmia, the symptoms of which are already too prominent. Leeches to the temples and mastoid processes sometimes seem to do a great deal of good, but this is not accomplished by the amount of blood abstracted, but by an influence exerted by the process of abstraction upon the vasomotor centres, probably causing vascular contraction. Constant and regulated application of ice and cold to the head—being cautious, however, of the depressing effects produced on some patients—is of great value, ameliorating the furious headache, and probably, through the impression made on the cutaneous nerves, reflexly influencing favorably the disordered circulation and morbid processes within the cranium. Shaving the head, or at least cutting the hair very short, a proceeding which is necessitated by the application of ice, is in every case proper, and of itself gives considerable relief. Counter-irritation has been generally recommended, but has appeared to the writer of little benefit in the acute stage of the disease. When used blisters should be of small size and applied behind the ears. Large blisters over the scalp and the extensive painting of the scalp with tincture of iodine are remedies of doubtful benefit, and certainly aggravate the distress of the patient. Some authorities (Quincke), however, recommend maintaining an active purulent discharge from a point on the vertex the size of a dollar by means of tartar-emetic ointment. Warm baths, the temperature of which is regulated somewhat by the feelings of the patient, relieve the headache and seem to exercise a beneficial influence on the progress of the disease. Cold baths are indicated when the temperature runs high, but caution is to be observed in their use, the patient sometimes not bearing them well. Reducing the temperature by drugs exercises no beneficial effect on the disease.

Of internal remedies, mercury is probably the most to be relied on to modify the morbid process constituting the disease, and may be administered by the mouth, hypodermically, or by inunction. The latter mode is to be preferred for rapidity and certainty. However it is exhibited, it should be pushed to the point of "touching the gums"—that is, when the spongy, swollen, and tender condition of the gums indicates that the whole system is under the specific influence of the drug. Iodide of potash may be given in the commencement, but is of

most value in the later stages of the disease, where there is a tendency to chronicity. It should be given in increasing doses until a drachm or more is given every four hours to an adult. There is hardly a limit to the amount that may be given with impunity if the dose is gradually increased.

Headache is generally the symptom most distressing and most difficult to relieve. Ice to the head is the remedy most obvious, most desired by the patient, and one of the very best one can employ. In spite of a certain prejudice against its use in brain disease, opium (and its preparations) relieves the headache more certainly than any other drug, and too little is known of its effect on cerebral circulation to make one forego its obvious benefits because of speculative doubts or the unfounded supposition that the sleep it produces is likely to merge into a state of real coma. The bromides should be freely administered, not only because of the relief they give to the headache, but because they have probably a beneficial influence on the cerebral circulation and a general quieting effect on the cerebro-spinal system, thus preventing convulsive seizures. Phenacetine and the kindred preparations may be used sometimes with good effect.

When the vomiting is frequent and distressing it may be alleviated by the patient swallowing morsels of ice, and sometimes, again, by sipping water or other fluid as hot as can be swallowed, by irritation to the epigastrium and spine by mustard, and by enemata of chloral hydrate.

The bowels must be kept freely open, and where the patient is strong purgation may be of benefit. The kidneys are such exceedingly important eliminators of toxic substances that it is well to use diuretics as part of the treatment.

When a septic condition of the system is conjoined to the meningitis there is nothing to which one can resort with such confidence as the tincture of the chloride of iron, given in unstinted measure.

One thing is to be borne in mind with all varieties of treatment and in all forms of the disease: it is to keep up the strength of the patient with a sufficiency of bland nourishment, such as milk, given with a regularity that nothing is allowed to interfere with. Stimulants are to be given when the failure of the strength of the heart calls for them, and under such circumstances there need be no fear of any injurious effects from their use.

Where meningitis is connected with middle-ear disease the greatest attention must be paid to the relief of the local affection. Not a few cases have been recorded in which marked symptoms of meningitis (even diplopia and optic neuritis) have been brought to a favorable termination by draining the middle ear through an opening in the mastoid process (A. Broca) or other outlet. The same attention must be paid to the local treatment in all other local suppurative processes.

The effort has frequently been made to diminish the intracranial pressure, to which is due so many of the grave symptoms of meningitis, by draining away the cerebro-spinal fluid. This is done by a puncture of the membranes of the cord between the laminæ of the lower lumbar vertebræ, already alluded to under the head of "Diagnosis." In cases of purulent meningitis it cannot be expected to effect much good. It would seem to be more applicable in serous meningitis, and is recom-

mended on good authority, although thus far the results obtained have not been very marked. Trephining the skull in meningitis has been followed by good results in some cases, and its usefulness will probably be developed in the future.

Chronic Meningitis.

In old alcoholic subjects the pia-arachnoid undergoes changes due to a chronic degeneration. It loses its transparency and becomes opaque, milky, and thickened, especially the outer layer (the arachnoid), but does not show the characteristic signs of inflammatory action, but rather those of malnutrition. It is scarcely possible, amongst the mass of symptoms arising from the cerebral poisoning and degeneration in these cases, to distinguish those, if there are any, which result from the meningeal alterations. Occasionally a chronic meningitis occurs in adults which is not the effect of syphilis, although it often is impossible absolutely to exclude it as a factor in the disease. Its usual seat is at the base of the cranium, where it produces thickening and gluing together of the membranes which are adherent to the brain.

Symptoms.—The general symptoms are more or less persistent headache, with occasional vomiting and giddiness. From the position of the disease we readily see how the optic and other cranial nerves become crippled, giving rise to local symptoms, as optic neuritis, strabismus, paresis of the muscles of the face, etc. The inflammatory process may close the foramen of Monro and the lateral openings of the fourth ventricle, and thus hydrocephalus may be an outcome of the disease. The mercurial is the most efficient treatment, and under it arrest or improvement may result. Chronic meningitis in children during the first years of life is more common than the affection just described in the adult. The disease generally confines itself to the posterior fossa of the skull. Here the post-mortem examination shows all the appearances of a chronic fibro-purulent inflammation, thickening, and adherence of the membranes to each other and to the brain. Vomiting occurs. General convulsions of an epileptiform character are common, and rigidity of the limbs. The nerves situated in the posterior fossa suffer, especially the abducens, and we observe strabismus. Blindness often occurs from pressure exerted on the optic nerves by an effusion into the third ventricle. The involvement of the pons and medulla causes retraction and rigidity of the neck, which sometimes come on gradually, sometimes suddenly. The tendency in this disease is to death by hydrocephalus, which occurs from the inflammatory occlusion of the outlets of the fourth ventricle. The disease may continue over a year, and sometimes it is then arrested and the patient may recover. Here, as in the chronic meningitis of the adult, the mercurial treatment gives the most hope, the more so that in many cases a syphilitic infection cannot be absolutely excluded in making the diagnosis, although our investigation fails to detect it.

THROMBOSIS OF THE SINUSES OF THE DURA MATER.

The circulation in the sinuses of the dura mater differs in its conditions from the venous circulation in other parts of the body.

The sinus lumen is not circular; in the longitudinal sinus it is triangular. The walls of the sinuses are unyielding and non-elastic, and thus cannot be diminished in calibre by the pressure of surrounding organs or by contraction adapt their capacity to a varying amount of circulating blood. At different points, moreover (longitudinal and cavernous sinuses), their lumen is crossed by trabeculæ. The cerebral veins of the hemispheres empty into the longitudinal sinus, mostly at an angle opposed to the blood current in the sinus. All of these conditions render the movement of the blood current in the sinuses sluggish, and, as we might suppose, very liable to stasis and the formation of a clot. Nevertheless, the stoppage of a sinus by a clot rarely occurs from what we would call a stagnation of blood, and by far the most frequent cause of sinus thrombosis is a morbid change in its endothelial lining—a phlebitis.

We divide sinus thrombosis into two kinds—viz. a primary, so-called "marantic" thrombosis, which results from an enfeebled general condition and heart weakness, and a secondary thrombosis, which is the result of an extension to the sinus of an inflammation from adjacent parts. The first has its location almost always in the longitudinal sinus, and occurs very much the most frequently in young children. Its principal cause is assumed to be weakness of the heart and consequent sluggishness of the blood current, and it occurs after the patient has been enfeebled by disease of an exhausting character. Thus in children thrombosis commonly supervenes upon profuse diarrhœa. It occurs in the adult in the course of exhausting diseases, such as phthisis, cancerous affections, septicæmia, and chlorosis, less frequently in acute diseases, as typhoid fever. It is supposed that the languid flow of the blood consequent on the enfeebled action of the heart, together with the peculiarities of the longitudinal sinus (its triangular lumen and trabeculæ), allow a clot to form. It is also supposed by some that the diminished volume of the blood in the body caused by disease aids by diminishing the force of the blood current. It is to be noted that in some general diseased conditions, as in chlorosis, in which thrombosis not seldom occurs, there is a fatty degeneration of the endothelium of the sinus and its trabeculæ, which may readily be a factor in the production of the clot (Bollinger).

Secondary thrombosis depends upon the well-known favoring influence the diseased endothelium has in causing coagulation of blood. A phlebitic condition of the sinus originates in a septic infection extended to it from neighboring parts. We can appreciate how readily this takes place—first, by the direct connection of the sinus walls with carious bones; and secondly, by the direct continuity of the lining membrane of the veins emptying into it. Some of these veins come from the outside of the skull—ophthalmic veins, emissary veins, from the middle ear, etc.—and some from the brain through the pia-arachnoid; and all of these serve as ready channels for infection. Thus we may have thrombosis from caries of the cranial bones, from purulent meningitis

within the skull, and from erysipelas, carbuncle, panophthalmitis, and other septic processes on the outside. Middle-ear disease of all the causes of sinus thrombosis is the most frequent—so much so that in that affection such a complication is alway jealously to be watched for. Lastly, direct traumatism or pressure by tumor or abscess on a sinus may cause thrombosis.

Pathological Anatomy.—The extent of the sinus occupied by the thrombus varies very much. Cases are on record in which nearly every sinus was involved, although it is usual for the thrombus to be confined to one point or one sinus. The clot may extend into the veins. Thus the internal jugular may look and feel like a cord, and the clot may even extend into the subclavian. With thrombosis of the longitudinal sinus the superficial cerebral veins are plugged with clot, and are seen hard and distended, "worm-like," on the surface of the hemispheres, that portion of them distal to the plug being greatly congested and contorted. This extreme venous congestion affects the cerebral substance, which shows capillary hemorrhages, points of softening, and sometimes hemorrhages of larger size. There may be meningeal hemorrhage. The thrombus varies in appearance and consistency, sometimes being dark, soft, and friable when it is recent; of a lighter color, tougher, and more adherent to the wall of the sinus when it has been longer formed. The secondary thrombus which forms under septic influence shows purulent changes, the walls of the sinus also exhibiting morbid alterations.

Symptoms.—The general symptoms caused by thrombosis of a sinus are so mixed up and obscured by the symptoms of the morbid conditions causing and accompanying it that it is often impossible to discriminate between them. The symptoms which characterize a thrombosis are those local ones which are caused by the arrest of the venous circulation of particular regions, or such as are caused by interference of the clot with adjacent nerves, or those dependent on the occurrence of septic foci or general septicæmia from the breaking down of a purulent clot. Thus in "marantic" thrombus of the longitudinal sinus the symptoms of the preceding cerebral anæmia would mask any general symptoms caused by the clot, and only the occurrence of convulsions and œdema of the scalp would enable us to make a probable diagnosis. In septic thrombus the symptoms of the almost invariably accompanying meningitis dominate the scene and may conceal those of the thrombus. Nevertheless, each sinus when occluded gives rise to special symptoms, mainly derived from the interference with the circulation of a particular region.

Thrombosis of the longitudinal sinus is almost always primary, and occurs most frequently in young children after profuse discharges from the bowels or some exhausting disease; also in adults when the system has been enfeebled and the heart weakened by protracted disease.

The general symptoms will correspond to the depressed condition of the system, and have nothing in them characteristic. The symptoms that depend directly on the obstruction of the circulation are epistaxis, caused by the arrest of the circulation in veins of the nose which empty into the sinus, œdema, and swelling of the side of the head caused by occlusion of the mouths of the parietal emissary veins. From the side of the brain there are symptoms depending on the congestion, and hem-

orrhages in the hemispheres caused by the plugging of the cerebral veins. There are headache, vomiting, delirium, stupor. The cortical centres being the seat of the congestion, there may be unilateral spasms or convulsions, or one limb alone may be affected, accordingly as one or more motor centres are involved. Finally, coma sets in, which may continue for days or weeks before death. When these cerebral symptoms occur suddenly in the course of wasting disease it makes us consider the probability of a thrombus; otherwise they so closely resemble those of meningitis that we cannot make the diagnosis.

Thrombosis of the Lateral Sinus.—The most frequent cause of lateral sinus thrombosis is an extension of an inflammation from the middle ear or from caries of the temporal bone. It may be sometimes occasioned by inflammations on the outside of the skull, carried through the emissary vein in the mastoid process, and not unfrequently inflammation of the mastoid cells is the exciting cause. Purulent meningitis so often accompanies lateral thrombosis (being, indeed, usually caused by the same conditions which produce the latter) that the general symptoms of the thrombosis are usually masked by those of meningitis, and here again one must look for the distinctive symptoms in the local changes produced by the disease of the sinus. Thus there is tumefaction over the mastoid process and pain on pressure, especially if made over the point where the mastoid vein enters the bone to empty into the sinus. Sometimes the clot extends from the sinus into the internal jugular vein, and may be felt as a hard cord which is tender to the touch, and is sometimes so painful that the patient keeps the face turned toward the affected side to relieve the pressure from the sterno-mastoid muscle. It has been affirmed that the plugging of the lateral sinus leaves the internal jugular empty, so that the external jugular can pour its blood into it with such facility as to cause a collapsed condition as compared with the external jugular vein of the unaffected side. Others (Jansen), however, have not confirmed this point in the symptomatology. Very rarely the nerves going through the anterior division of the jugular foramen (the ninth, tenth, and eleventh) are interfered with in lateral sinus thrombosis, so as to cause slowing of the pulse, difficulty of swallowing, contraction of the sterno-mastoid, etc. As these nerves pass through a distinctly separate portion of the foramen, one cannot suppose them subject to pressure by the distended vein, and their implication most probably is the result of an extension of the inflammation to them.

In *thrombosis of the cavernous sinus* there may be some very characteristic symptoms caused by the stopping of the circulation in the ophthalmic vein which opens into it. The obstruction of this vein causes an œdema of the conjunctiva and eyelids, and even of the face, with cyanosis of the skin about the orbit. Exophthalmos is produced by the congestion of the retrobulbar veins. The fundus of the eye shows congestion of its vessels and choked disk. From the peculiar position which the motor nerves of the eyeball and the ophthalmic branch of the fifth hold to the sinus (lying within its cavity) they are more or less involved in the lesion. Thus the movements of the eye may be crippled or the eye rendered immovable[1] by the pressure of the thrombus on these

[1] The writer has seen both eyes perfectly fixed in a case of double cavernous thrombus without meningitis.

nerves. The involvement of the branch of the fifth may give rise to neuralgia. The secondary or phlebitic thrombi are often the source of a general septicæmia by their breaking down and thus diffusing the septic matter through the system.

Diagnosis.—The diagnosis of thrombus of the sinuses is very difficult unless symptoms of impeded local circulation are present. In primary or marantic thrombosis, which is almost always confined to the longitudinal sinus, and which occurs from exhaustion and feebleness of the heart, the symptoms, especially in children, are those of cerebral anæmia or the "hydrocephaloid" condition. If, however, the somnolence and stupor occur suddenly, or if the general enfeeblement and heart weakness do not appear to warrant the cerebral symptoms; if the convulsions involve a single limb; above all, if there are repeated epistaxis and œdema of the side of the head,—one may make a diagnosis of probable thrombosis of the longitudinal sinus. In secondary or phlebitic thrombosis the difficulty of diagnosis is still greater, purulent meningitis and brain abscess giving rise to the same general symptoms and having the same etiology, and, moreover, thrombus of the lateral sinus being most frequently conjoined with meningitis. Here, again, a diagnosis must rest on the symptoms of local venous congestion, which have been already described. There is in thrombus of the cavernous sinus congestion of the ophthalmic vein, causing œdema of the conjunctiva and eyelids, exophthalmos, and the symptom, conclusive when it occurs, of paralysis of the muscles moving the globe of the eye, accompanied with neuralgia in the distribution of the first branch of the fifth. Plugging of the lateral sinus gives rise to tenderness and œdema over the mastoid process, and if the clot extends into the internal jugular, where it may be felt, the nature of the affection is no longer in doubt.

Prognosis.—The prognosis is extremely grave in all cases of sinus thrombus, but should not be entirely hopeless, cases of recovery having been recorded in which the symptoms left no doubt of the existence of thrombosis. In marantic thrombosis of the longitudinal sinus the chances for children are greater than for adults. It may be that the thrombus is sometimes absorbed or so gradually disintegrated as to cause no injury, and recovery, more or less complete, results.

In septic thrombosis the danger is not so much of a fatal interference with the circulation (if the lateral and cavernous sinus is plugged on one side, a compensatory circulation is possible) as of septic poisoning and the almost constantly accompanying meningitis.

Treatment.—Except remedies for keeping up the strength and stimulating the heart in marantic thrombus, and the use of tincture of iron in the secondary septic condition, we can hardly speak of internal medication in sinus thrombosis. Surgical interference fortunately presents more hope. Surgical prophylaxis should be invoked early in all those conditions which lead to thrombosis, such as otitis media, inflammation of the mastoid cells, carbuncle, etc., and all accumulations of pus or inflammatory products should be prevented or removed.

In thrombosis of the lateral sinus (and it is the one most frequently affected) the diseased point can be readily reached by the surgeon and the septic matter cleared away. This operation has been frequently performed, and often with success.

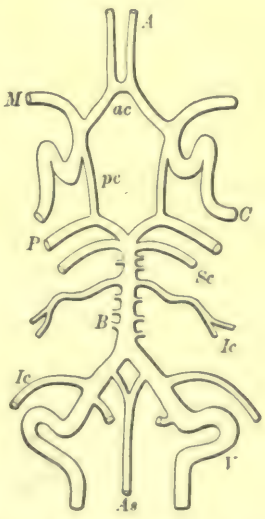
DISEASES OF THE BLOODVESSELS OF THE BRAIN.

By F. X. DERCUM, A. M., Ph. D., M. D.

The diseases of the bloodvessels of the brain give rise to some of the most important affections met with in daily practice. We need only instance the various forms of apoplexy, of hemiplegia, or of aphasia that have their origin in the rupture or obstruction of vessels. Further, so much is taught of the function and structure of the nervous system by these diseases that they deserve detailed consideration.

In approaching the subject from a comprehensive standpoint it is necessary first to review briefly various facts of anatomy, especially the vascular supply of the brain. The brain receives its supply of blood through the internal carotid and the vertebral arteries. The internal carotids, it will be remembered, make their entrance into the cranium on either side by a sigmoid course through the carotid canal of the temporal bone. Immediately upon entering the cranial cavity each carotid gives off two branches—one the anterior cerebral artery, and the other the middle cerebral or Sylvian artery, the last-mentioned vessel appearing rather as a continuation of the parent vessel (Fig. 38). The two anterior cerebrals approach each other and are joined together early in their course by a small anastomosing branch, the so-called anterior communicating artery. The vertebral arteries enter the cranial cavity through the occipital foramina on either side of the medulla oblongata. Each vessel proceeds forward and inward to the front of the medulla, and the two vessels become confluent with each other at the lower border of the pons to form a new vessel, the basilar artery.

Fig. 38.



Circle of Willis, basilar and vertebral arteries: *C*, internal carotid; *M*, middle cerebral; *A*, anterior cerebral; *ac*, anterior communicating; *pc*, posterior communicating; *P*, posterior cerebral; *Sc*, superior cerebellar; *V*, vertebral; *As*, anterior spinal; *B*, basilar; *Ic*, inferior cerebellar arteries.

The last-mentioned vessel, after giving off a number of branches presently to be mentioned, again divides at the anterior border of the pons into the posterior cerebral arteries. Each posterior cerebral artery is joined by a slender anastomosing vessel, the posterior communicating artery, with the internal carotid at the point where the latter gives off (or rather becomes)

the middle cerebral. It will be seen by consulting the figure that an irregular polygon is formed by these vessels at the base of the brain, the so-called circle of Willis. It is made up anteriorly by the anterior communicating artery and by the anterior cerebrals, laterally by the two posterior communicating arteries, and posteriorly by the posterior cerebrals.

In addition to the posterior cerebrals, the basilar artery gives off, first, the superior cerebellar arteries; secondly, the anterior inferior cerebellar arteries and a number of small branches on each side distributed to the pons. The posterior inferior cerebellar arteries are given off, not by the basilar, but by the vertebrals. The vertebrals also give off the anterior and posterior spinal arteries, with which, however, we are not at present concerned.

The formation of the circle of Willis ensures a free and ready anastomosis of the bloodvessels at the base—a fact of great importance, inasmuch as anastomosis does not occur elsewhere in the bloodvessels of the brain. It is necessary to state, however, that the anastomosis of the circle of Willis is not always perfect or complete, inasmuch as anomalies not infrequently occur. Sometimes the posterior communicating vessel of one side is absent, or one of these vessels may be exceedingly large while the other is exceedingly small. Sometimes, again, the anterior communicating artery is absent, or, on the other hand, may be represented by two vessels instead of one. Rarely it is represented by a large communicating vessel joining the anterior cerebrals very near their origin. These facts are of some importance, inasmuch as these anomalous vessels have occasionally been found to be the seat of aneurysmal disease.

The bloodvessels of the cerebrum are conveniently divided into cortical and central arteries. Inasmuch as the diseases of these several vessels give rise to special symptoms, it is important that we should review the exact areas of the cortex and other portions of the brain supplied by each vessel.

The middle cerebral, clinically and anatomically the most important, ascends outwardly in the Sylvian fossa and distributes its branches to the island of Reil and to the lateral surface of the cerebral hemisphere. Near its origin it gives off a number of fine central branches, which enter the substance of the brain through the anterior perforated space, and supply, as we will presently see, the corpus striatum and the capsules. The cortical branches consist, in addition to those distributed to the island of Reil, of the branch or branches to the third frontal convolution, to the ascending frontal, to the ascending parietal, and to the superior temporal convolutions (see Fig. 39). (In part the middle temporal convolution is also supplied by this vessel.) The most anterior branch is termed the inferior frontal artery, and it is distributed by three or four divisions to the external part of the orbital surface and to the third frontal convolution of Broca. The second branch is termed the ascending frontal artery. It is distributed to the lower two thirds or three fourths of the ascending frontal convolution, as well as to the adjoining portion of the second frontal convolution. The third is termed the ascending parietal artery, and is distributed to the ascending parietal convolution. The fourth branch is termed the inferior parietal artery, and is dis-

tributed to the superior and inferior parietal lobules. At times this branch is fused with the ascending parietal artery, and then forms with it a common trunk. The great importance of these branches from the

FIG. 39.

Showing the cortical distribution of the middle cerebral artery.

standpoint of the clinician is at once appreciated when we observe that they embrace in their supply the greater number of cortical centres the function of which has been definitely determined. These facts will be more particularly dwelt upon in speaking of the diseases to which these vessels are liable.

The anterior cerebral artery lies in the great longitudinal fissure between the hemispheres, and, in addition to a small number of vessels

FIG. 40.

Diagram showing the areas of the lateral aspect of the hemisphere supplied by the anterior, middle, and posterior cerebral arteries respectively.

= area of anterior cerebral;

= area of posterior cerebral;

= area of middle cerebral.

supplying the orbital surface of the frontal lobe, it is distributed (see Figs. 40 and 41) to the first and second frontal convolutions and the

FIG. 41.

Diagram showing the areas of the mesial aspect of the hemisphere supplied by the anterior, middle, and posterior cerebrals.

= area of anterior cerebral;
= area of posterior cerebral;
= area of middle cerebral.

mesial surface of the hemisphere as far back as the quadrate lobule, almost all of which is included in its supply.

The posterior cerebral artery supplies the mesial surfaces of the occipital and temporal lobes, as well as the lateral surface of the occipital lobe and the inferior portion of the lateral surface of the temporal lobe. Inasmuch as branches from this vessel supply the cuneus, it may also assume clinical importance.

The limits of the various areas of vascular supply are shown in the accompanying figures.

The central arteries—that is, those which supply the basal ganglia and capsules—next merit consideration. They consist, first, of the numerous small branches given off by the middle cerebral close to its origin, and which enter the anterior perforated space; these have already been mentioned. Secondly, they consist of vessels given off by the posterior cerebrals conjointly at their point of origin from the basilar. These enter the posterior perforated space. In addition, a small number of fine central nutrient arteries are given off by the anterior cerebrals and anterior communicating, and also by the posterior cerebral arteries after they have wound around the crura. The central arteries given off by the middle cerebral are clinically of great importance, and must be considered in detail. They consist, as already stated, of a large number of minute vessels which are given off from the middle cerebral almost immediately after its origin. They enter the anterior perforated space. They are by virtue of their distribution separated into two groups: first, the internal striated arteries, and second, the external striated arteries. The internal striated arteries are distributed first to the

inner and middle segments of the lenticular nucleus, then to the internal capsule, and finally to the caudate nucleus and in part to the optic thalamus. The external striated arteries, situated externally to the preceding, supply the outer nucleus of the lenticular body, the putamen. Some of them enter the substance of the putamen, while others ascend over its external face between it and the external capsule. These external striated arteries are in turn subdivided into two groups: the first, the lenticulo-striated arteries, which are composed of the arteries which supply the anterior portions of the putamen, the anterior portion of the internal capsule, and the caudate nucleus. The second group, the lenticulo-optic arteries, are distributed to the posterior portion of the lenticular nucleus, to the posterior portion of the internal capsule, and to the optic thalamus. The distribution of these arteries is shown in the accompanying figure (Fig. 42). Among the vessels of the anterior group there is usually found one more voluminous than the others, which passes along the outer surface of the external segment of the lenticular nucleus, then enters the segment, perforates the anterior portion of the capsule, and finally ends in the caudate nucleus. This is the branch which Charcot has named the "artery of cerebral hemorrhage," inasmuch as this vessel is especially prone to be involved.

FIG. 42.

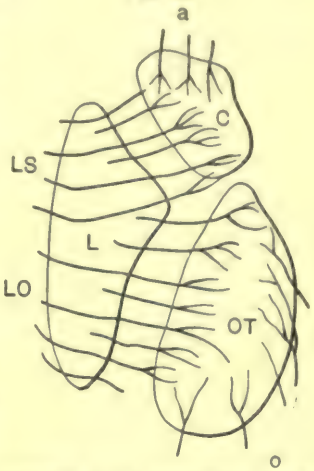


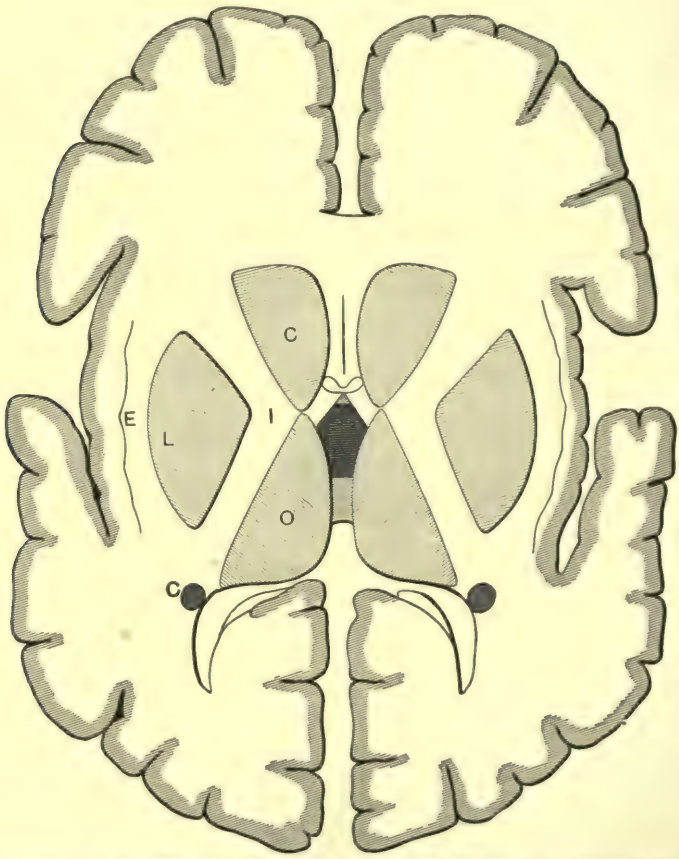
Diagram of the distribution of the central arteries to the basal ganglia and capsules: *C*, caudate nucleus; *L*, lenticular nucleus; *OT*, optic thalamus; *LS*, lenticulo-striated arteries; *LO*, lenticulo-optic arteries; *a*, central-branches from the anterior cerebral; *o*, central branches from the posterior cerebral (optic arteries).

As already stated, the posterior cerebral arteries give off a number of small vessels which enter the posterior perforated space. They are distributed to the optic thalamus and to the walls of the third ventricle. In addition, the posterior cerebrals give off other vessels supplying the posterior portion of the optic thalamus, termed respectively the postero-internal optic artery and the postero-external optic artery.

The regions most frequently involved in cerebral hemorrhage are the basal ganglia and capsules. In order that the symptoms presented in a given case of hemiplegia should be clearly understood, it is necessary to review briefly so much of the anatomical relation of these structures to each other as is of clinical value. Fig. 43 represents a horizontal section of the brain. Immediately beneath the cortex of the island of Reil is found a secondary layer of gray matter named the claustrum. Immediately subjacent to the claustrum, and separating it from the lenticular nucleus, is a layer of white matter termed the external capsule. The lenticular nucleus is seen as a lens-shaped mass consisting of three segments, the outer of which is known as the putamen, the middle and inner one constituting the globus pallidus. Immediately within the lenticular nucleus is seen a layer of white matter having the shape of an obtuse letter V. Anteriorly it separates the lenticular nucleus from the caudate nucleus and posteriorly from the optic thalamus. It is espe-

cially this V-shaped tract of white matter which is of importance in studying the symptomatology of cerebral hemorrhage. It therefore merits detailed description. The anterior arm of the V is termed the anterior limb of the capsule (see Fig. 44), while the posterior arm is termed the posterior limb. The portion by which the two limbs are joined is termed the knee of the capsule. The anterior limb is largely made up of fibres passing downward from the frontal lobe. A small

FIG. 43.



Horizontal section of the brain, showing basal ganglia and internal and external capsules: *C*, caudate nucleus; *L*, lenticular nucleus; *O*, optic thalamus; *I*, internal capsule; *E*, external capsule.

area, however, immediately in advance of the knee appears to convey fibres, lesion of which on the left side will give rise to motor aphasia. The knee of the capsule and the anterior two thirds of the posterior limb contain fibres which pass downward from the motor area of the cortex; the fibres in the knee are especially related to the movements of the face, tongue, and of mastication, while the fibres of the anterior two thirds of the posterior limb are concerned in the movements especially of the arm and leg and also of the trunk. They constitute those

FIG. 44.

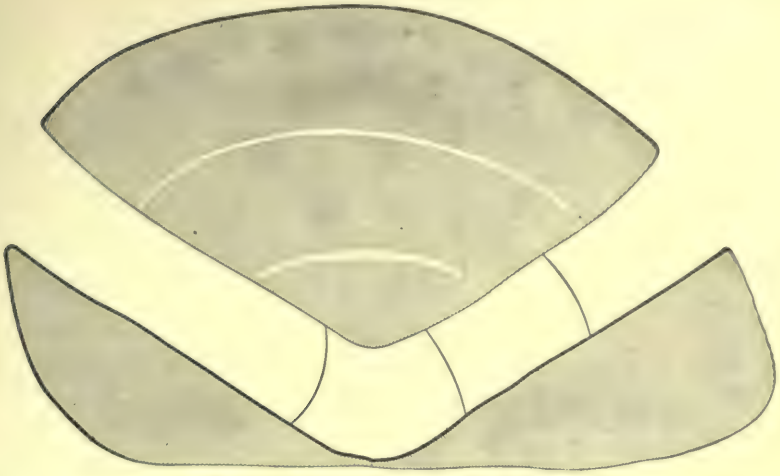


Diagram of the internal capsule (see text).

fibres which in the medulla and cord become the anterior pyramids and lateral columns. The posterior third of the posterior limb contains

FIG. 45.

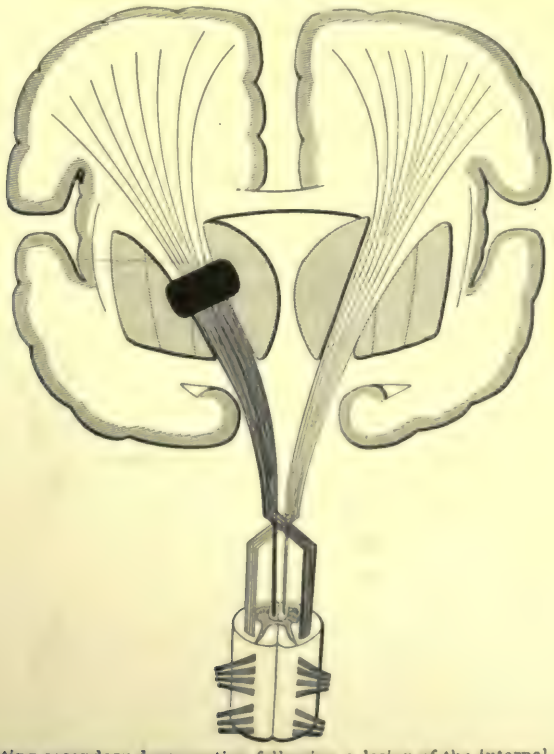


Diagram illustrating secondary degeneration following a lesion of the internal capsule (adapted from Edinger).

FIG. 46.

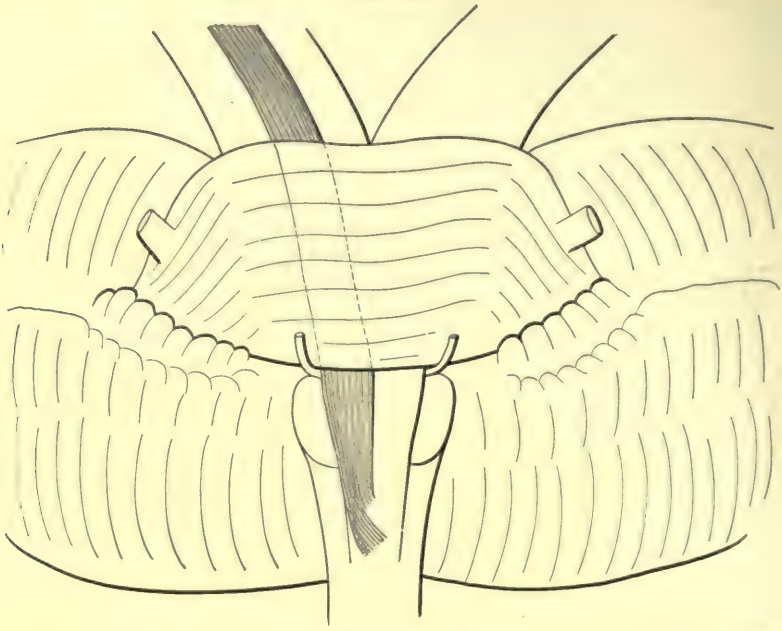


Diagram showing secondary degeneration through the crus and anterior pyramid of one side (adapted from Edinger).

fibres which pass upward to the sensory areas of the cortex; lesions of this tract give rise to anæsthesia of the opposite side of the body.

FIG. 47.

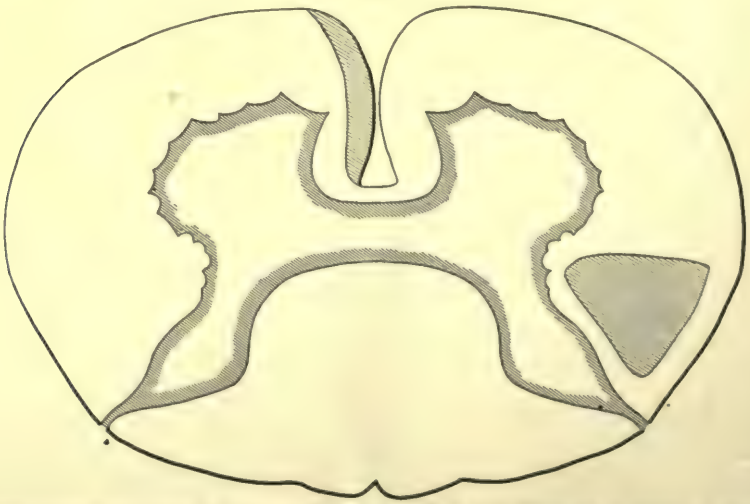


Diagram showing the tracts of degeneration in the cervical cord secondary to lesion of the internal capsule (see text).

The various motor fibres of the cortex descend in this narrow tract, the internal capsule, pass between the caudate nucleus and the optic

thalamus on the one hand and the lenticular nucleus on the other, then downward through the pes of the crus, the pons, the anterior pyramids of the medulla, where they decussate with the fibres of the opposite side, and then pass downward through the cord as the so-called pyramidal or lateral tracts. In the cord these fibres come into relation, though not into continuity, with the large multipolar cells of the anterior cornua of the gray matter. These multipolar cells in turn give off the motor fibres passing to the muscles. Our knowledge of the course of the motor tract is almost exclusively the result of clinical and pathological observation, inasmuch as a lesion of the tract at any point causes degeneration of the fibres below that point. In Fig. 44, for instance, we have a diagram showing the course of the degeneration through the capsule, pons, medulla, and cord; in Fig. 45 is shown the course of such a degeneration through the pyramids. In Fig. 46 we have a diagram illustrating the areas in which such a degenerative tract is found upon cross-section of the cord—namely, in the pyramidal tract or lateral column of the opposite side, and, in the cervical cord, in the antero-median column or column of Turck on the same side. It is evident that lesions of the knee and anterior two thirds of the posterior limb of the capsule alone are followed by descending degeneration.

Cerebral Hemorrhage.

Etiology.—*Predisposing Causes.*—The causes of cerebral hemorrhage naturally group themselves into predisposing and exciting. Among the predisposing we have, first, disease of the bloodvessels, and as still earlier causes the various conditions which predispose to such disease. Atheroma of the bloodvessels of the brain is a very common affection, and yet atheroma must be looked upon as rather infrequently related to cerebral hemorrhage. In by far the greater number of cases there are present minute dilatations known as miliary aneurysms. These are found sometimes in considerable numbers in the various nutrient arteries which enter the perforated spaces, especially those which enter the anterior perforated spaces. The changes in the vessel walls upon which the formation of these minute aneurysms depend will be considered in the section on Pathological Anatomy (p. 370); suffice it to say here that these aneurysms present areas in which the resistance of the vessels to changes in the blood pressure is much diminished.

Closely related in importance are the various causes of degeneration in the vessel walls. The relation between gout, for instance, and disease of the bloodvessels is well known, and gout in this sense distinctly predisposes to cerebral hemorrhage. This is likewise true of Bright's disease, especially granular degeneration of the kidney, in which vascular changes are more or less marked. In conformity with this fact, clinical experience shows that renal disease is a predisposing factor to cerebral hemorrhage. Disease of the kidneys is found in about one third of the cases of cerebral hemorrhage. Chronic alcoholism is also an important factor in the etiology of apoplexy, leading, as it does sooner or later, to arterial and renal changes. Among the rarer and special causes of arterial degeneration and of subsequent hemorrhage we should mention chronic lead-poisoning. Syphilis of the vessels should

be also considered. It is doubtless in a limited number of cases a factor in cerebral hemorrhage, but in by far the larger number of syphilitic hemiplegias the lesion is one of thrombosis and not of rupture. In a very small proportion of cases a disease of the blood which leads to hemorrhage or extravasation is the important factor. More or less extensive bleedings are not infrequent in purpura hæmorrhagica, leucæmia, and pernicious anæmia. In a case of purpura hæmorrhagica recently observed by the writer the symptoms were those of an ingravescent apoplexy, whilst the autopsy revealed extensive extravasations of blood into the meninges.

As might be expected from the remarks already made as to the relation between vascular degeneration and apoplexy, heredity constitutes a predisposing factor. Not infrequently we elicit a history of apoplexy in the ancestors—sometimes, indeed, for several generations. At other times the family tendency to cerebral hemorrhage is shown by its attacking several members of the same generation. Frequently too, if a history of apoplexy be absent, a history of hereditary gout may be elicited—a factor of almost equal significance.

Among predisposing causes we should also mention sex. More men than women suffer from apoplexy, and doubtless for the reason that men as a class are less temperate, and are in other ways exposed to influences which bring about arterial degeneration. Considerably more than half of all the cases of cerebral hemorrhage occur in men.

Age is also an important factor. Leaving out of account the cerebral hemorrhages occurring in the newborn or in early infancy, the disease is very rare in childhood, youth,[1] and early adult life. By far the larger number of cases occur after forty and forty-five years of age, and it is particularly from the middle period of life up to about sixty years of age that apoplexies are most frequent. That they occur at other periods of life is of course well known. When, however, an apoplexy occurs distinctly before the usual period—for example, at thirty or thirty-five—special causes exist, and they are to be sought for particularly in a history of syphilis or of some other pre-existing disease which has been attended by rapid and early vascular degeneration. In the absence of such causes a marked predisposition may sometimes be inferred from the family history. Among less important and less significant factors may be mentioned season and climate. It is a remarkable fact that more apoplexies occur during the winter months than in the summer, and also that more apoplexies occur in northern than in southern climates. It is interesting also to note that apoplexies occur more frequently at certain times of the day; thus the larger number of cases occur in the early morning, while the next most favorable period of the twenty-four hours is the night. Out of 47 cases collected by Dana,[2] 13 occurred during sleep or sleeping hours. The least favorable time is the evening after five o'clock.

Exciting Causes.—The immediate causes of cerebral hemorrhage are

[1] An interesting case is reported by C. Randoph (*Brit. Med. Journ.*, 1896, i. 785) of extensive cerebral hemorrhage involving the right temporo-sphenoidal lobe in a lad of sixteen. It is safe to assume, however, that in such cases a special cause of vascular degeneration must have been present.

[2] Dana: *New York Med. Record*, 1895, xlvii. 225.

not clearly understood. Unusual muscular exertion is often assigned as an exciting cause. It is true that cerebral hemorrhage occasionally occurs during some violent muscular exertion, such as lifting a heavy weight, straining at stool, or during unusual exertions in coitus. It must be admitted, however, that in by far the larger number of patients no such history is present. It is not infrequently stated by the members of the family or the patient himself that he was quietly seated or perhaps walking quietly when the attack occurred. Sometimes the attack comes on after eating a full meal. The occasional concomitance of a heavy intoxication and an apoplexy should also be borne in mind.

SYMPTOMS.—The symptoms must be divided into those occurring at the time of the attack, some of which are transient, and those which persist subsequently. The first group of symptoms are those commonly pertaining to the condition known as apoplexy. It is, however, important to state here that the term "apoplexy" is properly used in a less restricted sense, and applies not only to the sudden loss of consciousness and paralysis resulting from hemorrhage, but to the same symptoms resulting from any other cause—*e. g.* embolism, serous effusion, etc.—and, inasmuch as it is frequently impossible to differentiate the various causes of an apoplectic attack at the time of its occurrence, the symptoms about to be described apply in a measure to apoplexy in general.

A prodromal period is said now and then to occur. It is doubtful, however, whether the symptoms are not actually due to the beginning of the lesion itself. The patient complains of headache, of dizziness, of fulness in the head, or of numbness and creeping sensations in an arm or a leg; or a slight weakness in one limb may be the first thing noted. Most frequently, however, such prodromal symptoms are not noted, and the attack comes on without any warning whatever. Indeed, it frequently occurs while the patient is asleep, and his condition may not be discovered until the next morning. At other times it occurs while the patient is at work or attending to his routine affairs. When occurring during the waking period unconsciousness more or less marked and frequently amounting to coma is present. Even in comparatively slight attacks of hemorrhage this is apt to be the case; occasionally, however, mental confusion or dulness may be the only symptom of mental impairment. In such cases difficulty of speech and weakness of the arm and leg of one side, coming on suddenly, form the prominent features of the attack. Usually, however, the unconsciousness is so profound that it is impossible to rouse the patient. Slight or gradual loss of consciousness not infrequently occurs in slowly progressive cerebral hemorrhage; that is, in the so-called ingravescent form.[1] On the other hand, in cases whereby the rupture of a relatively large vessel, a large outpouring of blood, suddenly takes place, consciousness is completely or almost completely lost from the outset.

The breathing of the patient is, as a rule, very slow and labored. The lips and cheek of the paralyzed side may move passively in and out with each respiration. Frothy saliva may make its appearance on

[1] As Moyer points out (*Med. Fortnightly*, St. Louis, 1895, vii. 65), the hemorrhage may be so gradual in the ingravescent form that its nature may not at first be recognized, and the symptoms may even suggest a rapidly developing gumma.

the paralyzed side of the mouth. Very frequently, when coma is profound, the respiration is stertorous, and in fatal cases, due to an accumulation of frothy mucus in the pharynx and trachea, it may become rattling and gurgling. In other cases, again, more especially those in which the hemorrhage occurs in the neighborhood of the fourth ventricle or in which blood has passed down from the third ventricle into the fourth, it assumes the peculiar rhythm which is described as Cheyne-Stokes respiration; that is, periods in which the respiration is very slow and long drawn alternate with other periods in which the respiration is short and shallow. It is necessarily a symptom of ill omen.

The pulse is frequently slow and full, sometimes hard and tense. In other cases, again, and especially in those which terminate fatally, the pulse is weak and rapid or soon becomes so. The face is often flushed and the veins of the neck are distended. At other times, however, the face is pale; sometimes, though rarely, the pallor is extreme. The skin, as a rule, is wet with a profuse perspiration.

The apoplectic attack may in slight cases be unaccompanied by any change of temperature. Most frequently, however, a distinct fall of temperature can be noted at first, though this in a few hours gives way to a rise of temperature. The fall may be one or two degrees below normal; rarely it is greater and persists until death. The rise of temperature is very variable; it may be slight and transient or very high and persistent. Obviously, the course of the temperature depends upon the size and location of the lesion. If the initial shock has been very great, the fall may be correspondingly marked, and if the bleeding occur in certain situations—for example, the pons—the temperature may be very high. Dana[1] points out that the rise of temperature is higher upon the paralyzed side by about one degree.

The sphincters are, as a rule, found relaxed. The bowels and bladder may be spontaneously evacuated into the bed. In cases in which loss of consciousness is not pronounced the sphincters may be intact. The reflexes in severe cases are lost, both deep and superficial; no response is obtained from the conjunctiva; the knee jerks are absent. Exceptionally in mild cases they may be preserved in variable degrees. In such cases the loss of power in the arm and leg of one side may be recognized by the flail-like manner in which the limb falls back after it has been raised or extended. Complete relaxation is present. On the non-paralyzed side the arm or leg falls less readily, and more like the limb of a person asleep; flail-like relaxation is wanting. This difference between the two sides is often striking. Further, if the patient's body or limbs be pricked with a needle, he may react in such a way as to readily indicate that one or the other side is paralyzed. If he be profoundly unconscious, however, no reaction will take place. Sometimes also, in this way, a sensory loss can be determined upon the paralyzed side, which loss is absent upon the sound side. Attempts at rousing the patient are unsuccessful, save in a limited number of cases in which the impairment of consciousness is not profound or is of short duration. The patient may or may not be able to swallow.

Ordinarily, when the loss of consciousness is profound no movement is made, but in cases where the loss of consciousness is incomplete the

[1] *Trans. Association of American Physicians*, 1894.

patient may make for a time more or less active movements with the non-paralyzed limbs in vain attempts to rise to his feet or to change his position. Rarely the attack is ushered in by a convulsion affecting the side which is subsequently paralyzed. More rarely still a general convulsion occurs.

An examination also reveals, in the larger number of cases, that the side of the face corresponding to that on which the arm and leg are paralyzed droops and is flattened, and that the mouth is distinctly drawn over toward the sound side. If the patient be sufficiently conscious to be able to protrude the tongue, it is not infrequently found that the tongue deviates toward the paralyzed side, due to the unopposed action

FIG. 48.

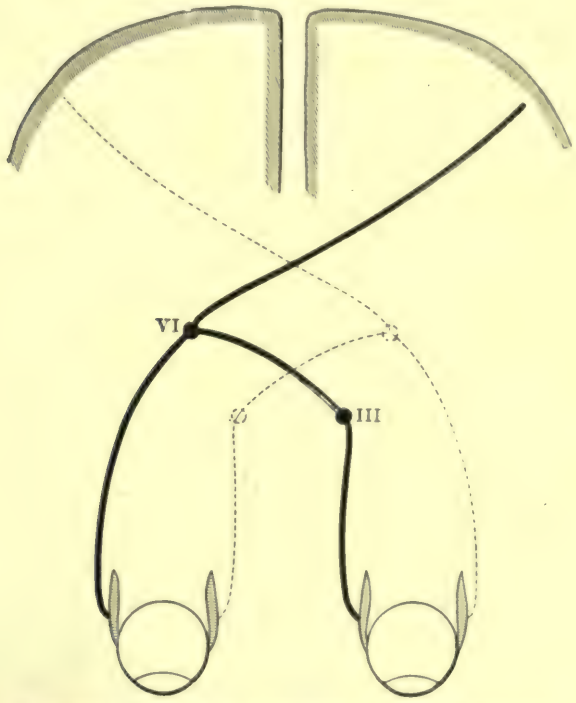


Diagram illustrating the mechanism of conjugate deviation.

of the genio-glossal muscle of that side. When we turn our attention to the eyes, we almost always find that the pupils are equal and that they present no abnormality of size—that is, are neither contracted nor dilated. It is further found that they do not react to light. Where unconsciousness is less profound, however, the reaction to light may be preserved, and in exceptional cases, depending upon the locality of the hemorrhage, the pupils may be unequal. Frequently a very striking symptom is present. It is seen at once, for instance, that both eyes are directed conjointly to one or the other side, generally toward the unparalyzed side. The symptom is termed conjugate deviation, and is accompanied by a rotation of the head in the same direction. It happens that

at times the eyes deviate toward the side that is paralyzed. In such instances more or less spasm of the affected external and internal recti muscles is present, and is not infrequently accompanied by some evidence of spasm or contraction in the paralyzed arm or leg. Spasm being excluded and the phenomenon being clearly established as paralytic, it is evident that the deviation, while away from the paralyzed side, is toward the side of the lesion in the brain. This statement, while perfectly correct, applies only to apoplexies in which the lesion is situated in the capsules or at least at a point above the pons. Inasmuch as the hemorrhage is most frequently in the capsules, the condition described is that which commonly obtains. If, however, the lesion is in the pons, the statement must be reversed. A pontine lesion involving the nucleus of the sixth nerve will produce conjugate deviation toward the paralyzed side and away from the lesion. A glance at the accompanying diagram (Fig. 48) will make this fact clear. The nucleus of the sixth nerve acts conjointly with the nucleus of the oculo-motor nerve of the opposite side. Consequently, a lesion of the pons involving the nucleus of the sixth nerve causes a hemiplegia of the opposite side of the body, and also a deviation of both eyes toward this paralyzed side.

In fatal cases unconsciousness persists until death, or it may be that after an interval of more or less complete recovery of consciousness it is again suddenly lost, and continues so until the end. Recovery from the apoplectic attack is gradual, and is manifested by efforts on the part of the patient to move and speak. Little by little consciousness returns. The full extent of the paralysis can then be determined. If special symptoms, such as aphasia, are present, they are now very evident. Difficulty of articulation is generally striking. Frequently headache is complained of and the patient may be somewhat confused or delirious. Difficulty in swallowing may persist for a little while, but sooner or later disappears. Control over the sphincters is also generally re-established. Along with the return of consciousness it is generally noted that the tendon reflexes can again be elicited. The duration of the apoplectic attack is very variable. The patient may be unconscious for a very short period or may be so for many hours. Persistence of unconsciousness is of course of evil augury.

The apoplectic attack itself having been safely passed, a change in the symptoms will be noted from day to day in the majority of cases. This is especially true of the paralysis. Some return of the movement, though very slight at times, can generally be noted from day to day, and there are indeed some cases in which the paralysis in the course of time entirely disappears. These are instances in which the amount of blood poured out was probably small, in which the fibres of the internal capsule were not seriously ruptured, and in which the paralysis was mainly due to the pressure of a clot subsequently absorbed. In other cases—and indeed in the larger number—while some improvement occurs, this is comparatively small and a more or less marked residual paralysis persists. Just how much recovery will occur in a given case it is impossible to say. However, in favorable cases decided improvement is noted at the end of ten days or two weeks. At this period it is of advantage to study the knee jerks, comparing the two sides, and, if it be found that the knee jerk of the affected side is not exaggerated or but slightly

so, it is fair to infer that degenerative changes are not taking place in the motor tract, and that the final recovery from the attack will be very marked. If, on the other hand, the knee jerk upon the paralyzed side is much more marked than its fellow, a pronounced lesion of the fibres of the pyramidal tract, together with an oncoming secondary degeneration, may be inferred as probable. As a rule, the recovery of power is far more marked in the leg than the arm, though this rule is not constant. If sensory losses have been observed early in the case, they are found to have disappeared. If persistent hemianæsthesia is noted, a lesion of the posterior third of the posterior limb of the capsules is to be inferred.

As week follows week, the symptoms gradually assume a permanent and residual character, and we have present the condition so familiar under the term "chronic hemiplegia." The exaggeration of the knee jerk on the paralyzed side becomes more pronounced, and an ankle clonus also frequently makes its appearance. The tendon reactions indeed become at times excessively marked. Farther on, grasping the paralyzed leg or arm and attempting passive movement, we discover that a new symptom has made its appearance—namely, rigidity. If we attempt to flex the leg, for instance, we find that we can only do so after overcoming a certain amount of muscular resistance. Again, it is noted that the paralyzed limbs tend to assume certain positions. Thus the leg when left to itself assumes the position of extension, while the foot becomes extended upon the leg. Sometimes the degree of extension is extreme. When asked to flex the leg, the patient can only do so with considerable effort; sometimes he cannot flex it at all. Exaggeration of the knee jerk and ankle clonus is under such circumstances generally very marked. When we turn our attention to the arm, we notice that it presents similar phenomena. Voluntary movements at the shoulder and elbow, which had perhaps been slightly regained during the convalescence from the apoplectic attack, are again diminished. The arm is held to the side somewhat adducted, the forearm is flexed midway upon the arm, the wrist is slightly flexed, and the fingers are generally markedly flexed. Considerable variation exists as to the detailed position assumed. If we test the elbow jerk, the biceps jerk, or attempt to test the tendon reaction of the wrist, it is found that they are all exaggerated, frequently to a marked degree. Occasionally, in slight cases especially, the rigidity may be very slight, while the position assumed may only slightly approximate that described. This is true of the leg as well as of the arm. On the other hand, the symptoms of secondary contracture, as they are termed, may become very marked. So great may be the contracture of the arm, for instance, that the physician will be able to extend it only after considerable muscular effort. The fingers, again, may be so contracted that the nails may burrow into the palm. The gait in such a case is characteristic. The shoulder on the affected side is slightly raised as compared with its fellow. The extended leg is thrown far out from the body—that is, strongly abducted—and brought to the ground again and toward the median line with a rotating movement of the trunk. As a rule, the toe or the sole of the foot is scraped or dragged along the ground, while the head and arm are held in the position of semiflexion already described.

The face also presents some changes. The upper half remains, of

course, as before, never having been involved. Rarely, however, a very slight smoothing of the brow and drooping of the eye may be noted. The paralysis of the lower half of the face becomes in the majority of cases much less marked; however, if the patient be asked to show his teeth, as in the act of grinning, it will be found that the paralyzed side responds imperfectly and the mouth is drawn to the sound side. It is interesting in this connection to state that should the patient be induced to smile or to laugh, the paralyzed side will respond as well, or almost as well, as the sound side, and the mouth will be quite even. The explanation of this strange fact is doubtless to be sought for in the difference of voluntary and automatic innervation of muscles habitually used together.

A number of facts regarding muscle involvement should be especially noted in hemiplegia. Even in the most marked cases certain muscles invariably escape. Thus the frontalis, the corrugator supercilii, and the orbicularis palpebrarum have their innervation never affected, or practically never. The slight smoothing of the brow and drooping of the eye mentioned above are rarely noted, and are then barely appreciable. None of the muscles of the neck or trunk are ever involved. It appears that the truncal and certain other muscles are thoroughly innervated by both sides of the brain, and are never paralyzed except by such rare lesions as destroy corresponding centres or tracts on both sides of the brain. It may be stated as a general principle that the more distant the muscle from the trunk and the less associated its action with its fellow of the opposite side the more completely is it paralyzed. For instance, the shoulder and thigh muscles are less involved than the muscles of the forearm and hand or the leg and foot. It is almost unnecessary to state that the degree of the paralysis varies greatly.

If anæsthesia, first noted during or shortly after the apoplectic seizure, persist, such anæsthesia is permanent and due to destruction of the posterior third of the posterior limb of the capsule. Hemianæsthesia is, however, an exceptional occurrence. Hemianopsia is occasionally noted as an accompaniment of or without hemianæsthesia, the blind halves of the retina being on the same side as the paralysis. It is found more frequently in the early or apoplectic period than one would suppose, but it generally soon disappears. Conjugate deviation also does not persist. Visceral complications are practically absent.

As might be expected, the detailed symptoms of a hemiplegia vary with the position of the lesion in the internal capsule. Thus, if the lesion is small and far forward, a little beyond the knee and on the left side of the brain, there may be present marked motor aphasia, while the hemiplegic phenomena may be comparatively slight. Again, if the lesion be a little back of the knee, paralysis may be markedly accentuated in the arm, while the leg may be comparatively unaffected. Such a case we speak of as the "arm type" of hemiplegia. If, on the other hand, the lesion be a little farther back, it will involve mainly the fibres going to the leg, and also the sensory tract, and paralysis becomes most marked in the leg, while the arm is comparatively uninvolved. Due to the involvement of the sensory tract, hemianæsthesia may be present, and such a case is spoken of as the "leg sensory type." Finally, the lesion may occur in the posterior third of the posterior limb of the

internal capsule, and involve not only the fibres of the sensory path, but also the optic radiations as they pass backward to their termination in the cuneus. In such an instance there would be hemianæsthesia and also hemianopsia, the blind fields corresponding to the side of the hemianæsthesia. At the same time, sensory aphasia may be present, the patient being utterly incapable of understanding what is said to him or of comprehending either written or printed characters. Sometimes, although the motor paralysis is slight, there is more or less ataxia in the movements of the affected side. This is doubtless due to lesion of the fibres conveying the muscular sense. These cases constitute the "hemiataxic type." When hemiataxia and aphasia coexist the cases are sometimes spoken of as the "hemiataxic-aphasic type."

The symptomatology thus far presented applies, as already said, to the most common form of cerebral hemorrhage—namely, hemorrhage into the capsules and basal ganglia. Cerebral hemorrhage is, however, by no means restricted to this region, but may occur in other situations —namely, in the cortex, in the centrum semi-ovale, in the crus, in the pons, in the cerebellum, or, in the medulla.

Cortical hemorrhages are exceedingly rare, at least in the adult, though they are met with in the newborn and in early childhood relatively frequently. The degree of the apoplectiform symptoms—for example, the loss of consciousness—depends of course largely upon the extent of the lesion, while the special symptoms present will depend upon the particular area of the cortex involved. If the hemorrhage has occurred in a so-called silent region of the cortex—for example, the frontal lobe—or into the right temporal lobe, no localizing symptoms may be noted. On the other hand, if such a hemorrhage occur in the motor area, it gives rise to focal convulsive and paralytic phenomena. Hemorrhage into the white substance may be unaccompanied by special symptoms unless the hemorrhage happen to be subjacent to a motor area, when special paralytic phenomena supervene. In hemorrhage immediately below the cortex these paralytic phenomena may be preceded by localized convulsive seizures in which rigidity predominates.

Hemorrhages occurring into a crus are very rare, but when occurring may give rise to the signs of an alternate hemiplegia involving the oculo-motor nerve—*i. e.* an oculo-motor palsy of one side and hemiplegia of the other. Hemorrhage into the pons usually presents bilateral phenomena, convulsive and paralytic, though a small lesion may cause unilateral symptoms, and then is apt to be accompanied by conjugate deviation of the eyes away from the lesion. Bilateral symptoms are, however, much more frequently present. The attack is generally ushered in by a convulsion and an apoplectiform syndrome. The paralysis when bilateral may preponderate in the legs or in the arms. Sometimes the convulsions are limited to the legs (Gowers). Such a symptom would suggest pontine lesion at once. The convulsive movements are often peculiar, insomuch that a tonic element is often present, rigidity alternating with sharp, jerking clonic movements. The pupils are frequently markedly contracted; they may, however, be widely dilated. Vomiting is apt to occur. Unconsciousness is apt to be pronounced; if the unconsciousness be less marked, decided anæsthesia of the legs or arms may be noted. Disturbances of respiration probably

due to passage of blood into the fourth ventricle are early and pronounced. Marked rise of temperature also occurs. As may be inferred, pontine hemorrhages are very apt to be fatal.

Hemorrhage into the cerebellum is generally accompanied by pronounced apoplectiform symptoms. Loss of consciousness may be marked, but at times consciousness is preserved. Great pain may be present, referred to the occiput. Vomiting is frequent. Sometimes there are no palsies or other motor disturbances. On the other hand, inco-ordination of movements and intense vertigo may be present. It not infrequently happens that a case presenting these symptoms is mistaken by lay persons for one of alcoholic intoxication. At times paralytic symptoms are present and predominate; for instance, hemiplegia. When occurring it is doubtless due to pressure upon the pons or medulla. At other times, depending upon the seat of the lesion, a marked tendency to turn to one side is presented or the head may be inclined to one side. Erection of the penis has been noted in a few cases. The breathing is not characteristic, but often becomes stertorous and shallow. If recovery follow, the symptoms due to pressure merely, such as hemiplegia, disappear. Distinctly cerebellar symptoms, such as titubation, ataxia, vertigo, and a tendency to rotation, may persist for a time, but frequently these symptoms also pass away.[1]

Hemorrhage into the medulla is rare and is almost invariably rapidly fatal. In those rare instances in which death does not at once ensue, due doubtless to the minute character of the hemorrhage, focal symptoms referable to various nuclei of the medulla may be present.

It rarely happens that hemorrhage takes place into one of the lateral ventricles or into the third ventricle primarily; such a lesion is not, however, an infrequent accompaniment of hemorrhage into the capsule and ganglia. As a rule, paralytic and convulsive phenomena are first marked upon one side, though rapidly they become bilateral. Very often rigidity is present. Loss of consciousness is pronounced. The pulse is slow and labored, but soon becomes more frequent. A rise of temperature is also apt to ensue, the rise being, as a rule, quite marked. The respiration is labored and difficult, and may give rise to characteristic Cheyne-Stokes symptoms, due to the passage of the blood into the fourth ventricle. The symptoms of hemorrhage into the ventricles vary of course according to the manner in which it occurs. If it occurs as secondary to a hemorrhage into the capsules, ordinary hemiplegic phenomena antedate—sometimes for hours—the bilateral symptoms.

Pathological Anatomy.—I have already alluded to the pathological changes in the bloodvessels in the section on Etiology (p. 361). The degenerations commonly met with are, as I have stated, atheroma and miliary aneurysms. These consist of minute dilatations found upon the nutrient or central vessels, and are the active factors in the production of ordinary cerebral hemorrhage. The first step in the formation

[1] In an interesting case reported by Lepine (*Lyon Méd.*, 1896, lxxxii. 114) the patient presented symptoms of vertigo and of staggering toward the side of the lesion; the latter involved the left cerebellar hemisphere and inferior vermiform process, but not the peduncles.

of a miliary aneurysm has been for a long time believed to be a periarteritis. However, it is exceedingly probable that the change begins in the intima. Dana has, in fact, pointed out that in most cases the original process is located in the intima, and that, this coat being destroyed, the arterial wall is weakened, and in consequence of the strain there is a secondary proliferation of connective tissue into the outer coat, and that in this way are produced the appearances of a periarteritis. By continued dilatation the walls of these minute aneurysms become excessively thin, so that changes in the blood pressure cause them to give way. They are often quite numerous, and can, not infrequently, be discovered at the autopsy by carefully drawing the vessels from their foramina of entrance in the anterior perforated spaces, floating them in water, and examining with a magnifying-glass.

Blood having been poured out into the internal capsule or basal ganglia, a clot forms which, if it be not too large, is gradually reabsorbed, so that should the case come to autopsy some time later little trace of it can be found on section of the brain, save a linear scar still tinted perhaps by the coloring matter of the blood.

As already stated in the section on the Bloodvessels of the Brain, the lenticulo-striated arteries (see page 357) are the ones which most frequently form the seat of cerebral hemorrhage, and especially a branch more voluminous than the rest which passes along the outer surface of the lenticular nucleus and perforates the anterior portion of the capsule. So frequently is this vessel the point of origin of the lesion that it has been named by Charcot the artery of cerebral hemorrhage.

In the nervous elements we find most interesting changes. If the clot has cut across the motor fibres of the capsule, an autopsy performed at a sufficiently late period will reveal changes in the motor tract below the clot which are clearly degenerative in character. The normal white color will have been replaced by one grayish or pinkish-gray in tint, and this tract of degeneration can in many cases be followed (even by the naked eye in fresh specimens) through the crus, the medulla, and the cord. In the crus it is found to occupy the middle portion of the pes; thence it can be traced through the pons, through the anterior pyramid of the medulla, and through the crossed pyramidal tract of the cord (see Figs. 38, 39, and 40). If the degenerated areas be examined by the microscope, it will be found that the nerve fibres have disappeared, and that they have been replaced largely by connective tissue; in other words, a sclerosis of the motor tract has ensued. The degeneration of the motor tract is always limited to the upper or cerebral segment; it never involves the lower. It always ends in the white substance of the cord. The motor cells of the cord reveal no changes, and this is also true of the nerve fibres which spring from them.

Meningeal Hemorrhages.

Meningeal hemorrhages must be considered with special reference to their site and etiology. We will first consider hemorrhages occurring external to the dura. These are almost invariably the result of blows or falls upon the head. Such hemorrhages may occur, it is important to state, without fracture of the skull, and even without marked lesion of

the external integuments. They occur most frequently in the distribution of the middle meningeal artery either in its anterior or posterior division. The main trunk of the vessel is seldom injured.

Symptoms.—If the initial violence has not been sufficient to cause loss of consciousness, the patient may merely be momentarily dazed. He may complain of the pain of the blow or of headache, but in a short time these symptoms may subside or attract no further attention. However, after some time, often several hours, disturbances of consciousness supervene. The patient becomes stupid and somnolent, and finally profoundly unconscious. This preliminary interval of consciousness [1] is present as a characteristic symptom in the great majority of cases of traumatic meningeal hemorrhage. It may be of longer or shorter duration—sometimes, though infrequently, it is very brief; at others it is very prolonged. It is not uncommon for a number of hours to elapse before unconsciousness sets in, and it may even happen that several days pass by. If the initial trauma has been very violent, the symptoms of traumatic meningeal hemorrhage may be obscured by those of concussion or gross laceration of brain tissue, but in uncomplicated cases the interval of consciousness will enable us to make the diagnosis. In addition to this symptom, others of a focal character may be present, such as a hemiplegia of the opposite side of the body, or, if the blow has been received upon the left side of the head, aphasia may be noted. Hemiplegia when present is frequently complicated by rigidity and by short, sharp muscular twitchings. Occasionally these motor symptoms involve also the other side of the body, so that the arms and legs of both sides are at times rigid and at others convulsed. Sometimes these symptoms are accentuated in the legs, a paraplegia being present. We should not forget, however, that gross motor disturbances may be entirely wanting.

The pupils are sometimes dilated, sometimes contracted, at others normal. Frequently, however, one pupil is widely dilated, and this dilatation always takes place on the side of the injury. When present this symptom, known as Hutchinson's pupil, is of great value. It appears to depend upon the degree of the extravasation and upon the amount of pressure exerted upon the subjacent hemisphere. As in all forms of meningeal irritation, the pulse at first is slowed, though later it may become frequent. Respiration, especially in the period of coma, may be slow and labored. Vomiting also may be present. A marked rise of temperature is also noted in the majority of cases, and this rise is sometimes very great.

Pachymeningitis Hæmorrhagica.

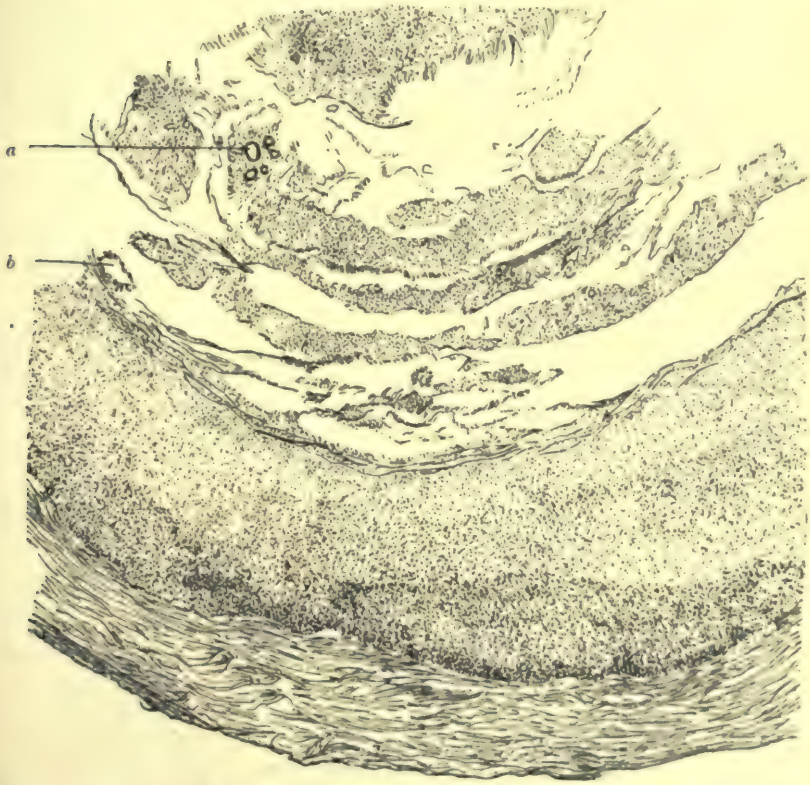
Hemorrhages occurring upon the inner surface of the dura are generally unassociated with trauma,[2] and give rise to a condition variously known as pachymeningitis hæmorrhagica, hæmatoma of the dura, or arachnoid cyst. This affection, which is rare, is found most frequently

[1] The interval of consciousness is not, however, always present. (See Battle: *Lancet*, 1894, i. 674.)

[2] Rarely, however, a history of trauma is present. (See Koell, *Correspond. Blatt. f. Schweiz Aerzte*, Basel, 1893, 783.)

in chronic and terminal insanities, and sometimes in cases where there has been a history of alcoholism or sunstroke. At times it follows exhausting fevers, and infrequently it is found in autopsies in cases of pernicious anæmia. The affection was for a long time considered by Virchow and others as inflammatory in origin. However, Wigglesworth, Hoyt, Whittaker, and the writer have pointed out that the lesions found can be attributed much more readily to hemorrhage. It is not improbable that sometimes the hemorrhage is due to a degeneration of the vessels. It occurs especially in cases in which degenerative changes are apt to be marked—namely, the chronic insane—and in which, in all

FIG. 49.



Section of membrane from a case of pachymeningitis hæmorrhagica; beginning vascularization of the deposit at *a* and *b*.

probability, the intracranial pressure is lessened, due to atrophic changes in the brain. At times, however, degenerative changes in the vessels are not evident, and we are forced to the conclusion that the exudation of blood is neuro-angio-paralytic in character. In a remarkable case published by the writer[1] there was no mental disease, no degenerative affection of the nervous system, and notwithstanding at the autopsy large hæmatomata of the dura were found covering both hemispheres. In this

[1] Dercum: *Univ. Med. Mag.*, Oct., 1889.

instance the affection was not limited to the internal dura, but affected the external dura as well, and with it the calvarium. The vessels of the external dura were exceedingly enlarged, and the calvarium was soft and spongy and was riddled with a large number of foramina containing distended vessels. The conditions found strongly called to mind the pathological changes in hæmatoma auris, and the writer has advanced the view that hæmatoma of the dura is really trophic in origin. It should be borne in mind, however, that the lesions of hæmatoma of the dura, when represented merely by layers of imperfectly organized fibrine, may be produced by a simple escape of blood, no matter what its source, into the subdural space. This was shown by Sperling, who injected blood into the subdural space of animals. It is not surprising, therefore, to learn that Hoyt has demonstrated that the hemorrhage (in cases of fibrinous deposits) at times comes from the pia.

After the exudation has taken place some attempt at organization is made, and sooner or later vessels with poorly differentiated walls are formed, which in turn give rise to renewed bleedings. Such vessels are readily seen in microscopic sections (see Fig. 49).

Symptoms.—The fact that the affection is generally met with late in the course of chronic insanity, chronic alcoholism, and allied conditions explains why we do not find the diagnosis of hemorrhage of the internal layer of the dura made frequently. However, if the patient is able to give an account of his symptoms, we find that headache, severe and persistent, is complained of. If the exudation is large and confined to one hemisphere, a hemiplegia, usually not very pronounced, may be noted. If the exudation take place suddenly, apoplectiform symptoms are present; somnolence, loss of consciousness, amounting finally to coma, convulsions, conjugate deviation of the eyes, contracted pupils, or nystagmus may be noted. Inasmuch, however, as these symptoms obtain in other apoplexies, and because the lesion most frequently occurs as a complication of chronic insanity or pre-existing cerebral disease, the diagnosis is often uncertain.

Hemorrhage in the Pia Mater.

Spontaneous meningeal hemorrhage having its origin in the pia mater is relatively rare. It occurs, however, under conditions similar to those which occasion an ordinary apoplexy; that is, middle or advanced life, renal and arterial disease. The symptoms presented are very variable, and depend entirely upon the seat and extent of the extravasation. Headache followed by convulsions, loss of consciousness, hemiplegia, and other palsies are among the symptoms met with. Spontaneous meningeal hemorrhage may occur, though rarely, in persons who are of full and plethoric habit, but otherwise well, and may ensue upon an attack of indigestion or the eating of a full meal, vomiting, or straining at stool. The attack is accompanied by sudden and agonizing headache, sometimes localized and at other times diffuse and radiating. Vertigo and mental confusion are generally marked. The patient is pale and depressed, as though suffering from excessive shock. The pulse rate is much diminished. Should the hemorrhage be extensive, unconsciousness may supervene, and the symptoms then closely resemble or are

identical with apoplexy due to hemorrhage into the cortex. Convulsions may be present. More frequently the hemorrhage is small, and gives rise mainly to symptoms referable to meningeal irritation, and these may persist for many days.

Cerebral Embolism and Thrombosis.

Etiology.—Apoplexy, instead of being caused by hemorrhage, is frequently caused by the arrest of circulation in a vessel. Thus a vessel, frequently a branch of the middle cerebral, is suddenly plugged by an embolus carried from some distant part, or, due to local disease of the wall of a vessel, a deposit of fibrine may take place, and this may increase until the lumen of the vessel is occluded. In the case of embolism the plug most frequently consists of a minute fragment of a granulation derived from a diseased heart valve. It may, however, be derived from other sources. Thus it may have its origin in abscess of the lung, though in such instance the emboli are generally numerous and minute and obstruct vessels of very small calibre. It is possible also, at times, for a fragment to have its origin from a diseased and atheromatous aorta.

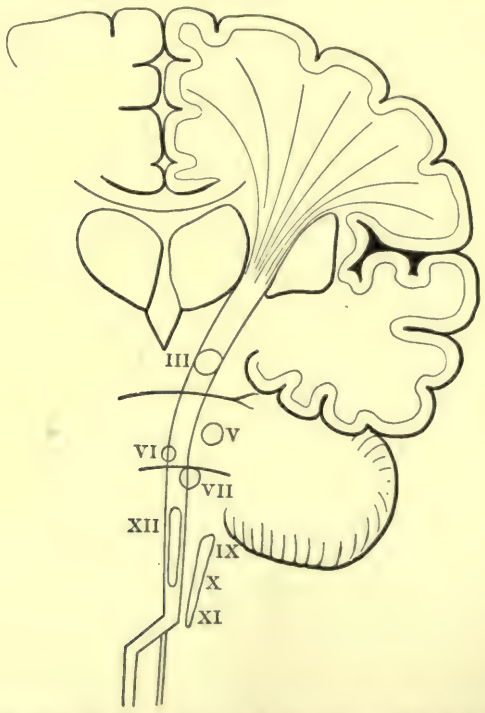
Among the causes of thrombosis, in addition to syphilis, should be mentioned atheroma. Here the change in the inner coat of the vessel is such as to lead to a deposit of fibrine and to the final formation of a clot occluding the lumen of the vessel. This deposit is much more apt to take place if the blood has undergone some change predisposing it to deposit of fibrine or to coagulation, as in acute inflammatory diseases and in certain other affections, such as tuberculosis and cancer, in which a profound deterioration of the blood takes place. Indeed, altered blood-states—*e. g.* chlorosis—may without the existence of atheroma produce thrombosis of cerebral vessels. It can readily be seen also that anything which retards the free flow of blood, such as a feeble or diseased heart, also favors thrombosis.

Symptoms.—The symptoms of embolism closely resemble those of cerebral hemorrhage. They are those of a sudden and gross cerebral accident. Loss of consciousness, hemiplegia, disturbances of pulse and respiration are likewise met with here, and they fail to present peculiarities which in themselves lead to the differential diagnosis between the cerebral insult of hemorrhage and the cerebral insult of embolism. Loss of consciousness may be equally profound. The pulse may be equally slow, respiration alike labored and stertorous. Nor is there anything in the subsequent rise of temperature to distinguish the attack from an ordinary hemorrhage. A clinical fact, however, remains that embolism is slightly more frequent upon the left side of the brain than hemorrhage. A right-sided hemiplegia, coupled with distinct signs of organic heart disease, argues very strongly in favor of embolism.

As might be expected, the symptoms of the subsequent hemiplegia depend largely upon the vessel which has been obstructed. The reader is asked to refer to Fig. 39 (p. 355), showing the portions of the brain supplied, for instance, by the middle cerebral artery. It will be seen, if an embolism obstruct the middle cerebral immediately at its point of origin from the circle of Willis, that not only will the vast area on the lateral

aspect of the hemisphere which it supplies be involved, but also the minute vessels entering the anterior perforated space. In such an instance gross and most extensive destruction of brain tissue, with persistent hemiplegia of the opposite side, will result. However, it more frequently happens that this vessel is obstructed just previous to the subdivision of the vessel into its four great branches. It will be seen at once that should an embolism occur at this situation and upon the left side of the brain, there would be, in addition to a marked hemiplegia of the opposite side of the body, also aphasia both motor and sensory. In such a lesion the third frontal convolution and the first temporal convolution would undergo softening, as well as the ascending frontal and ascending parietal convolutions. It can be readily understood that the symptoms will vary if the obstruction, instead of taking place at this point, will take place in any one of the subdivisions of the vessel. Embolism secondary to cardiac disease is far more frequent in

FIG. 50.



A lesion in the capsule gives rise to a hemiplegia of the opposite side of the body, without interfering with the nuclei of any of the cranial nerves. Lower down, however, in the crus, in the pons, or in the medulla these nuclei are involved, and so-called alternate or crossed hemiplegia is produced. Thus at III. a lesion will produce a hemiplegia of the opposite side of the body, plus an oculo-motor palsy on the same side; a lesion at VII. will cause a hemiplegia of the opposite side of the body and a facial palsy of the same side; a lesion at XII. will give rise to a hemiplegia of the opposite side and a palsy of the tongue on the same side.

the distribution of the middle cerebral than elsewhere. Embolism due to purulent and infectious processes in the lung or pleural cavity may affect other vessels as well, but in such instances the symptoms are less

like those of an ordinary apoplectic attack, and if due to an embolus carrying infection, the subsequent course of the case is apt to be that of cerebral abscess.

In thrombosis there are often, as might be expected, prodromal symptoms present. Drowsiness and dull headache are not uncommon, while the onset of symptoms may be quite gradual. This is not infrequently the case when the thrombosis depends upon ordinary atheromatous changes in the vessels. In syphilis of the cerebral vessels somnolence and headache are not only present, but may be very marked for some time preceding the actual hemiplegic onset; sometimes, too, other evidences of gross intracranial disease are present, such as ocular and other cranial nerve palsies. Alternate hemiplegia, which has already been alluded to, is far more frequent as the result of thrombosis than of hemorrhage or of embolism, and is especially common in cases caused by syphilis. This is doubtless dependent upon the fact that the vessels obstructed are preferably those which supply the crura and the pons, rather than those which enter the anterior perforated space. For the mechanism of alternate hemiplegia the reader is referred to Fig. 50. It will be seen that a focus of softening occurring in or above the internal capsule will give rise to pure hemiplegia; occurring at the level of the oculo-motor nucleus, it will give rise to hemiplegia, accompanied by ocular palsies of the opposite side of the body, for the reason that at this point the oculo-motor tract has already decussated, whilst the other fibres of the motor tract do not decussate until they reach the anterior pyramids of the medulla. Similarly, at the level of the facial nucleus a lesion will give rise to a hemiplegia of the opposite side of the body and to facial palsy of the same side. At the level of the hypoglossal nucleus we would have a hemiplegia of the opposite side of the body, but a paralysis of the tongue upon the same side.

General Diagnosis of Apoplexy and Hemiplegia.—The diagnosis of an apoplectic attack often presents difficulties; especially is this the case when the history of the attack cannot be definitely ascertained. If *sudden* loss of consciousness, together with loss of power of one half of the body, is present, the diagnosis of apoplexy can be made, but even then its nature—*i. e.* whether due to hemorrhage or embolism—remains to be determined. Even when an attack is of sudden onset, uræmia cannot always be excluded. Indeed, it is a fact attested by abundant evidence that although an uræmic attack usually comes on gradually, it may be accompanied by a sudden loss of consciousness, and at the same time a sharply-limited palsy of one limb or a hemiplegia in every way indistinguishable from that of hemorrhage or embolism may set in. In another class of cases—a class, fortunately, less frequently met with in private practice—we have apoplectiform seizures which closely resemble those due to actual vascular lesions, while the sequel notwithstanding shows that no such lesion has occurred. I refer particularly to the apoplectiform seizures which occur in paresis, and preferably in the prodromal and early stages of the disease. When the practitioner is summoned to a case presenting the symptoms of apoplexy, he should recall not only the possibility of hemorrhage, embolism, or thrombosis, but especially the hemiplegias of Bright's disease, and should not forget the possibility

of his patient suffering from an apoplectiform attack of paresis. It not infrequently happens that the previous history of the patient is entirely unknown to the physician, and this may enhance the difficulty, and may serve to act as a warning with regard to a hasty or too early expression of opinion regarding the nature of the attack. However, as regards the apoplectiform attacks of paresis, it is comparatively rare to meet with them in private practice, and even when met with their nature may be suspected from collateral evidence—for example, the past history gathered from friends, and also from the generally rapid disappearance of the symptoms. Apoplectiform attacks in Bright's disease are far more frequently met with, and are liable because of their close simulation of cerebral hemorrhage to be mistaken for the latter. Indeed, they are often practically indistinguishable from hemorrhage, and the difficulty of diagnosis is still further increased by the fact that hemorrhage itself is a not infrequent complication or result of Bright's disease.

In cases accompanied by a sharply marked hemiplegia it is generally necessary to await the outcome of the case before venturing an opinion as to the nature of the attack.

The majority of cases of apoplexy are due to hemorrhage. It is, however, far from possible to differentiate in the larger number between hemorrhage and the plugging up of a vessel. Certain facts may enable us to reach a probable diagnosis; for instance, hemorrhage occurs somewhat more frequently upon the right side of the brain than upon the left; again, the presence of valvular lesions of the heart is presumptive evidence in favor of embolism. However, that the presence of cardiac murmurs is not always a safe guide in making the differential diagnosis is shown by the case reported by Nammack,[1] in which an apoplectic attack occurred in a young man aged twenty-two years. Both aortic and mitral murmurs were present, and the diagnosis of embolism was made. The autopsy, however, showed the lesion to be hemorrhage. As Nammack says, the case recalls the statement of Austin Flint, that the physician who ventures upon a positive diagnosis in apoplexy incurs a risk of being convicted of an error at the autopsy. A far safer and more important element of diagnosis would appear to be the temperature. Dana's[2] studies show that hemorrhage is more apt to cause disturbances of temperature than necrotic processes. In acute softening a rise of temperature either does not occur at all or is very slight, while in hemorrhage a rise is common and may indeed be quite high. *Initial lowering* of temperature also points to hemorrhage.

The age, too, at which the attack occurs is an important factor. Cerebral hemorrhage is a disease of the middle and later period of life. Apoplectic attacks occurring at a relatively early age—say before forty-five—are not usually due to hemorrhage.

In thrombosis local prodromata, such as weakness, numbness, and tingling of the side subsequently paralyzed, are sometimes noticed for several days. We must be careful here, however, not to forget that in ingravescent hemorrhage the escape of blood may be very slow and the onset of symptoms correspondingly gradual. In embolism premonitory symptoms are absent. Loss of consciousness is apt to occur,

[1] Nammack: *New York Med. Record*, xliv. 7, 16. [2] *Loc. cit.*

but on the whole the apoplectiform symptoms are likely to be less pronounced. In syphilis, which is so frequently a cause of thrombosis, premonitory symptoms are common—*e. g.* headache, somnolence, and giddiness. The headache is apt to be worse at night. In thrombosis due to ordinary atheroma similar symptoms may be present, but they are much less marked. They consist of diffuse, dull headache, slight tingling or numbness, slight giddiness, and perhaps slight weakness of one side of the body.

The diagnosis of the paralysis resulting from the various vascular lesions above considered is, as a rule, readily made. As already stated, it assumes the form, in by far the larger number of cases, of a hemiplegia. Seen for the first time, there is readily elicited from the patient or his friends a history of a stroke, and of a subsequent improvement until the chronic stage of the paralysis was reached. The position of the patient is usually very striking. The arm is seen very frequently to be more or less fixed or held in a position of moderate flexion, while the fingers are decidedly contracted. If the face be examined, it is found that its lower half still shows the effects of the paralysis; the mouth droops slightly upon the affected side. The leg is held stiffly, and in walking is thrown far out from the body, while the toes are scraped along the ground. If the tendon reactions be examined, it will be noted that on the paralyzed side the knee jerk is markedly exaggerated, and in many cases a marked ankle clonus can be elicited. Similarly, the triceps, biceps, and wrist jerks are often found much increased. In the majority of cases no sensory losses are found. If hemianæsthesia exists, it signifies, as we have already pointed out, a lesion of the posterior third of the capsule.

It is frequently of great importance to determine the detailed nature of a hemiplegia, and for this purpose we should endeavor to differentiate between embolism and hemorrhage. The factors entering into this differentiation have already been pointed out. We should also differentiate between hemorrhage and embolism on the one hand and thrombosis on the other, especially the thrombosis due to syphilis. Inasmuch as in syphilis the lesions very frequently involve vessels of the pons and crus, we should in every case seek for signs of crossed or alternate paralysis. If in a given case we find that an ocular palsy, such as a dilated pupil or a ptosis, exists on one side while a hemiplegia exists upon the other, the evidence is strongly in favor of syphilis. In Fig. 50 (p. 376) the various situations in which lesions give rise to alternate hemiplegias are indicated. It is necessary to observe symptoms closely in order to refer the lesion to its proper location. In addition to the great frequency of crossed paralysis in specific hemiplegia, we should also bear in mind the other factors in syphilitic cases, such, for instance, as the headache and the sleep disturbances which so often precede for a time syphilitic hemiplegias; and in addition should we look for evidences of multiple nervous lesions. The history, it need hardly be stated, is also of enormous value. It cannot, however, be too strongly insisted upon at this point that by no means all specific hemiplegias are due to thrombosis of the vessels. As already stated in the section on Symptoms, these hemiplegias are every now and then due to a cerebral hemorrhage which

closely simulates cerebral hemorrhage occurring from other causes. In such cases we must rely upon such important factors as age. Gowers points out, for instance, that cases of cerebral hemorrhage occurring before forty-five years of age warrant our looking for some special cause. It is not infrequent for syphilitic cerebral hemorrhage to occur at a relatively early period of life. This fact is therefore of great significance.

In determining the nature of a hemiplegia we must not forget that every now and then hemiplegia makes its appearance in the course of tumor of the brain. Here, however, the history, course, and concomitant symptoms make the distinction relatively easy.

Prognosis.—Prognosis must first be considered in relation to the apoplectic state. Here we are met with great difficulties, and it is generally impossible in the first few hours after an apoplectic seizure has occurred to form other than a very general opinion. The reasons are obvious: first, as already pointed out, there is difficulty in determining the exact nature of the seizure; secondly, the nature of the seizure having been determined, it is difficult to determine its extent, because many of the symptoms of the early stage are often largely due to pressure, and not to actual disruption of nerve tissue. However, it may be stated, as a general rule, that unless decided amelioration, such as a more or less marked return of consciousness, sets in within the first twenty-four hours, a fatal termination may be looked for. Again, the immediate prognosis is bad in proportion to the severity or intensity of the shock. Great pallor, coldness of the surface, and feebleness of the heart's action are of course of ill omen. A steady deepening of the symptoms, or an extension of the paralysis from one side of the body to the other, or convulsive movements of the opposite or both sides of the body, are of like significance. An excessively high temperature is of similar import. Further, it should be borne in mind that cases in which the loss of consciousness and the onset of symptoms have been very slow and gradual—that is, cases of ingravescent hemorrhage—are very apt to be fatal. Irregular intermittent respiration, especially when assuming that peculiar disorder of rhythm constituting Cheyne-Stokes respiration, is also to be looked upon as auguring a fatal termination. The onset of œdema of the lungs and the occurrence of tracheal and coarse bronchial râles are also signs which precede death. As might be expected, the discovery of albumin in the urine is also a factor which tends to turn the scale against the patient.

Regarding the persistence of a hemiplegia, of its extent, and of its degree little can be said for a number of weeks after the attack. We must remember that the full extent and degree of the initial paralysis depend not only upon the fibres ruptured by the hemorrhage, but also upon those which are merely pressed upon. In other words, the initial paralysis depends upon both "direct" and "indirect" causes. Fibres which have merely been the subject of pressure recover their function as the clot is absorbed, and we are at a loss to predict, until some time has elapsed, the extent which the terminal hemiplegia will assume. However, while we cannot make an immediate prognosis in this respect, we are in a better position at the end of about two weeks. At that time a marked exaggeration of the knee jerk of the paralyzed side must be

looked upon as indicating that the motor tract has suffered seriously and permanently. A slight increase of the knee jerk is of but little significance. The early appearance of muscular rigidity on the paralyzed side is also an unfavorable symptom.

Little can be gained from a study of the cutaneous sensibility. In the larger number of cases anæsthesia, if present, rapidly improves. However, persistent anæsthesia is sometimes present, and may be associated with marked paralysis and rigidity of the leg, or with hemiataxia or with hemianopsia, and perhaps also with aphasia.

It may be stated as a general fact that the larger number of cases of hemiplegia improve, so that the patient is again able to walk, though, as a rule, with some difficulty. The maximum amount of improvement usually takes place within three months. After this period but little change ensues. It is true that persistent treatment is sometimes followed by benefit, but even when treatment has been persisted in steadily from the outset, the average case must be regarded as having reached a stationary condition at the end of a year. Regarding the prognosis in hemorrhage in other regions than the internal capsule we must be guided by general principles.

Treatment.—The first indication in the apoplectic seizure is to place the patient upon his back, with the head and shoulders slightly elevated and with the clothing loosened about the neck so that respiration may be in no way impeded. Absolute quiet is of course necessary. If the face be flushed, the pulse full and bounding, it may be justifiable to bleed, but regarding venesection, it must be borne in mind that it accomplishes little good unless it be done at the time of or very shortly after the apoplectic attack has occurred. We should remember also the great difficulty sometimes met with in making a diagnosis between a cerebral hemorrhage and an apoplexy due to other causes. Again, it must be borne in mind, while venesection may lessen the amount of blood poured out into the brain or may possibly prevent a fresh hemorrhage, that should the apoplexy be due not to hemorrhage, but to an embolism, venesection would favor the formation and extension of a thrombus. Practically, therefore, unless there are indications that cerebral hemorrhage is actually taking place at the time of the physician's visit, or that there is danger of a resumption of the bleeding, it is safer to leave venesection alone.

A plan that meets every indication. and is also perfectly safe, is to administer a brisk purgative. Inasmuch as there is marked difficulty of swallowing in the average case, the purgative administered should be of small bulk. Croton oil, because of the promptness of its action and of the smallness of the dose required, often answers the purpose very well. The writer is in the habit of using a drop of croton oil rubbed up with ten grains of calomel. This combination is readily administered in the majority of cases, and is a very efficient purgative. The calomel is also indicated, as tending to diminish the reactive inflammation about the clot.

As a rule, cupping and leeching are of little benefit, and, indeed, may be provocative of positive harm, because of the disturbance and handling of the patient which they necessitate. In those cases in which the patient is much shocked and depressed, and in which the pulse is

feeble or intermittent and the surface cold, stimulants may with great care be resorted to. Alcohol may be given in small quantities and very much diluted. Carbonate of ammonium may also be used. Sinapisms to the epigastrium are of value, as is also the "flying" mustard plaster so much used by the older practitioners—a mustard plaster which is shifted from place to place and limb to limb. Dry heat by means of hot bottles is in such cases also of service, although one must be careful, because of the unconscious condition of the patient, not to produce burns which may subsequently prove very troublesome.

In cases of average severity, it is a good plan to be conservative and to limit the treatment merely to position, to quiet, and to the administration of a purgative, especially as the measures above described are at times not only of doubtful utility, but may even be productive of harm. Bedsores should be looked for and guarded against as much as possible.

Some years ago Horsley suggested ligation of the carotid artery in cerebral hemorrhage, inasmuch as he had observed in his experiments upon the brains of monkeys that ligation of the carotid controlled very effectually the bleeding from vessels in the corresponding hemisphere. A serious and practical objection, however, to this procedure is the difficulty of making an absolute diagnosis of cerebral hemorrhage, and the fact that ligation of the carotid in a case of thrombosis would in all probability be productive of great harm. There is, however, one class of cases in which I believe this expedient should be carried out. I refer particularly to the ingravescent hemorrhages. If in a given case presenting all of the ordinary symptoms of apoplexy, with the exception that the loss of consciousness is exceedingly slow, gradual, or incomplete and the paralysis very slowly progressive, the carotid should be tied. This procedure was actually carried out in a case by Keen and the writer with the result, as we believe, of saving the life of the patient.[1] Compression of the carotid has also been suggested and at times carried out. This expedient offers the same objections as ligation, and should be limited, like ligation, to the ingravescent form; and here I believe it to be inferior to ligation. In cases also in which the symptoms, while not of the typical ingravescent form, are yet those of a steadily increasing paralysis, the writer believes that ligation should be practised. In a case seen by the writer in consultation, in which an apoplexy had occurred at four o'clock in the morning, there were present at first, it was stated, but ordinary symptoms, none of them foudroyant. Twelve hours later, however, the symptoms had become bilateral; the hemorrhage had evidently burst into the ventricles. Here is a case in which ligation at the proper time might have saved life. Lanphear[2] advocates surgical interference in hemorrhages from sound vessels beyond the circle of Willis. He thinks that operation should be undertaken as in abdominal hemorrhage. Whether this expedient can be applied to any but a very limited number of cases remains to be seen.

Regarding the treatment of the subsequent palsies we are to be guided by general principles.

[1] Dercum and Keen: *Journal of Nervous and Mental Diseases*, 1894, xxi. 586.
[2] *Transactions of the Med. Assoc. of Missouri*, 1893, 271.

When the immediate and alarming symptoms of an apoplexy have subsided, it is not unusual to administer small doses of some mercurial in the hope of influencing the absorption of the clot or the resulting local inflammatory reaction in the brain. Their use, however, is based upon theoretical indications only, and the benefit derived from them is, to say the least, problematical. Besides, we should be careful not to add to the distress of the apoplectic stroke the sufferings of a salivation. The iodides are also not infrequently prescribed with a view of stimulating absorption of the clot. To what extent, if any, they actually assist in this process we do not know. However, they are less objectionable than the mercurials, and, while positive good cannot be claimed for them, they cannot be accused of positive harm.

We must remember, further, that very common sequels of cerebral hemorrhage are rigidity and contracture. Evidently, while extensive changes are taking place in the cord, such as secondary degeneration, drugs which stimulate or excite the cord are to be avoided. There are two errors into which physicians now and then fall: The first is the administration of strychnine. No drug can be more valueless under the circumstances. It surely can in no way influence the pathological processes going on at the site of the hemorrhage, nor can it prevent the subsequent death of the fibres in the motor tract. On the other hand, it is highly probable that the drug is productive of harm. It is, to begin with, a powerful motor excitant, and appears to act especially upon the motor ganglion of the cord. Surely, when the cord is already in a condition of hyper-excitability, when the reflexes are abnormally increased, and when the muscles are in a condition of chronic spasm, it cannot be claimed that this drug is indicated.

Another error which physicians sometimes commit is the early employment of electricity. In the experience of the writer this agent may be productive of harm. It is as clearly contraindicated as strychnine. We must remember, first, that in palsies of cerebral origin the motor cells in the anterior cornua of the spinal cord and the motor nerve fibres are intact, and that for this reason stimulation of the nutrition of the muscles by electricity is not indicated. Further, to use an agent which will disturb the peripheral portion of the motor apparatus is of questionable value when chronic pathological changes of an irritative character are taking place in the spinal cord. The early use of electricity is therefore to be condemned. Only at a relatively late period, when we have reason to believe that active changes have subsided both in the cord and brain, is the use of electricity justifiable. It should be employed in the following manner: We should bear in mind that certain muscles of the paralyzed limbs tend to become spastic and bring about the secondary contractures which we have described. Surely it is bad therapeutics to stimulate such muscles. The use of electricity should be limited to those groups of muscles which *oppose* the groups concerned in producing the contractures. For instance, in applying the faradic current to the arm it should never be applied to the biceps or to the flexor and pronators of the forearm; its use should be entirely limited to the extensor group and supinators. On the other hand, in the leg which tends to assume the position of extension it is the flexor muscles on the back of the thigh and the flexor muscles on

the front of the leg which need stimulation. It should be added that the slowly interrupted faradic current is indicated rather than the rapidly interrupted current. The constant galvanic current has been suggested, and is used at various times, as an application to the muscle groups especially concerned in the contractures. Regarding this measure, all that we can say is that it is not contraindicated, but that it yields little appreciable benefit.

A very valuable agent, which gives not only a sense of comfort to the patient, but which for a time at least diminishes the contractures, is massage. When used judiciously and when combined with passive movements, the rigidity and contractures may become distinctly lessened. It cannot be regarded, however, as much more than a palliative agent.

Attention to general principles of hygiene and proper living are the real means by which actual good is accomplished. A very large number of hemiplegics recover to such an extent as to be able to walk and to attend to matters of business, and there can be no doubt that many of them become accustomed to the degree of palsy from which they suffer, and in many instances actually learn to disregard it. Over-eating and excesses of all kinds are of course to be interdicted, and the patient, as a rule, willingly complies with the rigid instructions given him, and as a result may be rewarded by an amount of general physical and mental health unknown before the seizure.

The treatment of spontaneous meningeal hemorrhage is that of ordinary apoplexy. However, in those rare cases in which a small hemorrhage occurs on the inner surface of the dura, giving rise to signs of sudden and pronounced meningeal irritation—symptoms which sometimes closely resemble a meningitis—free purgation, wet cups to the back of the neck, and even venesection, should be resorted to. It is almost always necessary to give morphine hypodermically to relieve the pain, as the latter is so severe. Ice to the head is also frequently of advantage.

Traumatic meningeal hemorrhage is most frequently extradural, and in by far the greater number of cases the source of the hemorrhage is the middle meningeal artery. Trephining is indicated, and the site of the injury generally offers a guide as to the locality. However, localizing symptoms should be sought for, as they frequently assist us in accurately determining the location of the clot. If no guide is offered either by localizing symptoms or by local signs of trauma, such as bruises or fracture, the operator should trephine first over the anterior branch of the middle meningeal, and, if the clot be not discovered here, he should immediately trephine over the posterior branch. The clot is best reached in the majority of cases by trephining one and a quarter inches behind the external angular process of the frontal bone, at the level of the outer border of the orbit (Keen). Hemorrhage from the posterior branch of the middle meningeal is best sought for by a trephine opening made at the same level as the preceding one, immediately below the parietal boss. (For further surgical details the reader is referred to works upon surgery.)

CEREBRAL ANEURYSMS.

In addition to the miliary aneurysms which play so important a part in cerebral hemorrhage, large aneurysms are occasionally found involving the larger arteries, especially of the base. Most frequently these aneurysms are found in the middle cerebral, and more often upon the left side than upon the right. Aneurysms of the basilar artery occur next in frequency. Some authors, however (*e. g.* Lebert and Killian), maintain that this vessel is involved more frequently than the middle cerebral. Third in frequency appears to be the internal carotid. According to Gowers, the other cerebral vessels are involved in the following order: anterior cerebral, posterior communicating, anterior communicating, vertebral, posterior cerebral, and inferior cerebellar. According to Hoffman, the order of frequency is as follows: first, the middle cerebral; second, the internal carotid; third, the anterior communicating; and fourth, the basilar. When occurring upon the arteries of the base the aneurysms are usually seated on some exposed portion of the vessels—*i. e.* before the arteries enter the substance of the brain. Most frequently they occur upon the main trunks of the arteries, though they are often found in the larger branches, and are especially prone to occur at points of bifurcation. Occasionally one vessel is the seat of several aneurysms or a number of aneurysms may be found upon different vessels. They may also occur in the brain substance itself, but this is excessively rare. Sometimes an anomalous vessel is the seat of the disease.

Etiology and Pathology.—The cerebral vessels are subject to the same degenerative changes as other vessels, and any lesion that weakens the vessel walls may lead to the formation of an aneurysm. Arterio-sclerosis plays here a rôle similar to that which it plays in the formation of aneurysms elsewhere. More frequently, however, special causes are at work, such as embolism, syphilis, and trauma. An embolus, for instance, may plug a vessel, and dilatation may ensue upon the cardiac side of the obstruction. At other times the embolus may be of such a character, perhaps septic, as to produce an endarteritis, which, even if the vessel has not been completely plugged, may lead to the formation of an aneurysm. It is not infrequent, therefore, to find as an etiological factor more or less marked signs of endarteritis, recent or old. As might be expected, such aneurysms are found mainly upon the middle cerebral artery. Syphilis is also a potent factor in the production of intracranial aneurysms by leading directly to vascular degeneration. The basilar artery is especially prone to suffer in this way. The other cerebral vessels when affected by syphilitic deposit are more prone to present signs of simple obstruction—*i. e.* thrombosis (see p. 380). Trauma is a relatively infrequent cause of intracranial aneurysms, though it undoubtedly is every now and then the true etiological factor, and it probably brings about aneurysms here as it does elsewhere—namely, by direct injury of the vessel wall and subsequent inflammatory degeneration.

In shape intracranial aneurysms are usually sacculated, sometimes projecting from the side of a vessel so as to appear sessile. At other times they are fusiform or cylindrical in shape. Especially is this true

of aneurysms of the basilar. As a rule, the aneurysms are not very large; they are most often as large or a little larger than a pea. Exceptionally they may attain the size of a walnut or even of an egg. They consist of thin-walled sacs and are true aneurysms, though it is not impossible that, as a result of trauma, a false aneurysm may be produced. Not infrequently the walls are lined by deposits of fibrine in various stages of organization, and arrest of the dilatation may be brought about in this way; even complete obliteration of the aneurysm may take place.

An aneurysm having formed, it compresses more or less the surrounding nervous tissue, in which, indeed, it may imbed itself, and special symptoms the result of interference with special portions of the brain and cranial nerves may be presented during life. Not infrequently rupture with hemorrhage into the brain occurs. Sometimes an aneurysm will erode the skull, and even perforation of the latter may take place—*e. g.* into the pharynx, nares, or elsewhere.

As might be expected from other etiological factors, intracranial aneurysms are much more frequent in men than in women, and are found in those periods of life, other things equal, when the causes of vascular and cardiac disease are active.

Symptoms.—The general symptoms of aneurysm resemble those of brain tumor. Headache is almost always present. At times it is made worse by muscular effort, as by straining at stool. Hebetude and vertigo may also occur. Convulsions and vomiting are not infrequent. Optic neuritis, however, is decidedly less frequent than in other forms of brain tumor. Occasionally apoplectiform attacks, due perhaps to sudden distention or to minute rupture of the aneurysm, interrupt the progress of the affection. Such attacks cannot, however, be regarded as characteristic of aneurysm, as they may also occur in other tumors.

At times these general symptoms are accompanied by more or less marked pulsating sensations or by sounds audible to the patient. At other times, though more rarely, a distinct aneurysmal murmur may be discovered by careful and repeated auscultation. It must be borne in mind, however, that such sounds may be present in other tumors than aneurysms. Again, it is worthy of note that they may be limited to certain situations—*e. g.* anteriorly or over the temporal or auricular regions—*i. e.* in the distribution of the middle cerebral artery; or posteriorly—*i. e.* in the distribution of the vessels supplied by the vertebrals. On the whole, however, it must be admitted that localization of the sounds, even if their existence is definitely determined, is both rare and difficult. In this connection it is also well to remember that Oppenheim has demonstrated that anæmic murmurs may be transmitted from the vessels of the neck to the head. Very frequently an aneurysm produces no symptoms whatever unless it ruptures, when its presence may be revealed by autopsy.

Special symptoms may be present, dependent upon the location of the aneurysm. For example, aneurysms of the middle cerebral may produce symptoms more or less unilateral in character—namely, hemiplegia and often convulsions. Again, if the aneurysm be extensive or be situated at such a point as to involve the branch given off to the third frontal convolution on the left side, aphasia may be present.

Aneurysms of the basilar artery may give rise to various pontine symptoms. There may be unilateral paralysis, which may be attended by the usual signs of a crossed or alternate hemiplegia. At other times the pontine symptoms are bilateral, and then palsies referable to both sides of the body are present. Of the cranial nerves, the fifth and the facial nerves are most frequently involved.

When aneurysms occur upon the internal carotid—say in its course through the cavernous sinus or just beyond this—symptoms are usually present indicative of interference with the third nerve. The paralysis of the third nerve may at first be limited to individual branches, but later, and not infrequently early, all branches seem to be equally involved. The optic nerve is also apt to suffer from compression, so that blindness may sooner or later supervene on the affected side. It is important in this connection to bear in mind that the fulness of the ophthalmic veins, œdema of the conjunctiva, of the eyelids, and, indeed, of the orbital tissues generally, so typically seen in thrombosis of the cavernous sinus, is absent here. Not only is the flow of blood to the cavernous sinus imperfectly interfered with, but the communication with the facial vein is such as to relieve any venous overfilling that may be present.

An aneurysm of the anterior cerebral artery, if it produce symptoms at all, may do so by interfering with the optic nerve and giving rise to dimness of vision, but no oculo-motor palsies are present, as in aneurysms of the internal carotid. Extensive aneurysm of the anterior cerebral may of course give rise to unilateral symptoms of various kinds, such as hemiplegia and, if on the left side, aphasia.

Aneurysms of the posterior cerebral may give rise to pontine symptoms, more especially crossed hemiplegia. Aneurysm of the cerebellar arteries cannot be diagnosticated, save perhaps in very exceptional instances. Aneurysms of the circle of Willis are apt to give rise to general symptoms only, unless they be sufficiently large to compress neighboring structures, such as the optic nerves, optic chiasm, optic tract, or crura. Paralysis of the third nerve without loss of vision would locate the aneurysm in the posterior communicating artery.

Diagnosis.—When the aneurysm gives rise to symptoms, the latter, as already stated, may resemble those of brain tumor. They may be distinguished from the latter, however, by the focal symptoms suggesting location in a special vessel at the base; secondly, in the infrequent occurrence of optic neuritis. If aneurysm at the base be suspected, we may differentiate between the various vessels upon the lines that have already been indicated in the discussion of the Symptoms (p. 386). Thus an aneurysm of the anterior cerebral may give rise to impairment or loss of vision upon one side, and, if sufficiently large, to hemiplegic symptoms on the opposite side of the body. Oculo-motor palsies, it should be remembered, will be absent. If the aneurysm be upon the internal carotid, on the other hand, loss of vision in the eye of the same side sooner or later follows, and there are also present marked, generally early, signs of oculo-motor palsy. If the aneurysm be upon the posterior communicating, oculo-motor palsy may be present, but in this instance without impairment of vision. If the posterior cerebral be involved, oculo-motor symptoms are again present, and also without loss

of vision, but now accompanied by a hemiplegia of the opposite side of the body, due to the pressure of the aneurysm upon the crus.

An apoplexy due to the rupture of an aneurysm is attended, as a rule, by very pronounced symptoms. However, at times the escape of blood is so gradual that the symptoms come on comparatively slowly. Diagnostic features of a differential value of an apoplexy due to intracranial aneurysm are wanting. However, a rupture of an aneurysm is perhaps suggested when the apoplexy is very pronounced, and is atypical as regards the motor symptoms, and also when such symptoms occur in a person the subject of syphilis, of disease of the heart, or when there is a history of severe trauma of the head; in every instance, however, other conditions must if possible be excluded.

Prognosis.—If the diagnosis has been actually established, the prognosis is necessarily serious. By far the larger number of aneurysms of the brain rupture sooner or later. The course of aneurysms of the brain is relatively rapid—more rapid than that of tumors. Their duration without rupture appears to depend to some extent upon their origin. Aneurysms the result of an infecting embolus pursue a much more rapid course than aneurysms due to simple degenerative processes in the vessel walls or to specific disease. Some cases rupture after they have been under observation for a few weeks or months. Others, again, may persist for a number of years.

Treatment.—As in aneurysms elsewhere, two methods of treatment are applicable—*i. e.* medical and surgical. Internally the iodide of potassium may be given, and it appears to act as it does in aneurysms of other vessels. We must remember, also, that syphilis is a not infrequent cause of aneurysms of the base of the brain, especially of the pons, and that this may explain the occasional value of the iodide of potassium. Laxatives should also be administered. A non-stimulating diet, a quiet life, the avoidance of all excitants or exposure should be advised. If the diagnosis of aneurysm be reasonably well established, it is perfectly proper to attempt surgical interference. This has been done repeatedly in aneurysm of the internal carotid by ligation. In aneurysm of the basilar it would be perfectly justifiable to attempt ligation of the vertebrals.

ŒDEMA OF THE BRAIN.

Etiology.—Œdema of the brain is an affection that is always secondary to some other pathological condition. It cannot, therefore, be regarded as a distinct clinical entity. For instance, œdema of the brain is met with in such cases of extensive disease of the intracranial structures as are accompanied by considerable loss in volume. Here the œdema of the brain tissue and of the membrane is compensatory in character. A notable example of œdema due to this cause is seen in the brain in advanced paretic dementia. Again, all causes of obstruction in the circulation within the cranium, such as occlusion of the sinuses, especially of the longitudinal sinus, may result in œdema of the brain. Causes of obstruction of the general venous circulation, more espe-

cially disease of the heart, may also produce this condition. Thus it may be met with in the terminal periods of mitral insufficiency, or, on the other hand, in mitral stenosis. It also occurs in the last stages of Bright's disease, and is commonly, if not always, present in uræmia. Œdema of the brain is also met as the result of acute alcoholic intoxication. Frequently, too, œdema of the brain substance and membranes is met with in general infectious diseases, even when there has not been present a frank meningitis.

The SYMPTOMS of œdema of the brain are, as a rule, overshadowed by those of the disease to which the œdema is due. However, drowsiness, stupor, or unconsciousness more or less marked are among the special symptoms present. It is not improbable that the convulsions met with in uræmia are really due to the concomitant œdema of the brain. In this connection also we should bear in mind the occasional occurrence of uræmic hemiplegia or uræmic hemieclampsia, the symptoms of which are probably due to œdema.

The TREATMENT of cerebral œdema is of course the treatment of the underlying disease.

CEREBRAL ANÆMIA AND HYPERÆMIA.

IT was formerly the custom to speak of anæmia and hyperæmia of the brain as though they were well-defined clinical entities. There is, however, reason to believe that these conditions are very rare, at least as primary affections. The researches of Geigel have shown that large fluctuations in the sum total of blood in the vessels of the brain do not occur, and it is extremely probable that the symptoms present in a given case are due rather to the *rate* of the flow of blood through the vessels than to their being unusually full or unusually empty. According to this view, a slowing of the flow of the blood through the brain means a a lessened interchange between the tissue of the brain and the blood, and symptoms are present corresponding to anæmia; conversely, an increase in the rate of the blood gives rise to the symptoms of hyperæmia. Whatever may be the real pathological condition, we must bear in mind that it is in the vast majority of cases secondary to general disturbances. We should remember, further, that the symptoms of nervous exhaustion are not those of cerebral anæmia; in the past the cerebral symptoms of neurasthenic subjects were, only too frequently, falsely attributed to this condition.

SYMPTOMS OF CEREBRAL ANÆMIA.—The symptoms of undoubted cerebral anæmia—such, for instance, as occurs in excessive general loss of blood—consist of ringing in the ears, dimness or loss of vision, dilatation of the pupils, vertigo, nausea, vomiting, headache, mental confusion and delirium, insomnia, and attacks of fainting. If the patient lie down, the symptoms are generally lessened, and this is true to a greater extent when the head is made to assume the position of the lowest or most pendent portion of the body.

Symptoms similar to those met with as the result of exhausting hemorrhage are such as accompany an ordinary attack of fainting or

any other sudden arrest of the heart's action. General anæmia and the various diseases of the blood, chlorosis, leucæmia, and the various cachexias and inanition, may be occasionally accompanied by cerebral symptoms suggesting cerebral anæmia.

Symptoms of Cerebral Hyperæmia.—Like anæmia, hyperæmia of the brain is generally accompanied by symptoms of some general affection. It is present in two forms—active or arterial and passive or venous. It is probable that the symptoms presented in the initial stages of meningitis and of some of the exanthemata and simple febrile affections, are due to an active cerebral hyperæmia. Among them, as is well known, are such symptoms as photophobia, auditory hyperæsthesia, confusion of thought, headache, and delirium. At other times, too, it is not improbable that hypertrophy of the heart, even when the latter is compensatory, is accompanied by active cerebral congestion. In such persons unusual exertion or the taking of stimulants so excites the heart's action as to give rise to a sense of fulness in the head, headache, throbbing sensations, cerebral excitement, tinnitus, vertigo, and insomnia. Whether hyperæmia of the brain occurs as a complication of neurasthenia is extremely problematical. It is probable that the symptoms in the last-mentioned affection are due to the exhausted condition of the nerve centres, and not to any disturbance of the circulation.

Passive cerebral hyperæmia or venous congestion results more especially from general obstruction, such as is present is diseases of the heart—for example, mitral stenosis and obstruction of the vena cava. It is also met with in emphysema. The symptoms of passive cerebral hyperæmia are not well defined. There is often a dull headache, some tendency to somnolence, and general weakness. The symptoms very much resemble those met with in œdema of the brain—a condition which is doubtless frequently associated with passive hyperæmia.

The treatment of cerebral anæmia or of cerebral hyperæmia is naturally the treatment of the associated and underlying conditions. Thus, the treatment of anæmia associated with loss of blood, chlorosis, leucæmia, general anæmia, and the various cachexias is of course the treatment of these various states. Similar remarks apply to cerebral hyperæmia. Thus in the case of disease of the heart attended by general venous obstruction our remedies are to be directed to this condition primarily. In the case of suppressed menstruation or of threatened apoplexy the remedies especially applicable to these conditions are to be employed. Symptoms of heat and fulness of head, ringing in the ears, vertigo occurring in plethoric persons, should be met by active purgation, general or local bloodletting, and the administration of bromides. If there should be reason to believe that a case of neurasthenia is complicated by cerebral hyperæmia, we should employ, together with other modes of treatment, the bromides, and especially ergot. Hot foot-baths or sitz-baths are also to be borne in mind.

THROMBOSIS OF THE SINUSES OF THE DURA MATER.

Thrombosis of the sinuses of the dura mater possesses a peculiar importance, because of the relation which the sinuses bear to the venous circulation of the brain, to various cranial nerves and sense organs, as well as to certain portions of the extracranial venous circulation. There are present various general symptoms indicative of gross intracranial lesions, and in addition various external phenomena, such as localized pain and tenderness and swelling at certain points—points at which the intracranial and extracranial venous circulations communicate. Thrombi in sinuses owe their origin to two distinct conditions: First, there may be present an altered state of the blood, which, when associated with a feeble circulation, may lead to deposits of fibrine. Such conditions are present in various adynamic states—for example, in the last stages of phthisis, in grave chronic diarrhœa, in typhoid fever, in cancer, in chlorosis, in profound anæmia, and, at times, in general septic conditions. Thrombi occurring under such circumstances are more frequently found in the longitudinal sinus than elsewhere. The slow rate of the blood current in this sinus, as well as the shape of its cavity and the numerous trabeculæ which traverse the latter, presents conditions peculiarly favorable to the formation of thrombi. Thrombi of adynamic or hæmic origin are practically never found in the other sinuses.

The other conditions which lead to sinus thrombosis are purely local, and consist either of a lesion of the sinus wall or of inflammation of veins emptying into or communicating with the sinus. The most common cause is inflammation of the wall of a sinus due to disease of some contiguous structure, such as purulent otitis media with disease of the temporal bone. Brain abscess, meningitis, or suppurative inflammation of any cranial bones contiguous to sinuses may give rise to thrombosis. Suppurative inflammations of certain cavities whose venous supply communicates with the sinuses, such as the orbit and the nares, may also give rise to this condition. To the list of causes we should also add inflammation of various structures external to the skull, such as phlebitis, cellulitis, erysipelas, and carbuncle. Wounds of sinuses and fractures of the skull also occasionally lead to thrombosis. Sonnenburg[1] records an interesting case of shot wound of the head followed by thrombosis of the cavernous sinus with characteristic symptoms.

A clot having formed in a sinus, it may extend into the veins of the pia mater, and as a consequence the flow of the blood in these veins is arrested, and the areas of the brain which they supply may become at first œdematous, and later undergo destructive softening. If the thrombi occur at certain points at which the extracranial veins communicate with the intracranial venous circulation, various local swellings, as already stated, occur, and in such case striking and characteristic symptoms make their appearance. In structure the thrombus consists usually of a laminated clot. When it owes its origin to purulent disease of bone or of some cavity, it may itself be purulent. Later, indeed, by extension into the veins of the neck it may give rise to general pyæmic infection or to metastatic abscess of the lung. By far the greater number of thrombi of the sinuses follow purulent disease of the mid-

[1] *Berlin. klin. Wochenschrift*, May, 1896.

dle ear; for instance, of 128 cases collected by Alport,[1] 118 followed chronic disease of this structure.[2] Sinus thrombosis occurs most frequently in youth and early adult life—that is, between seventeen and twenty-six years of age. A relatively large number of thromboses of the sinuses occur in infancy, more especially in the first three years of life. These are, however, due to various adynamic conditions, exhausting diarrhœas, and febrile affections.

Symptoms.—Thrombosis of a sinus gives rise to two groups of symptoms—namely, general and special. One or the other of these groups may predominate according to the location of the lesion. Thus in thrombus of the longitudinal sinus the symptoms may be so general that they may not be recognized, and the lesion may only be detected at the autopsy. Occasionally also the symptoms may be masked by other conditions. More frequently, however, decided general symptoms are present, such as severe headache, vomiting, rigors, fever, and slow pulse; at times also convulsions, and even rigidity of the back of the neck, may be observed. The patient is also apt to be dull and confused, or he may be delirious, somnolent, or stuporous. Sometimes palsies are added, and these may be pronounced and hemiplegic in type, and are due apparently to interference with the circulation of various cortical areas. When, as is not infrequently the case, a thrombus of a sinus is complicated by a purulent meningitis or brain abscess other symptoms are added which may complicate and obscure the symptoms of sinus disease.

The various general symptoms which we have detailed, it can readily be seen, are of themselves of but little value in making a diagnosis. Indeed, a diagnosis of sinus thrombosis can only be made when certain special symptoms are present which point to obstruction of a sinus. It is evident that these symptoms vary with the particular sinus involved, and it will be necessary for us to study the symptoms presented by thrombus of the lateral, longitudinal, and cavernous sinuses separately and in detail.

From what has been stated in regard to the relation of sinus thrombosis to disease of the middle ear, it can be readily understood that thrombosis occurs far more frequently in the lateral sinus than elsewhere. The most common cause of disease of the lateral sinus is caries of the petrous bone, the caries in turn depending upon disease of the middle ear; therefore in thrombosis of the lateral sinus evidences of chronic inflammation of the middle ear are usually present. Not infrequently the sinus thrombosis is preceded by a severe earache or intense headache. Later on, when the thrombosis has become established, fever, pyæmic in character, vomiting, and optic neuritis may be added. Rarely, nystagmus has been noted. When the involvement of a sinus is complete special symptoms, due to interference with the veins which communicate with the extracranial veins, are present. The lateral sinus communicates with these veins by means of, first, a small vein which passes through the posterior condyloid foramen, and, secondly, by means of another small vein which passes through the mastoid foramen (see Figs. 51 and 52). The latter, which

[1] *Journ. Amer. Med. Assoc.*, 1892, xix. pp. 690, 725.

[2] See also Jansen: *Archiv f. Ohrenheilk.*, Leipzig, 1893, xxxv. and xxxvi.

is the most important, communicates with the posterior auricular vein, and indirectly with the occipital vein and external jugular. When the lateral sinus is obstructed there is venous fulness and œdema back of the ear in the mastoid and occipital regions. The swelling is also tender to pressure, and especially so over the mastoid foramen. Bennett has called attention to this point.[1] Further, inasmuch as thrombosis of

FIG. 51.

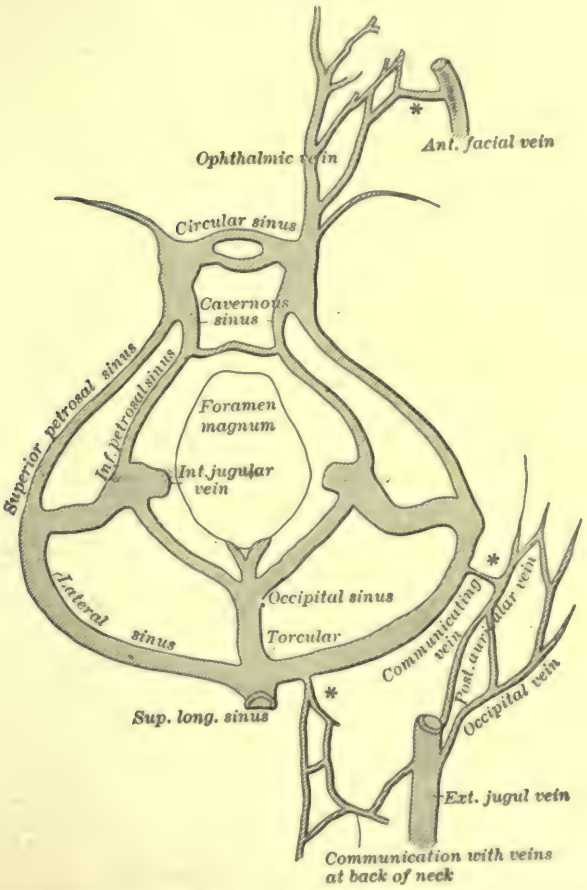


Diagram showing the communications existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by * (Leube).

the lateral sinus cuts off the flow of blood into the internal jugular vein, the external jugular empties itself more readily into the common trunk. Therefore this last-named vessel is less full than the corresponding vessel of the opposite side. This symptom, as has been pointed out by Gerhard,[2] becomes more evident by efforts at inspiration. In stout persons, however, it is sometimes difficult to judge of the relative fulness of these vessels. Again, if the thrombus has existed sufficiently

[1] *Lancet*, 1893, ii. pp. 619, 1001.

[2] *Deutsche Klinik*, 1867, ix. p. 437.

long, it may extend from the lateral sinus into the internal jugular vein for such a distance as to block the communication of this vessel with the external jugular. It can be readily seen that in such case the external jugular, instead of being less prominent than normal, is unusually distended. Further, if, as just stated, the thrombus extends into the internal jugular vein, the signs of deep-seated phlebitis of the neck are present. There is tenderness over the course of the internal jugular, and the vessel is firm and resistant to the touch—*i. e.* cord-like. The patient also tends to incline the head toward the side of the inflamed vein, so as to relieve the tension upon the vessel as much as possible, as

FIG. 52.

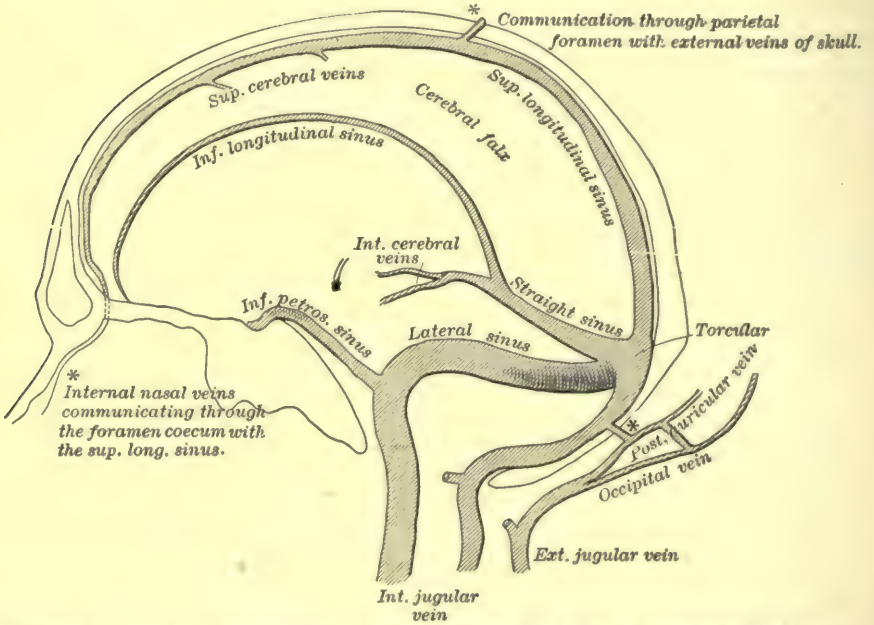


Diagram showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by * (Leube).

well as to relax the adjacent muscles. Septic infection of the lung and general sepsis occur as the disease advances. In rare cases there may be excessive slowing of the pulse, hoarseness, aphonia, spasm of the sterno-mastoid and trapezius, difficulty of deglutition, and even interference with respiration. When occurring these symptoms are properly to be attributed to irritation of the pneumogastric, spinal accessory, and glosso-pharyngeal nerves: these structures are involved in the jugular foramen, where they accompany the jugular vein. Inflammation of the vein sufficient to involve these contiguous structures must be of a high grade, probably always purulent. It must be repeated, however, that involvement of these nerves is rare.

Thrombosis of the cavernous sinus is much less common than that of the lateral sinus, but, like the latter, may be due to the extension of inflammation from contiguous structures: occasionally both lateral and

cavernous sinuses are involved at the same time. Deep-seated headache and other general symptoms already detailed are present, but, as in thrombosis of the lateral sinus, the special symptoms are the most important. They are very striking. Inasmuch as the ophthalmic vein empties directly into the cavernous sinus, obstruction of the latter gives rise to œdema of the eyelids, œdema of the conjunctiva, and, it may be, œdema of the upper part of the face. The eyeball also may be very prominent, and, if an ophthalmic examination be made, the retinal veins are found to be swollen and tortuous, and in addition there may be œdema of the retina and of the optic disk. If obstruction of the sinus be complete, these symptoms may be accentuated. There may also be venous pulsation, and retinal hemorrhages may be noted. If the thrombus be septic in character, purulent inflammation of the connective tissue of the orbit may result. In addition, various palsies of ocular muscles may be noted, and there may be exquisite tenderness and pain in the ophthalmic distribution of the fifth; associated with this symptom there may even occur a neuro-paralytic ophthalmia. These symptoms can very readily be understood when we reflect that the oculomotor nerves and the ophthalmic branch of the fifth are enclosed in the wall of the sinus, while the pathetic and abducent nerves pass directly through it.

In thrombosis of the superior longitudinal sinus symptoms pointing to some general morbid condition are almost invariably present. The lesion occurs not infrequently in various low adynamic conditions in childhood, such as result from chronic exhausting diarrhœas or long-continued fevers. It is therefore often spoken of as "marantic" in origin. It may occur under similar conditions in adults, but frequently escapes recognition, the symptoms being frequently masked by those of the general disease. As in obstruction of the other sinuses, special symptoms are present; for example, not infrequently marked epistaxis ensues from distention of the veins of the nasal cavity. The latter, it must be remembered, are in direct communication with the longitudinal sinus (see Fig. 51, p. 393). There is also cyanosis in the distribution of the anterior facial vein and œdema of the veins of both temporal and parietal regions, spreading up to the vertex. This œdema is, as a rule, quite marked in children, and is due to the arrest of the flow of blood from the external veins into the longitudinal sinus. This communication takes place through the veins of Santorini, which pass through the parietal foramina (see Fig. 51). In children the fontanelles become distended and prominent. This is due to the increase of intracranial pressure, the result of the arrested outflow from the veins of the convexity. As might be expected, headache, vomiting, somnolence, delirium, stupor, and coma are common. Symptoms indicative of interference with, or actual obstruction of, the veins supplying various cortical centres may also be present. Thus, convulsions and palsies may occur, and may be confined to or may be more marked upon one side of the body, or they may be general. Not infrequently thrombosis of the longitudinal sinus is complicated by meningitis. In such case the symptoms will be masked by those of the latter affection. In cases in which the thrombus is very slow in forming the symptoms may be very obscure, and may consist at first of nothing more than mental dulness and ill-defined

headache; the latter may often not be marked. In adults only would such symptoms as these be called to the attention of a physician. In children, as a rule, the lesion would not be suspected until complete obstruction had ensued; that is, not until characteristic signs of obstruction of the external parietal venous circulation and prominence of the fontanelles had made their appearance.

Diagnosis.—As may be inferred from the above account, many of the general symptoms presented by thrombosis of the sinuses are also met with in other affections, especially in meningitis. We need only mention such symptoms as headache, vomiting, rigors, high temperature, slow pulse, convulsions, delirium, and mental confusion. Optic neuritis, it must be remembered, may also be present. A diagnosis of thrombus of a sinus can therefore not be made unless, as above stated, special symptoms are present. These symptoms, it will be remembered, arise in most part directly from the obstruction of the particular sinus involved. Diagnosis of a thrombus of the lateral sinus, for instance, is based, among other things, upon œdema and tenderness over the mastoid process. Over the lower occipital and upper cervical regions some œdema and tenderness is also usually present. Occasionally the œdema is but slightly marked, but a painful point is always found directly over the mastoid foramen. It is due to the extension of inflammation from the sinus into the vein, which passes through the mastoid foramen and communicates with the posterior auricular and occipital veins. As Bennett has shown, this point is found about one inch behind and half an inch above the middle of the external meatus. In searching for it the finger should be passed from below upward along the posterior edge of the mastoid process. In addition, we may find that the external jugular vein upon the same side is less full than its fellow, and that the internal jugular feels hard and is tender to the touch. Above all things, however, we should examine the middle ear, inasmuch as thrombosis of the lateral sinus is especially associated with disease of this structure. We should question the patient carefully as to a history of purulent discharges, and should we learn that the symptoms followed a sudden cessation of such a discharge, and that headache, earache, and chills and fever were concomitant symptoms, the facts become very significant. It is exceedingly important to differentiate between lateral sinus thrombus and other encranial affections likewise dependent upon otitis media—namely, localized meningitis and brain abscess. In both of the last-mentioned affections symptoms resulting from mechanical obstruction of the sinus are absent. Difficulty can therefore only arise when these special symptoms are but slightly marked. Unfortunately, this is every now and then the case in children. The difficulty is further enhanced by the fact that in children suffering from headache or other severe pain, it is difficult to isolate the point of mastoid tenderness unless the tenderness at this point be very decided.

Thrombosis of the cavernous sinus is also recognized by the various special features present; these have been detailed in the paragraphs on Symptoms (p. 395). They consist especially of the symptoms dependent upon obstruction of the ophthalmic vein—namely, œdema of the eyelids, of the conjunctiva, and of the upper part of the face, together with more or less marked exophthalmos, tortuosity, and distention of the

retinal veins, and perhaps œdema of the retina and optic nerve. We should remember that thrombosis of the cavernous sinus every now and then has its origin in disease of the middle ear, and this organ should therefore always be examined. It is hardly necessary to recall the general symptoms, such as headache, somnolence, etc., for they are identical with those met with in thrombosis of the lateral sinus. In thrombosis of the longitudinal sinus the diagnosis is also to be made from the special symptoms—namely, epistaxis, œdema of the temporal regions, and fulness of the fontanelles, together with headache, somnolence, coma, delirium, and paralysis. As already stated, the lesion is frequently masked by the bodily disease upon which it is dependent, and is therefore not infrequently overlooked.

Prognosis.—Thrombosis of the sinuses is always a serious disease. If not amenable to surgical treatment, it is almost always fatal. The prognosis of thrombosis of the lateral sinus depends largely upon the stage which the disease has reached when the diagnosis is made, inasmuch as surgical interference at an early period is followed by a successful result in a large proportion of cases. In thrombosis of the cavernous sinus the prognosis is especially grave, because of the extreme difficulty of surgical interference.

Treatment.—As might be predicated, general measures are of no avail in the treatment of thrombosis of the sinuses. Surgical interference, especially in thrombus of the lateral sinus, is urgently indicated. Fortunately, thrombus of the lateral sinus occurs more frequently than any other form, and it is at the same time that form which is most amenable to surgical treatment.

Surgical interference was first suggested by Zaufal[1] in 1880, who proposed to remove the mastoid process, to open up and evacuate the sinus, and to ligate the internal jugular vein. He made his first attempt at carrying out this procedure four years later, but he was not successful. In 1886, Horsley[2] advocated an almost identical procedure, and following him a number of operators (Lane,[3] Ballance,[4] Salzer,[5] and others) have operated successfully. The operation consists of two separate parts. The internal jugular vein should first be ligated below the thrombus by two ligatures, and the vein should then be divided between the ligatures. Secondly, the mastoid antrum and walls should be opened and cleansed. The sinus should be exposed at a half inch behind and a quarter of an inch above the middle of the auditory meatus (Keen). The sinus should then be opened and cleansed by curettement until there is free bleeding. It can also be washed out from above downward, the internal jugular having been opened above the upper ligature. Parker,[6] Pickering,[7] Clutton,[8] Harris,[9] Cleghorn,[10] Battle,[11] and others have operated successfully. Körner[12] also has collected 20 cases, of which 13 were reported as successful. It is of

[1] *Prager med. Wochenschr.*, 1880, p. 576.
[2] *St. Thomas's Hosp. Reports*, 1886, xviii.
[3] *Brit. Med. Journ.*, 1889, i. p. 998.
[4] *Lancet*, 1890, i. p. 805.
[5] *Wiener klin. Wochenschr.*, 1890, p. 651.
[6] *Liverpool Med.-Chirurg. Journ.*, 1892, xii. p. 86.
[7] *Bristol Med.-Chirurg. Journ.*, 1891, ix. p. 155.
[8] *Brit. Med. Journ.*, 1892, i. p. 807.
[9] *Lancet*, 1893, ii. p. 93.
[10] *New Zealand Med. Journ.*, 1894, vii. 36.
[11] *Clin. Trans.*, 1895, xli. 409.
[12] *Die Otitische Erkrankungen des Hirns, etc.*, Frankfort a-M., 1894, p. 72.

course important that the operation be performed at the earliest possible moment.

Thrombosis of the cavernous sinus is rarely amenable to surgical interference. The expedient of opening up the lateral sinus may, however, be practised, and is perfectly justifiable: the lateral sinus should be allowed to bleed moderately in the hope of relieving, if possible, the orbital symptoms. More radical procedures can also be attempted. In a case reported by Bircher [1] there were thrombus of the cavernous sinus, thrombus of the lateral and of the inferior petrosal sinuses. Bircher removed almost the entire petrous portion of the temporal bone. The wound was freely cleansed and disinfected, and the patient recovered with diminution in the ophthalmoplegic symptoms, but with total facial palsy.

[1] *Centralbl. f. Chirurgie*, 1893, p. 483.

CEREBRAL PALSIES OF INFANCY.

By EDWARD D. FISHER, M. D.

Definition.—Under this head are included those palsies or paralyses which are prenatal or occur at birth or during early life, in which the paralysis either involves both sides of the body, being diplegic, or one side, being hemiplegic, and which are characterized by marked rigidity, spasticity, exaggeration of reflexes, and little, if any, atrophy. They mark themselves out, therefore, as distinctly different from the spinal palsies of infancy, which present atrophy of the muscles, flaccidity, and loss of reflexes. This condition was early described by Little, and his name has been associated with the disease, especially the prenatal and birth forms.

Etiology.—The causes of the disease are various. In the prenatal cases injury to the embryo may have resulted from a fall or a direct blow, leading to meningeal or cerebral hemorrhage or to concussion or laceration, with capillary hemorrhages which result in secondary sclerotic changes in the cortex of the brain. I believe this is more common than is generally supposed. It is difficult to directly prove it, as no evidence of the primary lesion may exist beyond the later sclerotic changes, with their secondary degeneration of the motor tracts.

Hereditary syphilis, alcoholism, consanguinity, etc. bear an important part in this class of cases, probably through vascular disease expressing itself by occlusion of certain vessels, especially of the middle cerebral and its branches. This may lead to a general malnutrition of the brain, resulting in diplegia and usually mental deficiency, associated with epilepsy, or, again, to a localized lesion with hemiatrophy of the brain and the usual hemiplegia. Under this head occur the cases of porencephalus, which may be most satisfactorily explained by an occlusion of some important cerebral vessel, with a resulting breaking down of brain substance, thus forming a cavity in the brain, with a well-defined wall, filled with fluid, and frequently in direct connection with the lateral ventricle. While cerebral hemorrhage may, at times, be the cause of this condition, I believe it is less frequently so than thrombosis. Inflammatory causes may be responsible at times, as evidenced by meningo-encephalitis, ependymitis, etc. found post-mortem. This condition was first described by Strümpell, and called by him "polyencephalitis," being an actual inflammation of the cortex of the brain analogous to poliomyelitis of the cord. While this is rare, I think it may occur, being due in many cases to some definite infectious process. This theory of many of the acute diseases of the nervous system marked by a rapid onset of the symptoms, and thus showing rapid destruction of the nervous structure, is being accepted by pathologists of the present time.

Cerebral palsies, therefore, as we would be led to expect, are frequent either during or following exanthematous fevers, as measles, scarlet fever, typhoid, smallpox, and diphtheria, although in the latter more rarely, as in that disease the peripheral nervous system seems most often affected.

A very common cause, and one of much importance to the practitioner, is of traumatic origin, due to pressure of the maternal parts in tedious, delayed labors, or from an abnormally narrow pelvis, or an unusually large head on the part of the child, or, again, from pressure in the use of the forceps. The last cause is less frequent than is usually supposed. Delayed labor, even without abnormality of the child or mother, is more often the cause. It is wiser, therefore, to apply the forceps early, rather than to submit to the dangers of compression. In these cases meningeal hemorrhage is often present, usually unilateral, but often bilateral (McNutt). Or, again, there may be only some injury to the cortex of the brain, leading to degeneration and sclerosis. At autopsy, made frequently years after the lesion, it is impossible to make out which was the actual cause.

Convulsions, as a cause *per se* of paralysis, I believe are rare. They are commonly present in these conditions, and go hand in hand often with the paralysis. The initial symptom may indeed be a convulsion, but it is more probable that we have the same cause for both the convulsion and the paralysis.

Pathological Anatomy.—The morbid conditions present have been more or less indicated in giving the etiology. The essential condition is atrophy of the cortex of the brain with sclerosis. This may be generally distributed, involving both hemispheres or strictly limited to one side. The brain, as a whole, is in the first case somewhat smaller than normal, and the ventricles may be overdistended. In the latter case we observe a much-atrophied hemisphere, often one third to one half smaller than its fellow; in fact, the whole cortex may be a mere shell enclosing a greatly dilated ventricle. The convolutions appear greatly shrunken and feel hard on palpation. In these cases also, especially if the frontal lobe is involved, we find the opposite cerebellar hemisphere much smaller than its fellow. Not infrequently this loss in the motor cerebral area may be macroscopically followed in the pons and medulla. Again, the condition previously alluded to (porencephalus) may be present. The surrounding convolutions are often not involved in this instance, indicating that a definite localized lesion has existed, as in the plugging of some vessel supplying a given area of the brain. This cavity frequently involves the motor area of the brain and opens into the ventricle of the same side, which is usually much dilated, its lining membrane being continuous with the cerebral cavity. The whole condition resembles that found in maldevelopment of the brain as observed in idiots. The absence of development in the lower tracts, in the pons, and medulla is also seen in these cases. Microscopically, we find sclerosis of the cerebral cortex, with decrease in the number of the cells, and also degeneration of many of them. There is evidence of change in their character. Secondary degeneration has often been traced through the pons and medulla into the lateral tracts of the spinal cord. This is present in both forms of palsy, the hemi-

plegic and diplegic. The seat of the disease is usually in the motor area, and the class of symptoms corresponds with the situation of the lesion. The skull shows evidence in its measurements of the brain lesion. In the hemiplegic type we have a definite decrease in the size on the side involved, and in diplegia there is frequently a decrease in the general measurements. This is secondary, however, to the cerebral disease, differing in this respect from the ordinary condition of microcephalus. In the prenatal cases probably there is no complete development of the motor tracts into the spinal cord; that is, the sheaths of the nerves are never acquired. In a few cases there may be spinal hemorrhage into the meninges, compressing the cord and causing a descending sclerosis in the lateral tracts, giving symptoms of spastic paraplegia without any cerebral symptoms. This condition is, however, without doubt a very rare one.

SYMPTOMS.—In congenital cases there may be from the beginning convulsions, which are often unilateral, but more frequently general. This condition is always a very serious one, as indicating some serious lesion to the brain, especially where the labor has been tedious or forceps have been used. Especially important is it if the convulsion is unilateral, as showing either a meningeal hemorrhage or a depression of the skull; for at this time relief of a permanent character may obtained by trephining or lifting the depressed bone if the latter is the lesion. Paralysis may not be recognized at first unless carefully looked for; probably in many cases it has escaped the notice of the parents. Later, however, it is observed that there is some loss of motion and a tendency to rigidity, the limbs taking the position of flexion. According as the lesion is unilateral or bilateral we have diplegia, paraplegia, or hemiplegia. The character of the paralysis is, however, the same in all cases; that is, it is of the so-called cerebral type. We therefore observe rigidity, increased reflexes, usually flexion of the extremities, absence of wasting of the muscles to any marked degree—in fact, not infrequently an increased tonus in the muscles, due to choreiform or athetoid movements which are often almost constant. An important condition of the muscles is also the continuance of the normal electrical responses both to the faradic and galvanic currents.

The contractures and deformities are very similar to those observed in cerebral hemiplegia of the adult, and also the appearance of the limb, the reflexes, etc. The fingers of the hand are usually flexed, the thumb drawn in on the palm. The flexion of the fingers may be very marked, at times absolutely fixed, but it can, as a rule, be overcome and the hand straightened out. Any mental excitement or ordinary mental action, as talking, causes the hand to again close, showing that impulses are constantly passing to the paralyzed part, although carried through channels which are diseased, and therefore which conduct them abnormally. This conduction of impulses through partly destroyed motor tracts is the cause of the choreiform movements and also of the athetosis. It demonstrates disease of the motor tracts, and not their destruction. Again, for the same reason, when the contractures and the paralysis are not too extreme, there is frequently present marked intention tremor, not differing from that observed in multiple sclerosis, and demonstrating the kinship of the diseases so far as the involvement of

the motor area is concerned. These excessive motor disturbances are not confined to the hand or arm, but may involve the face, the lower extremities, or indeed the whole body. They are especially called out by any cerebral action, as talking or even fixing the attention on any subject. Even manipulation of the paralyzed member will excite contraction, probably reflexly through the cortex. Movements of the unparalyzed side will often excite so-called associated movements in the opposite side. This may be explained by diffusion of the impulse from one side of the cord to the other, or it may perhaps be a direct impulse from the healthy or unaffected side of the brain to that side, the original impulse being carried to the opposite hemisphere through the commissural fibres which have not been destroyed. Either theory seems tenable.

As this disease is essentially one of early life, we observe considerable interference with the growth of the limbs affected. This is espepecially seen in hemiplegia, where comparison can be made with the opposite limb. With time there is frequently some improvement, so that little facial paralysis remains, and the leg will recover more fully, as a rule, than the arm and hand. The patient, therefore, in hemiplegia is usually able to walk fairly well. In diplegia and paraplegia the gait is the characteristic one of spastic paraplegia observed in lateral sclerosis of the cord, the knees being locked and the feet dragged around in a semicircle, with inability to lift the toes from the ground. Sensory disturbance is usually absent. The integrity and growth of the bones of the extremities depend on the cells of the anterior horns ; the slight interference with their development can depend, therefore, but little on the influence of the pyramidal tracts on these cells, but rather on their limited and restricted use and consequent lessened stimulation.

The mental development is frequently profoundly affected, the usual form being idiocy or imbecility ; however, many cases escape this complete condition and may be very slightly affected. Others, again, seem to have no mental weakness. The character of the later symptoms is the same whether we have the hemiplegic or the diplegic type. Epilepsy is very common, and, in my experience, generally continues through life, although in some cases it ceases after a few years. I have observed that it is apt to recur, especially at puberty. It is not to be forgotten that in many cases, even where imbecility is present, epilepsy may not be present. The character of the seizures does not differ from that observed in idiopathic epilepsy, and usually, in old cases at least, is bilateral, not affecting in hemiplegic cases that side either exclusively or primarily—in other words, it is rarely focalized or Jacksonian in type. There are many cases, in my opinion, which should be included in this class, where the paralysis, either unilateral or bilateral, is very slight, if present at all, but in which there seems to be some rigidity, or hypertonus at least, of the muscles, with exaggerated reflexes ; some mental instability, marked, perhaps, by slowness in mental development or inability to concentrate the attention ; and also ill-defined choreiform movements which continue during childhood or in after-life. In still more marked cases, which are called " chronic chorea," and in which the mental weakness is well defined, I believe, without doubt, we have a central lesion. These

cases are more frequently observed following the exanthemata, or indeed when there is a history of delayed birth. We are dealing probably with slight cortex disease with resulting degeneration.

Complications and Sequelæ.—There are few complications to speak of, for what have already been described belong to the disease itself. The sequelæ are the later developments of the disease of the brain and as a result of the secondary degenerations of the motor tracts into the spinal cord. We have, therefore, the athetosis, the chorea, spastic contractures, deformities, and exaggerated reflexes. These are, however, all the later developments of the disease.

Diagnosis.—The characteristic form of the paralysis should direct our attention to its origin. The absence of wasting and flaccidity, and the presence of rigidity with hypertonus of the muscles, associated with exaggeration of the reflexes, diagnosticate it absolutely from infantile spinal paralysis, the most common form of paralysis of childhood. Of essential importance is also the absence of electrical changes in reaction in cerebral paralysis. The distribution of the paralysis is also usually different in these two forms of infantile palsy. In the cerebral form there is either hemiplegia or diplegia, while in spinal paralysis, although there may be the same distribution, it is not as common as paralysis of one arm or leg, or, indeed as of a leg or arm on the opposite side. The contractures also are very different: in spinal paralysis they are dependent upon the contraction of the healthy or unparalyzed muscles, and are permanent, fixed contractures which do not in any way relax. They therefore lead to shortening of the tendons of the muscles—a condition which, when it is established, can only be removed by section or lengthening of the tendons. The contraction occurring in cerebral paralysis is due to a constant impulse passing down to muscles which are themselves not entirely deprived of activity, but which are unable to receive the impulse normally. The impulse is usually more completely received by the flexors, which therefore act more powerfully and overcome the action of the extensors. When cerebral activity is lessened, as in sleep or when the patient is absolutely at rest, the contractures also lessen, and there may be complete relaxation of the muscles, the hypertonus disappearing. In extreme paralysis or where it is of long standing, so that the flexors have become shortened by long-continued over-action, the contractures become more fixed; but even in these cases the continuous action of these muscles can be felt, especially on excitement, for there will often be greater increase of the contractures for the time being. This hypertonus of the muscles explains the partial failure which is always present when the tendons of these muscles are cut, the contractured limbs straightened out, and an apparatus applied. There is some degree of improvement, but there always exists the tendency, upon physiological grounds, to a return of the contractures, the impulses from the brain constantly passing down into the muscular system, which, in itself, is still capable of response. This necessitates repeated operation in many cases. In spinal cases, if the correction of the deformity has been complete at the first operation, nothing further is required, all the muscles being then placed in the position for the most favorable action. In many also of the cerebral cases we find some mental impairment and epileptic seizures—conditions which are absent in spinal paralysis. The

differential diagnosis, therefore, should not be difficult in these two diseases, and it is really between them that we are most often called to decide.

It is more difficult when the lesion has been a slight one and the symptoms are not clearly defined. The clinical picture, however, in diplegia and paraplegia of cerebral origin and spastic paraplegia of spinal origin is very similar in many cases, and the diagnosis can often only be made after a careful examination. In spinal paraplegia, however, while we have the same spastic condition of the muscles, the exaggerated reflexes, the normal electrical responses, as in the cerebral forms, the previous history, the absence of mental weakness or of epilepsy, will generally lead to a correct diagnosis. In the spinal cases also we must carefully examine for caries of the spine or tumor of the cord, and also for any symptom which early in the disease indicates either an acute or chronic myelitis. In the spinal cases also the vesical paralysis is usually more or less marked—in fact, in cerebral cases it is not usually present unless the mental weakness is well defined, and then it is due to lack of observation rather than to paralysis. The distribution of the paralysis in spinal cases is more frequently limited to the lower extremities. The diagnosis is at times, however, very difficult, if not impossible, especially when there is no mental disturbance. The explanation of the difficulty of diagnosis in these cases is apparent, as in both instances the same tracts in the spinal cord are involved—*i. e.* the crossed pyramidal or lateral tracts—and are both the seat of the same sclerotic degeneration. In the one case the lesion has begun higher up in the cerebro-spinal axis than in the other, but with the same result, as in both cases the source of nutrition of the motor fibres—*i. e.* the cells of the cortex of the brain—has been cut off.

One other condition may, with profit, be referred to at this juncture, and that is a not uncommon paralysis found in the newly-born, due to traction on the arm during delivery, causing a traumatic lesion of some of the nerves of the brachial plexus or of the plexus *in toto*. The paralysis may not be observed during the first few weeks, but later it is noticed that the child does not use the hand or arm. The flaccidity of the muscles and atrophy, together with the loss of faradic response and the presence of the reaction of degeneration, should sufficiently differentiate it from a cerebral palsy. When these cases present themselves for diagnosis in later life the same characteristic appearance of the muscles and their permanent contracture with marked atrophy would confound them with spinal paralysis rather than with the cerebral form. The history of convulsions at the time of birth would not necessarily point to the brain as the site of the lesion, as that might be explained more probably by the history of a somewhat difficult and tedious labor, leading to cerebral pressure, and consequently to temporary irritation, but not to permanent cerebral injury.

Prognosis.—From what has already been said the prognosis must always be unfavorable, the degree of the cerebral injury defining the character and extent of the paralysis which must result. In the extreme cases, associated with imbecility and epilepsy, the outlook is a perfectly hopeless one. The epileptic seizures may indeed cease toward adult life, and some increase of intelligence may be obtained by careful instruc-

tion, but little beyond this can be obtained, and in all cases the paralysis remains unimproved. In the less severe cases the usual remedies for controlling epileptic seizures, which certainly have a tendency to increase the dementia, may affect favorably in a degree the course of the disease. When, indeed, the cause is a meningeal hemorrhage or a depression of the skull occurring at birth, an operation undertaken early may prevent a serious lesion of the brain substance. These cases present the most favorable prognosis.

TREATMENT.—This has already been alluded to in the prognosis. Much has been accomplished by painstaking instruction in these cases when more or less mental impairment exists, or even epilepsy associated with paralysis. Bourneville at Bicêtre has done much in these cases. Perhaps we might expect most from this treatment when the paralysis involves only one side, so that it is fair to presume that one hemisphere alone is involved. In this relation we are, of course, referring to the development of the mental powers, for nothing is accomplished in the process for the paralysis. Bourneville exercises and develops the unparalyzed limbs by special gymnastic exercises requiring precision of action. He also pursues a course of kindergarten instruction. Possibly the unaffected hemisphere may thus be developed more especially, and be enabled to take on special functions. It seems reasonable to suppose this possible if the process of instruction is commenced at an early stage of cerebral growth. Operative interference later in life, even at the age of six or seven years, is of little benefit so far as affecting the condition of the paralysis, but may act favorably in controlling the epileptic seizures or athetoid movements. If evidence of a thickened dura is found or the remains of a meningeal hemorrhage, these can be removed. In athetosis it is well, perhaps, to excise that portion of the cortex corresponding to the part affected. The increase of paralysis thus resulting is not of serious moment, as at best the limb is almost useless. I would advise, however, deep excision of the cortex; otherwise there will almost surely be a return of the previous condition. In such a case of partial paralysis, with athetosis of the arm and hand, under my care, excision of the cortex caused complete cessation of the athetosis for six weeks, with, indeed, an increase of paralysis. I believe it well worth the trial to make the excision much deeper than I did. After six or seven years of age the prognosis becomes more unfavorable for operation. Many cases are benefited for some time after operation, and, I think, the return of the epileptic seizures may often be due to new adhesions forming between the brain and the skull, or the scalp when the former has been removed. I have tried, therefore, and believe it of advantage, placing gold-leaf over the cortex when the dura has been removed, between it and the skull or between the pia mater and the dura. Thin plates or leaves of platinum, although I have not tried them as yet, I believe will act even better. Protection of the skull can be obtained either by a bone-flap operation when it is applicable, or by the use of celluloid, which can be cut into appropriate forms. The results have not been brilliant in these operations, nor will they ever be, but we are more than justified in attempting them in view of the hopeless prognosis otherwise.

The paralysis can be somewhat relieved in some of the less severe

cases by section of the contracted, shortened tendons and the application of proper apparatus, locomotion being made possible thereby or very much facilitated. It is necessary often to repeat the operation after some months, as the contractures are spasmodic, and not permanent and fixed, in many of those patients. Massage and special gymnastic exercises directed to overcome especially defective movements can accomplish much. The nutrition of the muscles can be maintained by electricity. Galvanism is preferable, as the faradic current is apt to increase the hypertonus which is so often present.

Medicinal treatment is not called for except in those cases associated with epileptic convulsions, and here the same rules apply as in epilepsy in general. The bromides act as favorably as in other cases, controlling to a certain degree the frequency of the seizures.

ENCEPHALITIS; MULTIPLE SCLEROSIS; CHRONIC HYDROCEPHALUS; ABSCESS OF BRAIN; ATROPHY OF THE BRAIN.

By J. T. ESKRIDGE, M. D.

ENCEPHALITIS.

Inflammation of the substance of the brain may be acute or chronic, parenchymatous or interstitial, but, so far as we know, it is always secondary and usually focal. Of primary encephalitis we have no positive information. The parenchymatous varieties, polioencephalitis corticalis—supposed, especially by Strümpell and his followers, to be an important factor in the production of the cerebral palsies of childhood—and polioencephalitis, superior and inferior, will not be considered in this article.

Acute Encephalitis.—Probably the cortical substance of the brain is inflamed to a greater or less extent in all cases of leptomeningitis, but in such a case the meningitis is the essential disease. So also tumors and abscesses of the brain are surrounded by a zone of inflamed cerebral tissue. Acute softening, due to occlusion of a cerebral vessel, is necrotic in its origin and non-inflammatory in its nature, but the changes that result from this morbid process give rise to some inflammation in the adjacent brain tissue.

Etiology.—Traumatism and infective material that has found access to the brain are the principal causes of acute parenchymatous inflammation of the brain. Contiguous inflammation, such as osteitis, is given as a cause, but this, if unassociated with sepsis, gives rise to very limited focal cerebritis, and probably this rarely occurs without the membranes being involved. Of the subacute form, which is supposed to be caused occasionally by syphilis, we know very little. Injury to the brain substance which leads to inflammation may result from blows directly to the head or from falls on other portions of the body in such a manner as to violently concuss the cerebral substance. A fall in which the impetus of the body is suddenly arrested by striking against some unyielding substance with the lower end of the spine, the buttocks, the knees, or the heels may give rise to as serious brain disturbance as a direct blow to the head. The inflammation of the brain substance that results from violence in which the cerebral tissue has not been directly injured by fracture and depression of bone, or by the missile passing into the cranial cavity, is probably due to either lacera-

tion of the brain or capillary hemorrhage. The meninges are less likely to be inflamed when the traumatism to the brain has been unattended with solution of the continuity of the bones. The white matter is more commonly the seat of the inflammation than the gray. The inflamed tissue is usually near the seat of the blow when the bone has been fractured, but when this has not occurred it is not infrequently in a distant portion of the brain from *contrecoup*. A blow over the outer portion of one orbit will often cause inflammation in the centrum ovale of the occipito-temporo-sphenoidal region of the opposite side. Blows to the vertex may result in injury to the temporo-sphenoidal region of either side. It has not been demonstrated that cerebritis in which no septic material has found access ever results in abscess. Infective substances developed in the course of acute diseases, such as influenza, erysipelas, diphtheria, typhoid fever, and smallpox, may reach the brain through one of the numerous channels and cause foci of inflammation. When the infective material reaches the brain through the bloodvessels the mechanism of the lesion is by embolism or thrombosis, a necrotic softening taking place surrounded by a zone of inflammation, but when the channel of infection has been by the lymphatics, a pure inflammatory softening occurs. The larger the brain lesion, therefore, the greater the probability that it has occurred from occlusion of a vessel. While the tendencies for inflammation of infectious origin is to go on to suppuration, such a result will not occur if the toxins are not too numerous or vigorous to be destroyed by the antitoxin properties of the blood. When, however, the blood has become charged with numerous and vigorous toxins, or from other causes its normal antitoxin properties have been greatly lessened, suppuration in any portion of the body which becomes the seat of softening or inflammation will almost invariably result. Such conditions are present in pyæmia, and inflammation due to this probably always ends in suppuration.

Pathological Anatomy.—When the changes in the inflamed tissue are sufficiently marked to be seen by the unaided eye, the affected portion is swollen, ill defined at its periphery, and its consistency is lessened. The color varies from that of nearly normal brain tissue to a deep red, the degree depending principally upon the amount of hemorrhagic extravasation, but partly upon the distention of the smaller vessels. In some cases the hemorrhagic extravasation has been so pronounced as to give rise to the name hemorrhagic encephalitis. The microscopic appearances are—dilatation of the capillaries, hemorrhagic extravasation, accumulation of leucocytes in the tissues and around the vessels, and the normal nerve-tissue elements, nerve fibres, neuroglial and ganglion cells in various stages of inflammation and degeneration. In the cases in which the inflammation is very slight the fibres and cells are swollen and less translucent than normal, with here and there some granule cells, but no disorganization of any of the nerve elements. If the inflammatory process becomes arrested at this point, a return to normal appearance of the part may take place. In the majority of instances, however, the first stage of inflammation is passed, the nerve fibres become granular, and fusiform enlargement of the axis cylinder takes place, with the formation of granule cells in greater or less abundance. Even now a partial or almost complete return to the normal appearance

of the tissues may occur, but function will undoubtedly always remain somewhat impaired. Very commonly the structures forming the nerve elements atrophy and undergo vitreous degeneration, or all the tissues liquefy, a portion of the fluid being absorbed, leaving behind a straw-colored or nearly clear fluid. Other changes may take place in the course of time. It is probable that pus never forms without infection. During the early stage, in the acute cases, after the tissues have become diffluent, the part most altered may present, to the unaided eye, all the appearances of actual pus. The foci of cerebral inflammation, due to the infective material developed in the body during the course of some of the acute infectious diseases, may contain micro-organisms.

Symptoms and Diagnosis.—The symptoms of the injuries or diseases which have been the cause of encephalitis frequently so mask those of the latter trouble that it is not always easy to determine whether the manifest symptoms are due to the primary or secondary affection. The symptoms caused by direct injury to the brain come on immediately after the infliction of the wound, those from inflammation two or three days later, while those from abscess are rarely observed before the end of the second week. Headache, vomiting, and occasionally general convulsions, coming on in a person who has been subjected to the causes that are most likely to result in cerebral inflammation, should lead one at least to suspect encephalitis. If localizing symptoms, such as convulsions, beginning locally or limited throughout to one limb or one side of the body, resulting in monoplegia or hemiplegia, were to follow the general symptoms in cases favorable for the development of cerebritis, the diagnosis might be made with considerable confidence.

Prognosis.—In cases sufficiently pronounced to render the diagnosis more than probable the prognosis is unfavorable, unless the patient can be relieved by a surgical operation, by which the broken brain tissue and extravasated blood may be removed.

Treatment.—In the early stages and in cases in which surgical interference is out of the question the treatment must consist of those means adopted for the relief of meningitis.

Chronic Encephalitis.—A chronic interstitial inflammation of the brain, limited in extent and secondary in character, occurs as the result of chronic leptomeningitis, hemorrhage, especially meningeal, of childhood, tumor, etc. A primary form is observed in at least two varieties, the multiple and diffuse scleroses.[1] Besides these, a few cases, presenting only microscopic lesions of diffuse chronic cerebritis, have been observed.[2] A case reported by Gowers presented epileptoid and vertiginous attacks, later severe headache and vomiting, intense optic neuritis, and slight pyrexia, terminating in coma and death at the end of six months. It is evident that such cases require a more detailed clinico-pathologic study before an intelligent description of chronic encephalitis can be given. A disseminated form of chronic cerebritis (sclerotic), supposed to be due to syphilis, has been described by Charcot and Gombault.[3]

Treatment.—Until further light is thrown on these cases tonics and antisyphilitic agents should be relied upon.

[1] See Multiple, Insular, or Disseminated Sclerosis, p. 410.

[2] Gowers: *Diseases of the Nervous System.*

[3] *Ibid.*

MULTIPLE, INSULAR, OR DISSEMINATED SCLEROSIS.

THERE are several forms of sclerosis of brain tissue, both primary and secondary. The diffuse or disseminated cortical variety usually associated with thickening of the pia (chronic leptomeningitis or meningo-encephalitis), found in some cases of paretic dementia, is more appropriately described in connection with that disease. In some cases of chronic epilepsy a neuroglia sclerosis has been found. Several secondary scleroses and degenerations are the result of gross lesions of the brain, and need not detain us here. Of the diffuse or miliary primary sclerosis which may affect various portions of the brain little is known. The only form of sclerosis of the brain that deserves an extended description in this connection is the multiple or disseminated variety, which when typical affects in an irregular manner all portions of the central nervous system, not infrequently the cranial nerves, and occasionally the spinal nerve roots. The two typical varieties that have received attention, especially at the hands of Charcot, are the cerebral and spinal, in which the changes are supposed to be limited in the early stage of the disease either to the brain or cord. It is probable that the disease does not begin at the same time in the brain and cord, but, commencing slowly in the upper or lower portion of the central nervous system, it does not advance far before other portions are involved in the typical cases; but in the atypical forms the diseased process may have become quite extensive, either in the brain or cord, before the other is apparently affected.

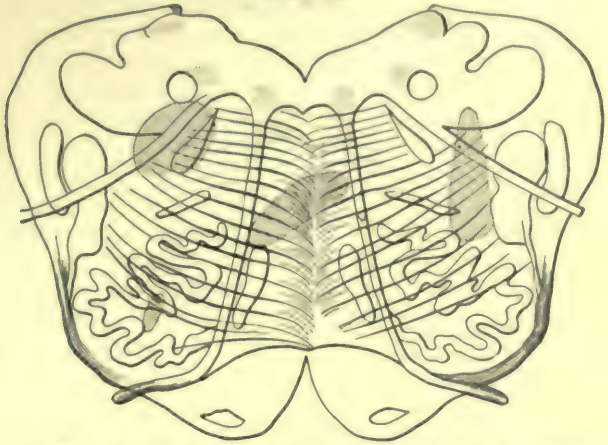
DEFINITION.—Multiple sclerosis is a chronic degenerative disease, limited largely to the central nervous system, attended by the formation of patches of sclerotic tissue, and manifested by a complexus of symptoms, the principal of which are tremor, muscular weakness, spastic rigidity, nystagmus, affections of speech, inco-ordination of muscular movements of arms and legs, and special and general sensory disturbances.

ETIOLOGY.—The disease occurs with nearly equal frequency in the two sexes. It is most common in the first half of adult life, infrequent in childhood, and extremely rare in old age. Direct hereditary influence is slight; more frequently a neuropathic tendency, with the history of cases of insanity, epilepsy, or some other nervous disease in the family, is found. Mental or physical shock, exposure to cold, worry, prolonged mental strain, or overwork may act as the exciting cause. The malady has been known to follow some of the acute infectious diseases, such as typhoid fever, smallpox, erysipelas, and diphtheria. Traumatism claims its victims, the disease following the accident one or more years after its occurrence, especially in cases in which the neuroses have been prominent. While syphilis gives rise to lesions of the cord similar to those of multiple sclerosis, it is doubtful whether the disease in its typical form is ever due to it. In nearly one half the cases no apparently adequate cause can be traced.

PATHOLOGICAL ANATOMY.—The macroscopic and microscopic changes in the central nervous system in multiple sclerosis are most striking. Irregular and sharply defined reddish-gray patches of sclerotic tissue may be found scattered throughout the central nervous tissue, and some-

times in the nerves, more commonly in the cranial. The islets vary in

FIG. 53.



The shaded areas in Figs. 53–56 represent the sclerotic patches seen in the brain and cord in disseminated sclerosis of the central nervous system.

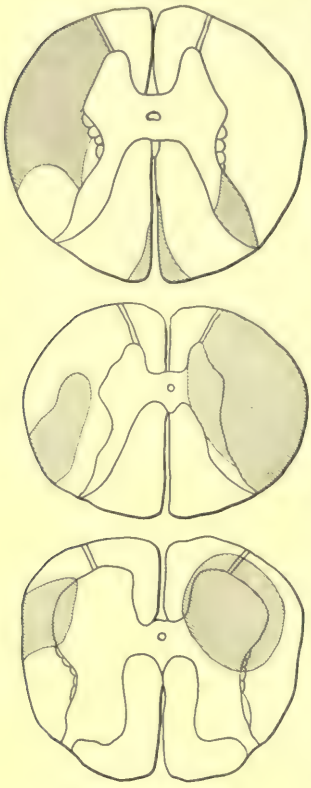
FIG. 54.

Same as Fig. 53.

diameter from a few millimetres to two or three centimetres. They are firmer than the surrounding brain tissue, but have less consistency

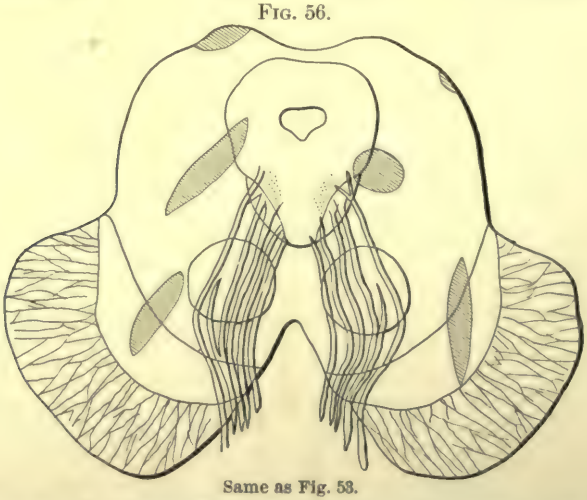
than connective tissue, except in very old cases, when they may be almost cartilaginous. They do not apparently increase the volume of the part in which they are situated, and on cutting through them in the centrum ovale they may present a depressed appearance, while in the crus, pons, and cord they may be slightly raised above the surrounding healthy tissue.[1] It has been taught that the cerebrum, the centrum ovale, central ganglia, and corpus callosum are mainly invaded by the islets. In the cerebellum they are principally confined to the white substance. Taylor claims, however, that—"1. White and gray matter are affected indifferently; 2. There is no seat of predilection for the development of the sclerotic patches; 3. The cortex of both cerebrum and cerebellum is affected."[2] In the crus and pons the islets are found on the surface and in the interior. In the medulla and spinal cord the sclerotic patches are often relatively more extensive, especially in the latter region, probably from the fact that life is compatible with greater destruction of tissue here than in the parts in which certain vital centres exist. In the cord the patches may have considerable vertical extent; they may invade horizontally a small portion of a segment, one half, or the entire substance of the cord. The cranial nerves are not infrequently involved, very rarely throughout their entire thickness, but commonly

FIG. 55.



Same as Fig. 53.

FIG. 56.



Same as Fig. 53.

[1] Gowers. [2] *Annual of the Universal Medical Sciences*, 1895, vol ii. A–68.

only a part of the nerve is gray. Sclerotic patches are sometimes found in the spinal nerve roots. The microscope shows in the sclerotic patches an increase of the connective-tissue elements, thickening of the walls of the smaller bloodvessels, and destruction of the white substance of the nerve fibres, with often the axis cylinders remaining. The preservation of the axis cylinders until the destructive process becomes complete allows nervous impulses to be transmitted, although imperfectly, through patches of sclerotic tissue, and explains some of the symptoms of this disease. Of the intimate nature of the disease little is known. Many of the supposed causes may be coincidences. The weight of evidence seems to point to the morbid process being due to an irritant, a toxin circulating in the blood. Such a blood state following the infectious fevers might lead to multiple emboli or thrombi in the capillaries of the central nervous system, and finally to an increase of the neuroglia and destruction of the nerve elements of the affected parts. Lebrun,[1] after a careful study of the etiology and pathogenesis, believes the disease has an infective origin and an endarterial inflammation takes place. Oleni[2] concludes that the point of departure is in the perivascular lymph spaces, causing an infiltration of the lymph elements and destruction from degeneration of the parenchymatous elements. That the wasting of the nerve fibres is entirely due to the increase of the connective tissue is doubtful. It is very probable that both processes result from the same cause. After the neuroglia has increased sufficiently to encroach upon the nerve fibres the wasting and destruction of the latter are thus hastened. The lesions of syphilis of the brain and cord bear a striking resemblance to those of multiple sclerosis. It is difficult to understand how such diffuse changes as are found in disseminated sclerosis—for in some cases scarcely a segment of the cord escapes—can occur from any other cause than that of a vitiated blood state. The formation of sclerotic tissue around the bloodvessels, as well as the thickening of the walls of the vessels, points to the blood as the medium through which the irritant is so widely distributed. Taylor, whose observations have been referred to, rejects the theory that the sclerosis has its primary origin in the bloodvessels, because the islets are not always found near the vessels, and in some instances these do not show any change in well-advanced degenerated areas.

Symptoms.—The first symptoms of multiple sclerosis may be apparently of a purely functional character, and unless the observer is familiar with the various manifestations of the disease in its early stages and makes a thorough and systematic examination of every nervous case, he will not infrequently mistake the symptoms of this disorder in its incipiency for those of hysteria. The reason for the seeming trivial character of the initial symptoms and their variability is not far to seek. The lesion at first is an irritative one, and before structure is destroyed function is simply lessened or perverted. A focus of irritation may take place in a portion of the cerebro-spinal axis or in a nerve, and disturb the functioning elements, but the power of resistance of the individual at the time may be sufficient to destroy the irritant and temporarily get rid of its effects, when, to all appearances, a condition of health is resumed. Such cycles of disease and health may be repeated

[1] *Annual of the Universal Med. Sci.*, 1895, vol. ii. A-68. [2] *Ibid.*

in some cases several times, and the physician may become convinced of the accuracy of his diagnosis of hysteria; but finally the integrity of structure gives way from the effects of repeated irritation, and loss of function becomes permanent. That subjects of multiple sclerosis are quite frequently hysterical should not be forgotten.

It is impossible to give in this article a reliable outline of the various symptoms by which the disease under consideration may be clothed during its irritative stage. For those who are interested in this subject no more suggestive work has been written than the little brochure by Thomas Buzzard.[1] It is quite evident that a disease attended with multiple lesions throughout the central nervous system must be manifest by numerous symptoms, and that these will vary in accordance with the seat and extent of the sclerotic patches. The symptom to which the patient's attention is first attracted most frequently is muscular weakness of the legs or arms. It may affect one leg or one arm. If one limb only is involved at first, it usually improves before a second is attacked, so that the latter is the weaker of the two. A peculiar jerky tremor of the arms may be the earliest appreciable symptom; incoordination of the legs, which usually accompanies the motor weakness, may precede the latter. In a number of cases subjective sensory disturbances, sensations of "pins and needles," numbness or dull pain, especially in the legs, have first led the patient to consult a physician. Less frequently, speech defect, eye symptoms, or evidence of the involvement of other cranial nerves is the first cause for complaint. In the typical cases loss of muscular power in the legs or arms, which is usually incomplete, being paraplegic, hemiplegic, or monoplegic in character, is soon followed by a peculiar jerky tremor in the arms, rarely to the same extent in the legs, the latter exhibiting not infrequently marked ataxia. The muscular weakness may at first be limited to a group of muscles. The parts first affected often improve for a time, but other limbs or groups of muscles become weak and the loss of muscular power slowly or rapidly increases. As time elapses the tremor becomes more pronounced—speech defect, usually a scanning speech, nystagmus, impaired vision, first affecting one eye, this improving, then the other becoming the worse, with floating specks before the eyes and temporary diplopia, are added to the catalogue of complaints. On examination the deep and superficial reflexes will be found increased, and not infrequently ankle clonus with clonus on tapping the tendon of Achilles will be present, but often more pronounced on one side than on the other. Extensor rigidity, and more rarely flexor contraction of the legs, take place. Weakness of the sphincters of the bladder and bowels is found in some instances. Buzzard calls attention to the plantar reflexes, which are usually well marked in this disease, and says that optic-nerve changes, frequently atrophy, are common. In some cases early in the disease, but commonly later in its course, apoplectiform attacks or epileptic seizures occur. Vertigo and subjective sensory disturbances are common. The latter consists of numbness, cold, or "pins-and-needles" sensations, rheumatoid pains, and less frequently of lightning-like pains, similar to those observed in locomotor ataxia. Loss of posture sense, especially in the legs, has been met with in a number of

[1] *On the Simulation of Hysteria by Organic Disease of the Nervous System.*

cases. Anæsthesia is not a pronounced symptom. When present it is often variable, disappearing and recurring from time to time. Hemianæsthesia has been observed. The most common mental change is a placid state in which the patient seems entirely unconcerned about his ailment—a condition which may be accounted for by the mental failure which prevents him from realizing the gravity of the disease. Loss of memory is common, and insanity occasionally develops and simulates paretic dementia.

From the typical forms of the disease, whose symptoms are quite variable in individual cases, there are atypical variations. In some cases a condition of spastic paraplegia is well marked, with bladder and bowel disturbance, but anæsthesia, girdle sensations, and other pronounced sensory changes, as well as trophic disturbances, serve to distinguish these from cases in which the lesion is limited to the lateral columns of the cord; while vertigo, not infrequently convulsions, and eye symptoms, such as optic nerve atrophy and nystagmus, will aid in determining the nature of the disease. I know nothing from practical experience of the purely spinal type of the disease, if it ever exists, and should be at a loss to distinguish it clinically from syphilis of the cord. The symptoms of the cerebral variety are very much the same as those of the ordinary cerebro-spinal type.

Individual Symptoms.—A peculiar jerky tremor, nystagmus, and scanning speech, when they occur together, are thought to be pathognomonic of multiple sclerosis. They probably are, but it must be borne in mind that one, or even all, of these may be absent in the early stage of this disease. Under such circumstances the association of other symptoms, upon which little stress has been laid by many writers, may be sufficient to prevent the physician at least from mistaking the disease for hysteria. One of the most striking symptoms of the disease, both on account of its frequency and its peculiar character, is the irregular jerky tremor. It is most typical and frequent in the arms, infrequently affects the legs, and still less commonly the neck, trunk, and lips. It is usually absent when the parts are at rest, but manifests itself immediately on attempts at voluntary movements, the excursions being rather small at first, and becoming more pronounced as the movements to accomplish a given object are continued. The nearer an object is approached with the hand the more marked the tremor becomes, but once the object is reached and the hand and arm have support, the tremor immediately ceases, differing in this respect from hysterical tremor, the excursions of which increase after the finger reaches an object, such as touching a given point on the wall. The tremor is so great in multiple sclerosis that a glass well filled with water is often nearly emptied before the mouth is reached. Attempts to overcome the tremor and excitement usually increase it. As it occurs, as a rule, only on intended or voluntary movements, it is called intention tremor. The exact nature of the cases of supposed multiple sclerosis in which the tremor is observed when the limbs are at rest is still in doubt. Nystagmus, most frequently lateral, but sometimes vertical, occurs in more than one half the cases. It is most marked, and occasionally only observed, when the eyes are looking fixedly at a near object. The scanning speech, also termed staccato or syllabic speech, is nearly as frequent

as nystagmus, and probably more constant than tremor. It consists in slow and deliberate utterance, with undue separation of syllables and words in speech, and frequently in eliding the ends of certain words. The patient seems indifferent to, if not unconscious of, the extraordinary effort which he is sometimes required to exert in making himself understood. Muscular weakness, which of itself is not so characteristic of the disease as tremor, is usually the earliest symptom and probably the most constant. It may affect a group of muscles and assume the form of monoplegia, paraplegia, or hemiplegia. It has these characteristics: the paralysis is rarely complete, the degree of lessened power is variable from time to time, one limb becoming weak for weeks or months, then improving, and after a lapse of months or years another limb is affected, and is weaker than the one first affected. Buzzard calls attention to an observation of a case in which atrophy of a group of muscles occurred, disappeared, and recurred with varying electrical changes.[1]

Ataxia is very common in the legs, and sometimes it affects the arms and trunk muscles. A form of ataxia similar to cerebellar ataxia occurs in some cases. A careful study of the reflexes is important. Both the deep and the superficial reflexes are often increased, and ankle clonus, frequently one-sided, or if bilateral usually more pronounced on one side than the other, is common. The eye symptoms, other than nystagmus, are quite frequent. Impaired central vision, with contraction of the fields, sometimes occurs in one eye. After a time the vision in this eye improves and the other eye becomes affected. Floating spots before the eyes are not infrequently complained of. The pupillary reaction to light and accommodation is usually normal. Diplopia is an occasional symptom. The ophthalmoscope in some cases reveals optic neuritis, which may lead to atrophy. Infrequently the facial, trigeminal, and hypoglossal nerves are affected. In cases of extensive lesions of the pons swallowing becomes difficult. Headache, usually of a dull character, is more than an occasional symptom, and occurs early in the disease. Vertigo is a symptom that occurs with almost as great frequency as nystagmus or scanning speech. It may be an early symptom, sufficiently intense to interfere decidedly with the comfort of the patient, and may be associated or entirely disconnected with vomiting. It often disappears late in the course of the disease. The convulsive seizures or apoplectiform attacks, already mentioned in certain cases, may occur early in the course of the disease and be repeated at infrequent intervals, with increased mental impairment following each seizure. Sometimes the patient passes into a stuporous or comatose condition without a convulsion, but with a rise of the body heat several degrees above the normal. In a few instances death has occurred during the stage of coma, especially when it has followed one or more convulsions. The general nutrition is usually good. Muscular atrophy and other trophic changes, depending upon the involvement of the lumbar region or anterior horns of the cord, are such as are met with in poliomyelitis or myelitis, and are extremely rare. The mental symptoms and sensory phenomena have been considered at sufficient length.

Course and Duration.—In a few cases the disease is uninterruptedly progressive, and may prove fatal in one or two years from serious

[1] *Op. cit.*

involvement of the pons and medulla. More often it is marked by repeated remissions, some varying from a few months to several years. In the early stage of the disease the patient may seem in almost perfect health during the remissions. Each recurring period of advancement of the morbid process leaves the patient in a more enfeebled condition than the preceding one. The disease, if we reckon from the beginning of the prodromal period, may run a course of from one or two years to fifteen or twenty years, the patient finally dying from some intercurrent disease, such as phthisis, or from exhaustion brought on by vesical and renal complications.

DIAGNOSIS.—In typical cases the age of the patient, the gradual onset of the disease, muscular weakness, ataxia, and increase of the deep and superficial reflexes in the legs, a jerky intention tremor in the arms, nystagmus, scanning speech, vertigo, headache, ocular symptoms, and paroxysmal disturbances, such as convulsions or apoplectiform attacks, would render the diagnosis easy. Even in such cases the disease has probably existed for some years before all the symptoms are present, and, as the irregular cases are far more numerous than the typical ones, a diagnosis is often required when all the so-called characteristic symptoms are absent. Under such circumstances errors can be prevented or lessened only by a careful study and analysis of all the symptoms and circumstances connected with each individual case. The manner of invasion and progress of the disease, together with the association of certain symptoms, if carefully considered, will usually lead to a reliable conclusion.

The diseases with which mutiple sclerosis may be confounded, especially in its atypical forms, are—hysteria, some of the other functional neuroses, paretic dementia, tumors of the brain attended with a jerky tremor, thickening of the meninges on the convex surface of the brain or in the posterior fossa from inflammation or tumor, cerebellar ataxia, bulbar paralysis, paralysis agitans, Friedreich's ataxia, spastic spinal paraplegia, locomotor ataxia, and postero-lateral sclerosis. It will be impossible in the small space assigned this article to enter into a detailed differential diagnosis between each of the above diseases and multiple sclerosis, but a few of the salient points may be given.

Hysteria and Some of the Other Functional Neuroses.—There is probably no other organic disease of the nervous system whose symptoms so frequently and so clearly resemble those of hysteria as those of multiple sclerosis in its incipient stage. In doubtful cases the first question that the physician should endeavor to answer is, "Is the disease organic or is it functional?" It should always be borne in mind that a multitude of symptoms, apparently of a functional nature, are not sufficient on which to base a diagnosis of hysteria if there is present one symptom alone pointing positively to an organic lesion. Buzzard has called attention to the following: "Multiple sclerosis, like hysteria, is common in women at puberty; a history of some moral shock often precedes both; there are few cases of multiple sclerosis in which there are not hysterical symptoms added; and many symptoms of the former have long been looked upon as hysterical."[1] The same writer states that the plantar reflexes are usually well marked in multiple sclerosis, and

[1] *Op. cit.*, pp. 97, 98, 107, 108.

feeble or absent in hysteria. Paralysis is usually sudden in its onset, and more complete and flaccid in the latter than in the former. When blindness occurs in one eye, it is generally complete at first and comes on suddenly in hysteria, whereas in multiple sclerosis absolute blindness in one eye is rare. The acuity of vision lessens gradually with contraction of the fields, until the eye is almost useless; then vision improves in this eye and fails in its fellow. Atrophy of the optic nerves and nystagmus are probably never of hysterical origin. The tremor of multiple sclerosis may be simulated by an irregular tremor occurring on voluntary movement in hysteria, but in the latter the excursions are usually less; there are a tardiness of the initial muscular effort and a contraction of the antagonistic muscles. Gowers lays considerable stress upon the diagnostic importance of the presence of the last symptom. The hysterical patient will often touch an object with her index finger without much difficulty, but after the finger has remained a few seconds in contact with the object the arm becomes affected with an irregular or jerky tremor, differing from the tremor of multiple sclerosis, in which great effort is frequently required in bringing the finger in contact with an object, but the tremor ceasing as soon as this has been accomplished (Buzzard).

Paretic Dementia.—Irregular pupils, loss of the iris reflex, marked tremor of lips and tongue, drawling speech, with inabilty to pronounce certain consonants, and prominent symptoms of dementia are always in favor of paretic dementia, but in rare cases the difficulty of diagnosis is very great. Tumors of the brain are attended by a jerky tremor manifest only on voluntary movement. The presence of the tremor is the only symptom suggestive of multiple sclerosis, and the other indications of tumor are usually sufficient to prevent a mistake if the observer is on his guard. The tremor occurring from a tumor in one crus or one side of the pons is unilateral. Chronic meningitis in the posterior fossa surrounding the pons and medulla, or thickening of the membranes on the convexity of the brain from inflammation or from tumor, may be attended with tremor, but other symptoms of these pathological processes are present.

Cerebellar Ataxia due to Tumor.—Double optic neuritis, projectile vomiting, and severe headache are in favor of tumor.

Bulbar Paralysis.—The lesion being limited to the muscles of the motor nerves of the bulb is sufficient to prevent mistaking this for a disease like multiple sclerosis.

Paralysis Agitans.—The peculiar posture of the patient and his age, the almost characteristic rigidity of the limbs, the slow initial movements in attempting to make a muscular effort, the continuance of the tremor during rest, except in the very early stage of the disease, and the absence of nystagmus and syllabic speech, are sufficient for a diagnosis of paralysis agitans.

Friedreich's Disease or Hereditary Ataxia.—In this disease and multiple sclerosis nystagmus, tremor, speech defects, and ataxia may occur. Hereditary ataxia is a family disease, multiple sclerosis is isolated. The speech defect consists in eliding the ends of the words in the former; in the latter, besides this, in spacing between syllables and words—syllabic speech. The tremor in the former is less marked than in the latter, and

is limited to the neck muscles. Knee jerks are increased in multiple sclerosis, but they are almost invariably absent in hereditary ataxia.

Spastic Spinal Paraplegia.—If the lesion in the multiple sclerosis is limited to the dorsal region of the cord, a differential diagnosis will be impossible; in other cases the bulbar and cephalic symptoms will enable the nature of the disease to be determined.

Locomotor Ataxia.—The loss of the iritic reflex and the knee jerk, with sensory phenomena and inco-ordination, without irregular jerky tremor, nystagmus, or syllabic speech, leaves no doubt in the majority of cases as to the diagnosis. Postero-lateral sclerosis has neither speech defect, nystagmus, nor a coarse intentional tremor. Little if any difficulty in determining the nature of the disease would be encountered, except in cases in which the multiple sclerosis should be limited to the spinal cord, when a diagnosis might be impossible.

Prognosis.—Remissions of variable lengths may occur, but recovery is unknown. Bulbar symptoms are unfavorable. In such cases, if the progress of the disease is steady, although gradual from the first, life will not be prolonged more than one or two years after the diagnosis is positive.

Treatment.—There is no known method of treatment by which the disease can be more than temporarily arrested. The health should be kept in the best possible condition by tonics, such as arsenic, quinine, and iron, by an abundance of nutritious food, and by careful attention to the digestive organs, bowels, and kidneys. Potassium iodide and mercury seem to affect the morbid process more than any other medical agents, especially early in the disease. Nitrate of silver has been used and arsenic has been employed hypodermically, but results have been far from brilliant. Occasionally periods of rest in the recumbent posture, with massage, appear to do good in lessening the tremor and in bringing about remissions in the progress of the disease. It is doubtful whether electricity has any other effect in this malady than keeping up muscular nutrition.

CHRONIC HYDROCEPHALUS.

Definition.—Chronic hydrocephalus is a condition brought about by the gradual distention of the ventricles (usually the third and lateral) of the brain by a watery fluid. It is often congenital, but may be acquired. An increase of the subarachnoid fluid sometimes takes place, either from arrested development of the brain or from inflammation of the meninges, and has been termed external hydrocephalus. This is entirely different from the internal form, and the morbid process which gives rise to it is the serious one, so that no further reference will be made to it in this section.

Etiology.—Beyond the fact that most of the cases begin at birth or soon after, that the disease occurs more frequently in some families than in others, and that poverty and poor nutrition seem to favor its development, we may as well admit that we are ignorant concerning the etiology of the congenital form. The acquired variety is usually due to

mechanical causes, but occasionally cases occur after early childhood for which no adequate pathological lesion has been found at the autopsy. These are the so-called idiopathic cases.

Pathological Anatomy.—All the ventricles may be enlarged and distended with fluid; more commonly the fourth ventricle is nearly normal, the lateral and third ventricles and the aqueduct of Sylvius being affected. In extremely rare cases the fourth ventricle alone is involved. When the foramen of Monro is obstructed the effusion is limited to one or both lateral ventricles. The fluid, often nearly as clear as spring water, alkaline in reaction from the presence of sodium chloride, contains a small quantity of albumin, and not infrequently urea and cholesterin. The quantity of the fluid varies from one to several pints, and is greater in the congenital cases, as the bones of the cranium readily separate and permit an extreme degree of hydrops ventriculorum. The brain suffers from compression in the congenital cases in proportion to the quantity of fluid in the ventricles, but after the cranial sutures have become ossified the brain substance may be greatly damaged by the accumulation of only ten or twelve ounces of fluid in the lateral ventricles. The white substance of the brain seems to waste more than the gray, but the convolutions are greatly flattened, and in very severe cases they may be almost completely obliterated. Under such circumstances the ventricles are converted into one enormous sac, and this is surrounded by the cerebral hemispheres, which form a wall not more than a few millimetres in thickness. The basal ganglia are flattened, the choroid plexus is thickened, the lining membrane of the lateral ventricles is roughened and granular, the normal relations of the structures of the interior of the brain are destroyed, and the bones of the cranium are thin. In the congenital cases, and occasionally in the acquired, the cranial sutures are widened and the shape of the head is distorted. Occlusion of the foramen of Monro, the aqueduct of Sylvius, the foramen of Majendie, or the foramina of Mierzejewsky, or pressure on the veins of Galen, is often found in the acquired cases, and sometimes in the congenital variety.

Symptoms.—When the condition has taken place in utero the head may be so large at birth as to interfere with delivery. In some instances spontaneous rupture has occurred and the child is born dead. In others it may be necessary to evacuate the fluid before the head will pass through the pelvis. In the cases in which the morbid process has occurred before birth and the child has been delivered alive, the head is unnaturally large and rapidly increases in size during the first weeks of extra-uterine life. The enormous size of head then presents a striking contrast to that of the face and neck, over which it projects on all sides, very much as a mushroom overhangs its own stem, so that neither the face nor the neck can be seen when the head is viewed directly from above. The sutures and fontanelles are greatly enlarged, and the head may seem like a fluctuating mass. In a child six weeks old seen by me not long since, in consultation with E. J. Rothwell, apparently less than half of the cephalic enlargement was covered with bone. The circumference of this head measured twenty-four and a half inches. Death occurred about one week later. When the disease has made much progress at birth it is rare for the child to live more

than a few weeks. More commonly the head does not begin to enlarge until some time during the first few months of extra-uterine life. It is observed that the forehead projects unnaturally over the eyes, the sutures widen, and the fontanelles enlarge. Soon the parietal bones give an angular appearance to the head by their upper portions projecting laterally outward; the occipital bone extends backward and seems more horizontal than normal. The skin of the head is stretched and thin, and may present a crackling sensation when it is touched with the finger. The head is poorly covered with hair and the veins of the scalp are unduly prominent. As the orbital plates of the frontal bones project obliquely outward, the eyes are directed downward, so that most of the cornea may be covered by the lower lids. If the child lives one or two years, the head may reach an enormous size, measuring 50, 80, or 100 centimetres in circumference, or even 107.6 centimetres (42.6 inches), as in a case reported by Klein.[1] The child is usually irritable and restless; the nutrition is poor, although a fair amount of nourishment may be consumed. Growth is retarded, and muscular weakness and sometimes contracture are well marked. The muscles of the neck are often not sufficiently strong to support the head, so that the child can neither sit nor stand unless its head is supported. Hydrocephalic children do not learn to walk before their second or third year. Occasionally convulsions are common, and nystagmus is sometimes present. Strabismus and optic-nerve atrophy are not rare. Sometimes the atrophy of the optic nerve is complete, with absolute blindness; more frequently some vision is retained. A condition of bitemporal hemianopsia has been observed in the acquired form, due to pressure on the optic chiasma. Mental defect is present and quite pronounced in most cases, although one occasionally meets with children whose heads are hydrocephalic, but who show a fair amount of intelligence. The mental defect is usually that of imbecility, rarely an extreme degree of idiocy. In the progressive cases vomiting, convulsions, and finally coma supervene, and death takes place. Even in cases in which the morbid process has reached a marked degree an arrest may take place and life be prolonged for a number of years. In those children in whom the effusion has occurred most gradually the fewest symptoms of cerebral compression are manifest. When the effusion does not take place into the ventricles until after the sutures of the skull have become united and the fontanelles closed, the head does not appreciably enlarge, and the symptoms will be those of cerebral compression and irritation, such as mental failure, muscular weakness and contracture, occasional convulsions, vertigo, sometimes vomiting, and finally coma and death. During the course of the trouble in most cases, in either the congenital or acquired, occasional febrile processes, with nausea, vomiting, and headache, occur. In some instances these febrile processes are ushered in by one or more convulsions. In the cases in which the morbid process has been arrested and the patient reaches adult life, more or less muscular weakness and mental defect persist throughout the remainder of life. Hydrocephalic children are especially liable to succumb to the usual diseases of childhood or some intercurrent malady.

DIAGNOSIS.—The diagnosis must rest upon the enlargement of the

[1] Gowers: *A Manual of Diseases of the Nervous System*, 2d ed., vol. ii. p. 586.

head and the distended fontanelles. The acquired form, taking place after the sutures and fontanelles have become permanently closed, cannot be diagnosticated, although the condition may be suspected if the symptoms follow an attack of meningitis or are associated with lesions likely to cause effusion into the ventricles. The only condition with which chronic hydrocephalus is likely to be confounded is enlargement of the head from rickets and thickening of the cranial bones from syphilis. In neither of these are the fontanelles disturbed, and in both the symptoms caused by distention of the lateral ventricles are absent.

PROGNOSIS.—This is grave in all cases, even in those in which the morbid process is arrested, as some mental and physical weakness remains, and epilepsy is likely to develop.

TREATMENT.—Beyond protecting the unfortunates from severe changes in the temperature and undue exposure of the head to the rays of the sun, and keeping the digestive organs and general nutrition in the best possible condition by appropriate measures, it is doubtful whether much can be done for them. The results of surgical procedures have not been brilliant or even encouraging. For those who have sufficient intelligence proper training, both mental and physical, should be provided.

ABSCESS OF THE BRAIN.

DEFINITION.—Abscess of the brain is a circumscribed collection of pus on the surface of the brain or within its substance, occurring as a direct result of local purulent inflammation of microbic origin.

ETIOLOGY.—One of the most important facts taught us by the brilliant achievements of modern surgery by means of thorough asepsis is that suppuration is impossible in the absence of a micro-organism. It seems strange in the face of this demonstration that in some of the recent and best works devoted to the diseases of the nervous system the position is taken that it is not proven that all cases of abscess of the brain have a microbic origin. Until it is incontrovertibly demonstrated that suppuration is possible without the presence of a pathogenic organism it seems that we are justified in assuming that suppuration in the brain, as well as elsewhere, is due directly to organismal infection. To the indefatigable labors of Macewen of Glasgow, and the marvellous results obtained by this brilliant surgeon in the treatment of abscess of the brain, the medical profession especially, and humanity in general, owe a debt of gratitude. Acute and chronic local softening of the brain of necrotic origin, due to cutting off the blood supply, takes place, with diminution of the consistency of the most affected part to that of thin liquid, and in this pus corpuscles are not infrequently found. With the many sources of infection, especially from the mucous surfaces of the nose, throat, bronchi, and intestinal canal, and with the many avenues by which infecting material may be conveyed to the brain, it is not surprising that suppuration should take place under conditions so favorable to pus formation. On the other hand, it appears remarkable that ab-

scess of the brain is not more frequent in advanced life, a period so commonly attended with disease of the cerebral arteries and resulting in imperfect brain nutrition.

The predisposing causes of abscess of the brain mainly relate to age and sex. In the third decade of life is found about one third of the cases of abscess of the brain from all causes; the second, fourth, and fifth decades, in the order named, yield the next greatest number.[1] It is exceedingly rare before the second year and after the sixtieth, and it is a very infrequent exception for it to be met with after the seventieth year. I have recently observed a case in a man seventy-five years old.[2] It occurs from all causes about three times as often in the male as in the female. The relative frequency between the sexes is least when the cause is disease of the ear—2 males to 1 female; and greatest when the cause is traumatism—5 males to 1 female.[3]

The exciting causes may be divided into local and distant.

Local Causes.—These are by far the most important factors in the causation of suppuration within the cranium, and probably give rise to more than three fourths of all cases of abscess of the brain, although actual statistics only give about 70 per cent. from local causes. We must bear in mind that septic material has been found in the bony structures containing the organs of hearing, and has given rise to abscess of the brain when disease of the ear was unsuspected. Of the local infective causes, disease of the organs of hearing stands first, and probably is the source of infection in at least one half of the cases of abscess of the brain. The statistics given by Gowers ascribe 42.5 per cent. to this cause. In most cases there is a chronic discharge from the ear of several months', or more commonly of several years', duration. Less frequently the otorrhœa has existed only a few weeks before cerebral mischief takes place. If the discharge has been free from the ear before the brain becomes infected, it usually lessens, and sometimes ceases entirely, after the brain becomes involved. A chronic otitis media that is giving little discomfort to the patient seems to be most likely to infect the brain on exposure to cold, resulting in a renewal of the inflammation of the ear. Occasionally the ear continues to discharge profusely during and after the formation of an abscess of the brain.

Recurrent attacks of otitis media on account of the changes that take place in the structures of the middle ear from repeated inflammation are more likely to be followed by intracranial suppuration than the primary ones. Caries of the petrous portion of the temporal bone and inflammation of the mastoid cells are usually found in the chronic ear diseases that are most dangerous. But infection of the brain may take place without such complications. Polypi of the ear are a source of danger, as they are a direct cause of irritation to the parts, prevent a free exit to the pus, and thus favor infection of the surrounding structures, which may lead to sinus thrombosis. Disease of the nose is a comparatively infrequent cause of abscess of the brain. The disease may be limited to the mucous membrane, but more commonly there is caries of the nasal, ethmoid, or sphenoid bone, usually of syphilitic origin.[4] I have seen two cases of abscess of one frontal lobe of the brain due to a collec-

[1] Gowers: *Loc. cit.*, p. 471.
[2] Eskridge: *The Medical News*, July 27, 1895.
[3] Gowers: *Loc. cit.*
[4] Gowers: *Loc. cit.*

tion of pus in the frontal sinus. One of them was associated with suppurative meningitis, but no disease of the bone was found in either case. Disease of the orbit, causing sinus thrombosis, may give rise to abscess of the brain. Gowers states that caries of other bones than the temporal rarely leads to abscess of the brain. Meningitis is usually associated with abscess of the brain due to disease of the nose or orbit. Tumor of the brain is a rare local cause of cerebral suppuration. I have met with two abscesses associated with growths. In one the tumor was cancerous and in the other tubercular. In each the suppuration took place in the immediate neighborhood of the growth, which seems to be the rule. I have reported one case of abscess of the brain[1] apparently due to erysipelas of the face, but as the patient was brought to the hospital without a history in a moribund condition, and I did not see him until after death, it was difficult to determine whether the sinus thrombosis and abscess formation had resulted from the condition of the face or whether the latter had been caused by the former.

Traumatism.—Injuries to the head, according to the statistics given by Gowers, cause about one fourth (23.2 per cent.) of the cases of abscess of the brain, but, as this author suggests, the proportion from this cause is even larger, because old and apparently trivial blows to the head are often forgotten. The most common form of injury that leads to suppuration in the brain is a direct blow or fall on the head, resulting in an open wound in the soft parts and fracture of the skull, which is not infrequently attended by depression of the internal table. Sometimes, instead of fracture, caries of the bone follows. Macewen regards punctured wounds penetrating into the bone as the most likely to be followed by intracranial suppuration. Hirt believes that injuries to the head unattended with an open wound, and in which there is no solution of continuity of the soft parts, are not likely to lead to abscess formation.

Distant Causes.—According to Gowers, thrush in the mouth, with a growth of oidium albicans, in two recorded cases has given rise to abscess of the brain containing the same fungus,[2] and the same author refers to a case reported by Böttcher, in which lung pigment was found in a brain abscess. It is probable that septic material from any portion of the body may find lodgement in the brain under favorable circumstances. Pyæmic abscesses, disintegrating pneumonia, fetid bronchitis, bronchiectasis, gangrene of the lung, ulcerated endocarditis, infective ulcers of the intestines, and similar low-grade processes in the abdominal organs are capable of giving rise to abscess of the brain. Periostitis, involving bones other than those of the head, is among the most infrequent causes of pus formation in the brain.

In some cases of cerebral abscess no appreciable cause has been detected, but the number included under this group is lessening as the etiology of the disease is becoming more thoroughly investigated, especially in regard to latent disease of the ear and the presence of septic material in the mouth and nose.

Pathological Anatomy.—Abscess of the brain is always more or less circumscribed in its appearance, and varies according to whether

[1] *Transactions of the College of Physicians of Philadelphia*, 3d series, vol. vi.
[2] Gowers: *Loc. cit.*, p. 478.

PLATE V.

Abscess of the Brain.

it is studied in its formative, crude, or in its chronic stage; somewhat in regard to the virulence of the poison that has given rise to it; and slightly with reference to its situation, whether deep or superficial. In the formative stage the macroscopic appearance may be very similar to red or yellow softening, depending upon the degree of congestion immediately preceding the pus formation and the amount of hemorrhagic extravasation that has taken place. Usually the abscess in this stage presents the appearance of an area of yellow softening with irregular edges, surrounded by a zone of congested brain substance, but differs microscopically from pure yellow softening in containing numerous pus cells. At a later stage, yet before a capsule has formed, the abscess in a rapidly fatal case is an irregular-shaped cavity with eroded edges, filled with pus of a green or a darkish-green tint. In the most violent cases a slough, consisting of broken-down brain substance, may be found. As a rule, the more profound the symptoms attending upon the formation of an abscess of the brain the greater the area of congestion surrounding it. In some cases the contents of the abscess present a chocolate appearance from the admixture of pus, brain detritus, and extravasated blood. In an encapsulated abscess the cavity tends to become more regular and assumes a spherical or ovoidal shape, and the pus is thickest and most tenacious near the capsule. The latter, if well formed, has a smooth internal surface, a fibrous wall composed of one or more layers, and a roughened, irregular, external surface. Fig. B, Plate V., represents an old, chronic abscess in the centrum ovale of the right occipital lobe. The brain was frozen at the time of the dissection, and W. C. Bane has beautifully delineated the exact appearance of the abscess *in situ*.

The brain tissue surrounding a chronic abscess is œdematous, and often anæmic and softened. The capsule is usually closed, but it may be perforated by a fistulous channel which connects its cavity with a fractured bone, the ear, nose, or even with the lateral ventricle. Suppurative meningitis or sinus thrombosis or phlebitis, more commonly of the superior petrosal or lateral sinus, may occur in connection with abscess of the brain. The microscope shows the contents of the abscess to be made up of granular pus cells, often mixed with disintegrated nerve fibres and leucocytes. Culture tests show presence of various pathogenic micro-organisms, commonly the streptococcus pyogenes and the staphylococcus pyogenes aureus.[1] The contents of the abscess are usually slightly acid in reaction and are fetid in about one fifth of the cases (Gowers). The pus in an encapsulated abscess has a greenish tint as a rule.[2]

The capsule deserves more than a passing notice, as by its presence the length of time that has elapsed since the pus formation may be approximated. Lallemand states that a delicate, thin membrane may be detected in some cases by the end of the second week, but that it is rarely distinct before the end of the third week. Such a capsule is very thin, presents a mucoid lining, and is very easily overlooked. A firm and distinct capsule, composed of connective tissue, is rarely found before

[1] Macewen: *Pyogenic Infective Diseases of the Brain and Cord.*

[2] An exception to the rule was observed by me recently, and reported in *The Medical News*, July 27, 1895, Case II. of the series.

the end of the seventh or tenth week. Gowers says that a capsule is found in only about one half of the cases of abscess of the brain in those that have existed long enough for one to form. If this be so, the absence of a capsule would be of no value in determining the age of the abscess, and the presence of a well-formed one would only indicate that the abscess had existed for at least seven weeks or thereabouts. With one exception all the chronic brain abscesses that I have seen were encapsulated.

The size and number of brain abscesses vary considerably. The usual variation in size is from half an inch to two inches in diameter, but in exceptional cases the abscess may be large enough to occupy one half, or even two thirds, of one cerebral hemisphere. In some instances the abscess is only one or two centimetres in diameter. When multiple the abscesses are usually small. About 80 per cent. of brain abscesses from all causes are single, but the number varies greatly according to the cause. In 93 per cent. of traumatic abscesses and in 87 per cent. of those from ear disease, according to Gowers, the abscess is single, but when the suppuration is due to distant pus formation and to general pyæmic processes, more than one abscess occurs in one half to two thirds of the cases, and in some instances, especially where the brain infection is due to general pyæmia, the number of suppurative foci in the brain may be quite numerous—fifty, or in one recorded case sixty-eight, were counted (Gowers).

Situation.—The white substance of the brain is more subject to suppuration than the gray, and the process occurs from all causes four times more frequently in the cerebrum than in the cerebellum. It rarely takes place in the great ganglia, pons, or medulla. When the abscess is due to ear disease it is found in the cerebrum about twice as frequently as in the cerebellum, and in both cases it is almost always situated on that side of the brain which corresponds with the affected ear. The cerebrum may be the seat of the suppuration from any cause, but the cerebellum is rarely involved except when the cause is ear disease, distant suppuration, and traumatism. Abscess of the cerebellum usually occurs in the hemisphere, and the central lobe is seldom directly affected. Both sides of the brain may be affected in general septic cases, but rarely when the cause is local. Macewen believes the right side of the brain is more frequently the seat of abscess from ear disease than the left, and thinks the increased liability of the right side to suppuration is due to the fact that the right sigmoid sinus is larger than the left, and consequently encroaches more upon the temporal bone. Gowers states that both hemispheres are affected with equal frequency from disease of the ears. When the suppuration is due to other local causes than ear trouble there seems a remarkable predilection for the right side of the brain to be attacked. From all causes suppuration occurs 87 times in the right cerebrum to 70 times in the left; from traumatism, 22 times on the right side to 15 on the left; from disease of the nose, right 7, left 1; from caries of other bones than the temporal, right 4, left 1; from disease of the orbit, right 3, left 0 (Gowers). The abscess is usually situated in the temporo-sphenoidal lobe or in one hemisphere of the cerebellum when the cause is disease of the ear. It is stated, and pretty generally accepted, that disease of the tympanum causes suppura-

tion in the cerebrum (usually the temporo-sphenoidal lobe), and septic material from the mastoid cells leads to abscess of the cerebellum. Toynbee was the first to make this statement, but Macewen calls attention to the fact that when the tympanum is diseased the mastoid cells and antrum are inflamed.

The situation of a local cause, as a rule, determines the seat of the abscess, which is commonly in that part of the brain nearest the source of infection. When a blow to the head is followed by intracranial suppuration the abscess may be deep or superficial, and is usually situated near the seat of the injury, but in some cases it may be found on the opposite side of the brain. Gowers mentions an instance in which a blow to the head was followed by an abscess in the corresponding frontal lobe and another in the cerebellum. An abscess that occurs from disease of the nose or orbit is usually situated in the frontal lobe, rarely in the parietal. The frontal and temporo-sphenoidal lobes are the parts of the cerebrum commonly involved in suppurative processes. For some reason the occipital lobes are not infrequently the seat of suppuration when the cause is a distant one. In children under ten years of age an abscess from ear disease is usually situated in the cerebellum.[1] Gowers states that when the cause of suppuration is unknown the abscess will generally be in the frontal lobe, and believes that this is due to the fact that obscure and unsuspected disease of the nose is the origin of septic material which often finds its way into the brain.

Intracranial pressure is increased from abscess, but not to as great a degree as usually results from tumor; and optic neuritis, which is not an infrequent symptom of the former, is less common and attended by less swelling than occurs from the latter. The abscess exerts pressure on the adjacent brain substance, so that the convolutions over it may be flattened and neighboring tracts of nerve fibres may be irritated or rendered functionless.

Several contiguous abscesses may coalesce and form one large one, or a small abscess may be found near a larger one. Whether the former is the result of the influence of the latter is questionable. During the formation of the capsule the pus may extend into the surrounding brain substance, and the subject may succumb to the depressing influence thus exerted upon cerebral function. If the cerebral substance which surrounds an encapsulated abscess is less firm and resistant in one direction than in another, the abscess may burst and the pus find its way into the ventricles or on to the surface of the brain. Hemorrhage into an abscess cavity is one of the rarest complications of cerebral abscess.

When intracranial suppuration takes place from sources of infection distant from the brain the vascular channels convey the micro-organisms to the brain or its membranes. When the cerebral trouble is caused by infecting material in the parts adjacent to the brain, the infection involves the brain, its membranes, or sinuses, either by the direct extension of the inflammatory process or by the organismal matter gaining access to the cranial cavity, often without any discoverable tract of inflammation leading from the extracranial source of infection to the sup-

[1] Dana: *Diseases of the Nervous System.*

purating process within the cranium. In the latter event the vascular system is the principal means by which pathogenic micro-organisms find a lodgement within the brain. A thrombus may form in the veins of the affected parts outside of the brain and extend into the meninges or brain substance, or fragments of a disintegrating thrombus which contain infective micro-organisms may be carried into the brain when the current in the veins is reversed by being blocked up. Such a reversal of the normal blood current in these veins and sinuses may readily take place, as they contain no valves. When a cerebral sinus becomes affected and blocked, the current is reversed in the cerebral veins that normally empty into the occluded sinus, and hence the blood which has been in contact with an infected sinus and the thrombus is carried into the substance of the brain, where it may set up a suppurative process. Numerous veins empty into the sigmoid sinus from the mastoid cells and antrum on the one side, and from the cerebellum on the other; the superior petrosal sinus receives a number of small veins from the tympanic cavity and tegmen tympani on the one hand, and from the temporo-sphenoidal lobe on the other.[1] As the cerebellum and temporo-sphenoidal lobes are the usual seats of abscess from ear disease, it is probable that the veins that empty into the sigmoid and superior petrosal sinuses are the principal channels through which septic material finds its way into the brain from the bones of the ear. The perivascular sheaths of the arteries may, and sometimes do, convey infective material into the brain substance. On this point Gowers says: "It is not improbable, however, that the perivascular lymphatic canals are the paths by which the infection generally occurs." If the walls or contents of the arteries become affected, arterial thrombus may form, and the thrombus may be carried into the white substance of the brain and cause the formation of an abscess, and the convolution over it may show no trace of inflammation (Macewen). Sometimes a large arterial trunk becomes inflamed, and a complete or partial infective thrombus may form in the vessel, leading to abscess in distant portions of the brain. Macewen saw a specimen of extensive erosion from the middle ear into the carotid canal, the artery being surrounded by granulation tissue, its coats thickened, especially over one side, and its lumen lessened by a soft thrombus. He refers to a case reported conjointly by Gairdner and Barr in which there was complete thrombotic occlusion of the internal carotid, resulting from intratympanic infective inflammation.[2] Extension of inflammation along the lymphatic vessels of the brain is given by Macewen as a means by which suppuration of the brain may result from disease outside of the cranial cavity. The perineural sheaths may be the channels by which micro-organisms find their way through the cranial bones, but Macewen thinks that meningitis is more likely than abscess to result from such a mode of infection.

Symptoms.—There is probably no other gross organic lesion of the brain the symptoms of which are so variable as those of abscess. This is due to several factors. The symptoms of intracerebral suppuration are often complicated, obscured, or completely overshadowed by the symptoms of the causes which have led to the abscess formation in the brain, as those of traumatism, local or distant suppuration, infective

[1] Macewen: *Loc. cit.*

[2] Macewen: *Loc. cit.*, p. 52.

processes in the lungs, heart, or other organs, or general pyæmic conditions. Regions of the brain whose functions are not definitely known, as the frontal and temporo-sphenoidal lobes and the hemispheres of the cerebellum, are most frequently the seats of abscess, and may be unattended by any direct localizing symptoms. In some cases of abscess of the brain the initial symptoms are those of violent irritation, and the course is tumultuous and rapid; while in others, owing to the gradual manner in which the brain has been infected and the mild form of the resulting intracerebral irritation and reaction, the early symptoms are unobtrusive, the abscess becomes encapsulated, and, if situated in a latent portion of the brain, may give rise to no symptoms, or if to any they may be so indefinite as entirely to escape recognition by the most careful observer. Finally, meningitis, thrombosis of a cerebral sinus, or septic phlebitis may precede, accompany, or follow suppurative processes in the brain substance, and, as these are attended by temperature and pulse records so different from those of brain abscess, the medley and confusion of symptoms under such circumstances become quite perplexing to the most clever and careful diagnostician.

The division of abscess of the brain into acute and chronic is of much less importance for clinical description than for diagnostic purposes. Most, if not all, cases of suppuration of the brain begin acutely, but the early symptoms of some are so insidious that they are overlooked, or, if they attract attention at the time, their apparent insignificance prevents them from being considered of much importance. In other cases the initial symptoms of intracerebral inflammation are quite distinct, but in the course of days or weeks these subside and are replaced by those of moderate intensity, until finally the patient is considered fairly convalescent; but the physical state of the patient indicates considerable depression, and chronic head discomfort, irritability, disturbed sleep, and the sense of mental and physical fatigue point to cerebral mischief.

Acute Abscess of the Brain.—The symptoms of acute abscess of the brain may be divided into three classes: the constitutional, those that occur from general cerebral disturbance, and the localizing. The last results from all forms of brain abscess when regions of the brain whose functions are known are affected by the morbid process, and will receive attention after the constitutional and general symptoms of acute and chronic abscess have been described. The symptoms will vary in accordance with the activity of the morbid process. When this is active and decided irritation and inflammation result, the brain symptoms will be pronounced and of an irritative character; when the abscess has formed and becomes more or less circumscribed, the functions of the brain will be obtunded, and the symptoms will resemble those of inflammation of the brain, to which the element of compression has been added. Dividing the symptoms into three stages—the initial, those of the fully-formed abscess, and the terminal stage when uninterrupted by treatment—adopted by Macewen, has certain advantages.

The Initiatory Stage.—The most acute forms of cerebral abscess are those of traumatic or pyæmic origin. The initial symptoms are often unrecognized when the cause is pyæmia because of the presence of the general symptoms of the blood state, and the rapidity with which cere-

bral suppuration takes place under such circumstances, so that the brain complication is not usually suspected until the second stage is reached.

The following account of the early brain symptoms after traumatism to the head is based largely upon the results of observations of 7 cases which were under my care a short time ago. In 3, after prolonged and rather serious symptoms of brain disturbance, complete recovery took place without suppuration; in 2 acute cerebral abscesses formed, death resulting at the end of the third and fourth week respectively; and in the remaining 2 the patients were not under my supervision in the early part of their illness, but in each occurred a cerebral abscess which became encapsulated and ran a chronic course. Following injuries to the head, which may suppurate or heal readily, especially those attended by fracture of the cranial bones, the patient may feel no discomfort except from the scalp wound for several days, when localized headache, usually situated in the region near the scalp wound, rarely on the opposite side of the head, is experienced. Except in the very acute cases the pain at first is not severe, but it has two characteristics which make it suggestive of cerebral irritation—constancy and localization. The pain is worse at certain times of the day than at others, when it is scarcely complained of at all, but at all times the patient feels a slight discomfort, usually deep in the head. He is constantly more or less conscious of his head. The pain may radiate to other portions of the head, but the site of the original pain is rarely free from discomfort. At the same time, the patient becomes irritable and restless, sleep is disturbed, and slight delirium is not infrequent; the tongue is coated, the appetite is lessened or lost; the bowels are constipated and nutrition suffers. The temperature is usually elevated from a half to one degree in the evening, and not infrequently is highest during a paroxysm of pain. The pulse varies from 80 to 100, and is small and irritable. Chilly sensations may be complained of, but distinct chills are rarely experienced until inflammation of the brain has become evident. The condition may have one of several terminations. In the severest cases all the symptoms may rapidly increase; the headache becomes more intense, a convulsion occurs, localizing symptoms may or may not develop, the patient presents an apathetic condition, and finally passes into a comatose state, and the disease runs the course of suppurative meningitis, with which it is often complicated. In less acute cases the morbid process shows a gradual increase, as is manifested by the slight augmentation of the headache and all the other symptoms which indicate a slow but grave cerebral disturbance. The patient becomes more irritable, and, if he is not confined to his bed, he shows a disinclination to mental or physical exertion. The digestive tract is inactive; sometimes the stomach is irritable and the bowels fail to act, except in response to a rather strong cathartic. Flesh is steadily lost, prostration grows more pronounced, the pulse is increased in frequency at first, but soon becomes quite slow, and the temperature is a little above normal. Usually a chilly sensation or a distinct chill indicates that suppuration has taken place. The abscess forms, and may run a subacute or chronic course. In the mildest class of cases, with evidence of cerebral irritation follow-

ing a blow to the head, after the disturbances have lasted several days or weeks all the symptoms begin to abate; the appetite increases, the headache lessens, nutrition shows improvement, and the patient makes a complete but rather tedious recovery.

Brain abscesses from ear disease are rarely as acute as those that have their origin in traumatism or pyæmic processes. The inflammation of the middle ear has usually existed for a considerable time. Even in the adult it may have been present since early childhood, and started with one of the exanthemata, from which the patient suffered. The otorrhœa may have been intermittent, or there may have been no perforation of the tympanic membrane, no discharge from the external auditory meatus, and the occasional attacks of pain over the mastoid process, especially following exposure to cold, are the only subjective evidences of chronic ear trouble. It is unusual for cerebral abscess to result from the primary attack of acute inflammation of the middle ear, no matter how severe the inflammatory process may be. In persons who are suffering from chronic otitis media exposure to cold or a slight blow over the ear is the usual exciting cause that determines a fresh attack of inflammation in and about the ear. The pathological changes which have resulted from the chronic inflammation of months, or more commonly years, in structures composing the tympanum, mastoid antrum and cells, tegmen tympani, and the vascular connections between these parts and the brain and its membranes, have been such that infective material more readily involves the brain than in the primary attack of otitis media, when the parts adjacent to the focus of inflammation were in a comparatively normal condition. The first symptoms are those of a subacute attack of otitis media, and these are so similar to the early symptoms of cerebral suppuration that it is not always possible to distinguish the one from the other. The symptoms that are common to both the early stages of a severe attack of otitis media and brain suppuration resulting from the inflammatory ear trouble are—pain in the ear, often severe and radiating to other portions of the head, usually on the side corresponding with the affected ear; elevation of temperature and increased rapidity of pulse; coated tongue, nausea, and vomiting; vertigo and rigors. The symptoms point to brain mischief when severe and deep-seated pain becomes localized in the temporal, occipital, and frontal regions, is remittent or occasionally intermittent, and is attended by paroxysms of great suffering, during which the patient may turn the head from side to side or try to bury it in the pillow; when the vomiting is disconnected with the ingestion of food, unassociated with nausea, but attended with almost complete anorexia; when the rigor is prolonged and associated with distinct pallor of the skin and lips and an appearance of the skin known as cutis anserina; when the temperature is only slightly above normal and the pulse slow, and the patient's condition is such as to indicate great prostration. If the discharge from the ear has been free before, but now becomes greatly lessened or ceases entirely, the evidence in favor of intracranial suppuration is very strong, and if now a convulsion occurs, general or partial, all doubt of such a condition may be dismissed, as a rule. The duration of the initiatory stage of abscess of the brain, during which symptoms of intracerebral inflammation and irritation are the prominent ones, varies from

a few hours in the most acute and violent cases to several days, or even a week or more, in the less acute ones.[1]

Stage of Fully-formed Abscess.—If to the symptoms of the first stage we have added in the second those of increased intracerebral pressure, by which, with the other results of the encephalitis, all the patient's senses are obtunded, there is no longer any doubt of organic brain lesion. The pain, which was severe and one of the most prominent symptoms in the early stage, is now less acute, and sometimes is not complained of unless the patient's attention is directed to it. The probable factors which lead to this result are lessened violence of the inflammatory process and the blunted sensibility which has resulted from the encephalitis and increased intracerebral pressure. Macewen thinks that lessened pressure is one of the causes of decreased pain in this stage. At times the patient still experiences severe paroxysms of pain in the ear, temporal, frontal, or occipital region, but it has lost its acuteness. That there is yet more or less constant discomfort felt is evidenced by the patient keeping one hand over the affected ear or one or both hands grasping the head over the occipital region, and by the occasional moans heard even while he is dozing or appears to be asleep. The patient becomes apathetic, and slow cerebration is well marked. In the early stage of the disease there was considerable irritability and the patient showed apprehension concerning his condition, but now he seems more or less indifferent, and often dozes while the physician is conducting an examination. If he is asked a question, the answer is delayed, but usually it is correct when made. Sometimes the patient stares at his questioner, and as a question is about to be repeated lest he should not have understood what was said to him, he begins to reply. Prolonged examinations or numerous questions exhaust and sometimes irritate the patient. He finds it difficult to keep his attention fixed on any subject. Motor weakness, in the absence of paralysis, is less than it seems if judged by the amount of muscular effort the patient is able to put forth, as in grasping the dynamometer. This is on account of the weakness of the power of the will. The temperature and pulse show a marked change in this stage. The former, in the absence of such complications as meningitis and phlebitis, is normal or subnormal, while the latter is slow and often weak. Some cases of fully-developed abscess are not attended with a slow pulse, but usually in such, if the abscess is large, meningitis or a septic inflammation of one of the sinuses will be found. The pulse may be 60, 50, or much less frequent. In a case of abscess of the cerebellum reported by Murray the pulse was on one occasion 20 per minute.[2] Respiration may show but little change from normal, except when the contents of the posterior cerebral fossa are involved by the suppuration, when it may be slow (10 or 12 a minute) and distinctly intermittent or Cheyne-Stokes in character. Optic neuritis, if I may judge from my own observations, is much more frequent in abscess of the brain than the writings of most authors would lead one to believe. The edges of the optic nerves may often be found to be indistinct and

[1] With the above symptoms, slow pulse and nearly normal temperature indicate abscess; pulse of 100 or thereabouts, and temperature varying from 101° to 102° F., point toward meningitis; while great temperature variation, especially with repeated chills, should suggest sinus phlebitis.

[2] *British Medical Journal*, Jan. 5, 1895.

the retina presenting a hazy, velvety appearance during the latter part of the first week of the second stage, but distinct papillitis with swollen disk is not pronounced until a week or more later. If the eyes are carefully examined every day, the gradual changes that may be observed are of great diagnostic import. The swelling rarely becomes very great, not more than $\frac{1}{80}$ to $\frac{1}{40}$ of an inch, requiring +1 D. to +2 D. to see the disk distinctly, while in tumor +4 D. to +5 D. are not infrequently necessary. The appetite is usually lost, the tongue coated, sometimes dry and brownish, but vomiting is not frequent, except in cerebellar abscess, unless the patient tries to sit or stand. Constipation is the rule, and sometimes it becomes obstinate. The patient emaciates rapidly and shows evidence of great prostration. The urine is retained, and has to be drawn by the catheter. According to Macewen, it often contains albumin. Unless septic phlebitis or meningitis is present, rigors are uncommon during the second stage. General convulsions sometimes occur, but they are rare in this stage. Localized convulsions may occur when the so-called motor areas or fibres from them are involved. Paralysis, paresis, or rigidity, if present, depends upon the situation of the abscess in or near the motor cortical areas or the fibres that conduct impulses from them to the spinal centres.

Anæsthesia is a rare symptom of abscess of the brain, and is present commonly when the fibres in the posterior third of the hinder part of the internal capsule are destroyed or pressed upon or when the centrum ovale at the junction of the temporo-sphenoidal, occipital, and parietal lobes is the seat of suppuration. The latter has happened recently in two cases of abscess of the occipital lobe coming under my observation.[1] Direct cranial nerve involvement is exceedingly infrequent, but indirectly, through pressure, it has been recorded in a number of cases, and in some cases it has been so marked as to cause it to be mistaken for tubercular meningitis.

The face is usually expressionless, shows evidence of great prostration, and sometimes presents a dusky hue, but a similar condition is observed in many acute diseases attended with emaciation and pronounced prostration. The odor of a patient suffering with acute cerebral suppuration from otitis media is often most striking and significant. It is very offensive, and is similar to the odor given off from a foul-smelling discharge from the ear. But it does not depend upon such a condition of the ear, for when the ear is kept scrupulously clean the same offensive odor is encountered on approaching the patient.

Percussion over the site of the abscess often gives the patient discomfort and causes him to wince. Sometimes the whole of the affected side of the head is more sensitive than the opposite side. Macewen claims to be able to detect a higher pitch of the percussion note over the abscess than over other portions of the brain. I have studied the pitch of the percussion note in two cases of abscess and in three of tumor, but have not been able to satisfy myself that the change is sufficiently great to be of much diagnostic value.

If the abscess is complicated with meningitis of the posterior fossa, the head will be retracted, and when disease of the sigmoid sinus accom-

[1] Eskridge: "Diagnosis of Chronic Abscess of the Brain," *Amer. Journ. of Med. Sciences*, Aug. and Sept., 1896.

panies an abscess in the temporo-sphenoidal lobe or cerebellum, the sterno-mastoid muscle of the same side is rigid and the tissues around it are painful, and other symptoms of sigmoid-sinus affection may be found, such as pain and tenderness along the course of the internal jugular vein and in the apex of the posterior cervical triangle. Macewen calls attention to the importance of aural examinations in abscess of the brain, as erosions of the tegmen tympani, and sometimes pus oozing through this bone, may be discovered. The latter is always positive evidence of suppuration within the cranial cavity.

Terminal Stage.—An acute abscess, uninterrupted by surgical measures, in which the symptoms do not sufficiently abate for it to become chronic, usually proves fatal, without a period of latency, within a few days to three or four weeks, death taking place from extensive softening of the brain tissues and exhaustion, or from bursting of the abscess into the ventricles or on to the surface of the brain, and giving rise in the former instance to symptoms similar to those from a hemorrhage into the ventricles, and in the latter to those of a suppurative meningitis.

Chronic Abscess.—Under this designation may be included all cases of abscess of the brain that have reached the so-called latent stage, whether the initial symptoms have been pronounced or so insidious as to have attracted little or no attention. Prof. Augusto Murri of Bologna in an exceptionally able address has so graphically pointed out the necessity of a closer description and more accurate knowledge of chronic abscess of the brain than are found in most text-books that I feel free to quote from his essay:[1]

"Abscess of the brain, which when clinically considered seems to be an essentially unique morbid process, resolves itself into two varieties, which differ utterly in diagnosis—viz. acute abscess and chronic abscess. Writers aiming at the unity of the process lose sight of the clinical side, and do not bring into sufficient relief the practical difference which exists between the two varieties. What they say of the abscess applies chiefly to the acute form.

"When we come to the chronic form of cerebral abscess, the descriptions and opinions of pathologists, inspired by the observations of such facts,[2] no longer correspond to the facts themselves. In these the exciting causes are less intense or less apparent. It is one thing, for example, if an embolus from a pulmonary vein closes the artery of Sylvius, and another if pus penetrates unperceived into the brain by one of those narrow ways which lead there from without. The slightness of the latter is not only marked by the absence of disorders perceptible to the medical man and patient, but it also causes a slower succession of changes around itself; the surrounding tissues, not previously affected and less violently attacked, have time to organize themselves for the defence, and in time circumscribe with a solid barrier (pseudo-membrane) the hostile element (pus) which tends to destroy it. This mysterious faculty which the brain possesses of adapting itself to the most serious

[1] An "Address on Experimental Craniotomy and Diagnosis of Cerebral Abscess," *Lancet*, Jan. 5, 12, 26, and Feb. 2, 1895, pp. 9, 79, 206, and 267.

[2] He here refers to the morbid processes and rapid changes taking place in acute abscess.

lesions as long as they are gradual has full opportunity to act completely. Thus it follows that from ignoring or forgetting the primary cause it seems as though no external cause exists; and here we have the idiopathic abscess, which is almost always chronic. Even when the primary cause is known, if it be not violent the process which succeeds follows with less rapidity, and hence the slight changes of the brain may not arouse perceptible functional disorders. Here we have the latent abscess or the latent period of the chronic abscess. The slowness with which brain alterations proceed proves that a length of time is required for their development, and here, we see, the abscess becomes chronic. The perception of these internal processes is often deceptive, as is frequently seen in the doctrinal works of pathologists and in the reasoning of clinical physicians, who write and think of cerebral abscess as though it existed only in the acute form. But the abscess exists also in the chronic, idiopathic, latent, or semilatent form, and this, though less frequent than the acute, is by no means as rare as statistics would make us believe."

Latent Period.—The latent period of chronic cerebral abscess may not be attended by any symptoms that lead us to suspect organic disease of the brain. In such cases, which are probably comparatively rare, one is not called upon to make a diagnosis until symptoms of the terminal period manifest themselves. In all probability, the large proportion of the chronic forms of brain abscess, even in the latent period, present symptoms, more or less definite, of organic brain changes, but usually such symptoms as are common to several forms of brain disease, and a correct interpretation of them will depend upon the experience, diagnostic skill, and thoroughness of the physician. I cannot emphasize too strongly the precaution urged by Murri, that the physician should always bear in mind the possibility of the presence of a chronic cerebral abscess in cases of obscure brain disease. The frequency of this form of cerebral abscess is not great, but the presence of chronic cerebral suppuration is too often overlooked for the credit of medical science or the welfare of the unfortunate sufferer.

A careful study of a detailed chronological history is one of the best safeguards against error in diagnosis. Conclusions arrived at by an analysis of the most accurate history may, in some cases, lead from the truth, but in the vast majority of instances information thus obtained, together with a thorough examination of the mental and physical condition of the patient, is helpful in reaching a diagnosis. In a goodly number the patient will give an account of some condition which is known to be favorable to abscess formation in the brain. The cause may have been active months, and in rare instances years, before, and may, at the time of the examination, be forgotten by the patient, and is only revived by a careful and intelligent line of questioning. It may have been a blow on the head, a fall from a height and alighting on the feet or buttocks, a painful condition of one ear with or without otorrhœa, suppuration in some portion of the body,[1] or some exhausting disease, possibly influenza, diphtheria, rheumatism, or typhoid fever, from which recovery has never seemed complete. In typical cases the

[1] This more frequently results in acute abscess than in chronic, except where the pus is in the pleural cavity or possibly in the lungs in some instances.

history will reveal the fact that headache, intermittent or constant, has been a prominent feature for a greater or less part of the time that has intervened since the occurrence of the illness that left the patient's health impaired. In some cases irregular febrile phenomena, dimness of vision, vertigo, loss of appetite, gastric irritability, failing nutrition and strength, and possibly monospasm, monoparesis, or general convulsions, have been experienced. In the examinations of cases of chronic abscess one would expect to find abundant evidence of organic brain disease, but in this one is often disappointed.

The most careful examination of the motor apparatus and of the phenomena of general sensation may yield negative results. The reflexes may be entirely normal or slightly exaggerated. If the motor cortical areas or motor tracts of the brain are affected, either directly or indirectly, by the abscess, symptoms of paresis, paralysis, or muscular rigidity will be present, corresponding to the extent and situation of the abscess and the softening resulting from it. Nutrition and muscular strength usually fail in the later stages, but in some cases these are unimpaired. Mental failure, especially slow action of the mental faculties and lessened power of memory, together with irritability of temper, are not infrequent. Recurring convulsions, sometimes mistaken for those of idiopathic epilepsy, are found occasionally in the latent period.

Among the special symptoms of the latent period headache is the most important. Its significance is that its beginning dates from exposure to certain conditions that are known to be capable of giving rise to abscess of the brain. It is rarely constant, but there are a few cases in which headache has not been present during a portion of this period. At times the headache is severe, and at other times it amounts simply to a dull or uncomfortable sensation. When the terminal period, caused by softening, œdema, or extension of the abscess, is reached, the headache becomes very prominent, and its severity marks the beginning of the end. The location of the pain does not always correspond to the seat of the abscess. The typical temperature of this stage, if the abscess exerts sufficient influence to cause much depression, is a half to one degree F. below normal. In a case of chronic abscess of the brain reported by me to the College of Physicians of Philadelphia[1] the temperature was normal or subnormal throughout most of the course of the disease, and was lowest on the unaffected side of the body. In some cases there occur irregular febrile processes which are frequently attended by cool and clammy perspiration. Optic neuritis, when present, is a symptom of great importance. Some observers seem inclined to think that it is of infrequent occurrence, while others have observed it sufficiently often to regard it as a valuable aid in the diagnosis. In the cases that I have observed it has been present more frequently than absent.

Terminal Period.—This period may extend over several weeks or a few days, and possibly in some cases may last only a few hours. Its duration depends upon the manner in which the abscess causes death. The latent period may end suddenly by the abscess bursting into the lateral ventricles and giving rise to all the symptoms of intraventricular hemorrhage. The rupture may occur on the surface of the brain in

[1] *Transactions of the College of Physicians*, 3d Series, vol. vi., 1883.

the posterior fossa, and cause local meningitis and irritation of the pons and medulla, and thus directly interfere with the cardiac and respiratory centres. If the patient survives the shock, the symptoms will be those of cerebro-spinal leptomeningitis, prominent among which are retraction of the head, rigidity of the muscles of the back of the neck, opisthotonos, and shock-like convulsive rigidity of the muscles of all the limbs, especially of the extensors of the arms. If the pus finds its way on to any portion of the surface of the brain, leptomeningitis, which proves rapidly fatal, usually results. The symptoms correspond to the seat of the irritant and the extent of the inflammation. Œdema and softening are the cause of death in about one-half the cases of chronic abscess of the brain. In many of these cases, in which the motor or sensory areas of the brain are not especially affected, either directly or indirectly, by the abscess, the softening may take place so gradually that no symptoms of it may be manifest until sudden collapse or death results from the apparent arrest of cerebral function. It is probable that if these cases which end suddenly from softening and œdema were carefully watched, symptoms of failing brain power and general vigor, together with increased headache, irritability of temper, and a tendency to depression, might be detected weeks before the fatal end. In all the cases, except one, of chronic abscess of the brain which have come under my observation during the terminal period, the symptoms of this stage have developed in a subacute manner. The prominent symptoms have been headache, usually dull and heavy in character, but at times attended with severe paroxysms of pain; failure of nutrition, strength, and energy; mental dulness and irritability of temper; vomiting, especially associated with paroxysms of pain in the head; optic neuritis in a number of instances; slow or normal pulse when the patient is quiet, but easily accelerated by exercise; subnormal temperature in nearly every case; convulsions occasionally at the beginning or end of the terminal period; paralysis, usually hemiplegic in character, or paresis with rigidity of the affected muscles, resulting from pressure in more than half the cases, and hemianæsthesia in three cases. As the terminal period progresses the symptoms increase in gravity, the sensory functions become more and more blunted, the patient becomes apathetic, and finally stupor, followed by coma, takes place, and the patient dies after remaining unconscious several hours to several days. It is rare, after symptoms of the terminal stage become prominent, for the patient to show more than transient improvement.

Localizing Symptoms.—The most usual seats of abscess of the brain are in those portions, the cerebellum, frontal and temporo-sphenoidal lobes, the functions of which are not definitely known, and the symptoms of abscess are less distinctive of its location than in the case of tumor. Localizing symptoms are as frequently absent as present in abscess of the brain.

Frontal Lobe.—The peculiar mental alteration observed in the case of tumor of the frontal lobe is rarely so characteristic of abscess in this region, because abscess of the brain, no matter where located, is attended with lack of mental vigor and the patient becomes apathetic. A large abscess may exist unsuspected in the anterior portion of the frontal lobe,

and a small one may be situated just anterior to the motor region, without giving rise to symptoms indicative of its seat. It is only when softening or œdema extends backward to the motor area of the cortex or to the white substance beneath it, or pressure is exerted on this portion of the brain, that localizing symptoms develop, except in a few cases in which pupillary symptoms have been found when the abscess occupied the posterior and lower portion of the frontal lobe. If general symptoms of abscess of the brain should follow a blow on the front of the head, develop during a septic condition in the lungs, or should be associated with an infective frontal sinus, a gradual increasing paresis or paralysis of the face or arm on one side should occur, preceded or not by convulsive movements of the affected muscles, it would point to the frontal lobe as the seat of the abscess. If the pupil on the side opposite to the one on which the muscles are involved should become sluggish or inactive, while its fellow responded normally to light and accommodation, it would aid in locating the abscess in the posterior portion of the frontal lobe on the side corresponding to the affected pupil. It must be borne in mind that the same pupillary phenomena may be present when the anterior portion of the temporo-sphenoidal lobe is the seat of the abscess as occurs from a similar lesion in the posterior portion of the frontal lobe, but suppuration in the former usually results from middle-ear disease. Macewen's observations are interesting in this connection, and may be quoted in full: "When the abscess is seated in the temporo-sphenoidal or frontal lobe, the pupil on the same side as the abscess may either become myotic or mydriatic, accompanied by a degree of stability. When the abscess is small and produces cerebral irritation, the pupil on the affected side may be more or less contracted and sluggish. When the abscess is large and exercises considerable pressure, the pupil on the affected side is prone to stabile mydriasis. An abscess which is small and produces a stabile myosis to begin with may, as it augments in bulk, produce a stabile mydriasis in the same eye. Occasionally the only difference detectable is sluggishness of one pupil to both light and accommodation, while its neighbor acts freely. In a case of cerebral abscess a sluggish pupil with either mydriasis or myosis on the same side as an infective injury to the skull or a unilateral otitis media, affords further evidence of the abscess being on the corresponding side of the brain."[1] I have observed four cases of abscess of the frontal lobe, and in none of them have the pupillary phenomena described by Macewen been present, but in none was the abscess situated in the posterior lower portion of this lobe.

Temporo-sphenoidal Lobe.—A small abscess, and even one of considerable size, may be situated in the temporo-sphenoidal lobe without giving rise to any distinct localizing symptoms. Under such circumstances in acute abscess pain, which is usually in the region of the corresponding ear, especially in the beginning, taken in connection with the affected ear, might be the only symptom present of a localizing character, while if it were chronic the history might afford the only aid. A large abscess, on the other hand, would probably develop localizing symptoms from the effects of pressure on the surrounding structures, and if extensive softening should occur regions of the brain whose func-

[1] Macewen: *Pyogenic Infective Diseases of the Brain and Spinal Cord*, p. 145.

tions are fairly well known would become involved. If the abscess were on the left side, in the posterior portion of the temporal region, so as to affect the first and second convolutions, a form of sensory aphasia (word-deafness) would be present. On either side, in the anterior portion of this lobe, a large abscess might cause paresis or paralysis—first, in the face, and secondly, in the arm on the opposite side, with pupillary symptoms on the same side, such as sometimes occur in abscess of the posterior portion of the frontal lobe, and paresis or paralysis of the third cranial nerve on the same side as the abscess. Complete motor aphasia, while it is possible from abscess of the temporo-sphenoidal lobe, practically does not occur. So far as I know, disturbances in smell or taste have not been observed in abscess of the temporo-sphenoidal lobe. A careful study of the direction in which the paralysis extends after the first group of muscles is involved will often afford valuable information upon which to base a localizing diagnosis. Not infrequently the middle-ear disease which leads to abscess of the brain paralyzes the facial nerve as it passes through the Fallopian canal. In such a case the facial paralysis is complete, is on the side of the affected ear, and emotional and voluntary power over the paralyzed muscles is lost. In facial paralysis from cortical lesion the upper portion of the face is less completely paralyzed than the lower, and emotional power over the muscles is retained to a considerable extent.

Occipital Lobe.—In three cases[1] of chronic abscess of the occipital lobe observed by me paresis and muscular rigidity, nearly complete anæsthesia, and hemianopsia of the opposite side existed. In each case the abscess was large and situated in the white substance, so as to damage the fibres in the posterior half of the internal capsule. It is probable that fibres coming from the cortical centre of sight in the cuneus were rendered functionless, either by pressure or softening, as the cortex of the cuneus did not seem to be directly involved. A small abscess might remain in the white substance of the occipital lobe for a long time without giving rise to localizing symptoms.

Cerebellum.—An uncomplicated abscess of considerable size in one lateral lobe of the cerebellum, especially when situated in the posterior portion, so as not to cause pressure symptoms upon the pons, might give rise to no localizing symptoms. When meningitis of the posterior fossa exists with the abscess, there will be retraction of the head and rigidity of the posterior neck muscles. When the abscess is situated farther forward, so as to affect the pons and medulla, especially when complicated with meningitis, pulse and respiration will be slow and feeble. Macewen has called attention to a mechanical opening and shutting of the mouth and clenching of the teeth from rigidity of the masseter muscles in connection with abscess of the cerebellum. The pulse in abscess of the cerebellum is sometimes very slow, and sighing respiration has been frequently observed. The localizing symptoms of abscess of the pons and middle lobe of the cerebellum do not vary much from those of tumor in the same situation, except that abscess is more likely to be complicated by symptoms of meningitis of the posterior fossa.

Diagnosis.—The diagnosis of abscess of the brain is one of the most difficult problems, if indeed it is not the most difficult problem, in clini-

[1] *The Medical News*, July 27, 1895.

cal neurology, especially when the abscess is chronic and attended by a more or less distinctly latent period. A few fortunate diagnoses—which, perchance, were more the result of a combination of happy circumstances rather than the logical conclusions from data—may have misled some able diagnosticians into the error of underestimating the difficulties and uncertainties of the task. The fact remains, however, that the greater number of the most able diagnosticians with the most varied and extended experiences confess their inability to make more than a problematical diagnosis in a large percentage of the cases of chronic abscess of the brain. In the diagnosis of the acute variety, which is usually attended by more obtrusive symptoms than the chronic form, difficulties arise from the complications, such as meningitis and sinus phlebitis, that are so frequently associated with it. The cerebral symptoms may result from other organic brain diseases, such as a growth, meningitis, simple or tubercular, or from cerebritis. Some of the best textbooks on the diseases of the nervous system teach that the symptoms gain significance when associated with conditions suggesting a suppurating process, especially fever and rigors. This is true in a small number of cases, but rigors are most frequent and most pronounced in sinus phlebitis, and a temperature nearly normal or slightly subnormal is more common in abscess of the brain than a distinct febrile process. In a "Report of Three Cases of Abscess of the Brain"[1] I called attention to the prolonged subnormal temperature in chronic abscess of the brain. The observation was made that the temperature was higher on the paralyzed than on the unparalyzed side of the body, extending over a period of more than two months. This observation has been verified in three cases of chronic abscess of the brain recently observed by me. The temperature was from half a degree to a degree below normal, and on one occasion three degrees, on the non-paralyzed side, while it was almost invariably normal or slightly above normal on the paralyzed side. Macewen's observations have led him to believe that acute abscess of the brain, as well as chronic, is attended with a temperature varying but little from the normal. He often found it subnormal, normal, or from a half to one and a half degrees above normal, except in the initiatory stage, when it may be a little more elevated. Slow respiration and pulse seem to be more marked in suppuration of the posterior fossa, especially when pressure is exerted on the pons and medulla, than in other organic diseases of this region. A combination of symptoms of organic diseases of the brain following causes which sometimes give rise to abscess of the brain, such as disease of the ear, traumatism of the head, distant suppuration, especially in dilated bronchial tubes, or a purulent discharge from the nose, should lead one to suspect abscess. When the stage of latency is complete, the symptoms of the terminal stage may be ushered in suddenly, suggestive of a vascular lesion, but more commonly this stage is preceded by several weeks of failing health and variable cerebral symptoms. The presence of optic neuritis would exclude a vascular lesion, but it must be borne in mind that lead-encephalopathy, renal disease, and profound anæmia may give rise to optic neuritis.

In acute abscess, cerebritis, meningitis, and septic phlebitis with

[1] *Transactions of the College of Physicians of Philadelphia*, 3d Series, vol. vi., 1883.

sinus thrombosis have to be considered in the diagnosis, and if the symptoms have followed a blow to the head, the effects of trauma (probably attended by capillary hemorrhage into the brain substance) must be taken into account. The symptoms from the direct effects of traumatism come on immediately after the injury, and it is only when confused consciousness, occasional delirium, headache, loss of appetite, restlessness, insomnia, and impaired strength and nutrition continue for two or more weeks that abscess need be seriously apprehended. Cases of traumatism of the head, followed by the above symptoms for a prolonged period, have in my experience been attended by a temperature varying but little from the normal. Such symptoms when due to the direct effects of the blow are most pronounced soon after the injury, and when no serious brain lesion exists there is a gradual improvement of the symptoms; while, on the other hand, if evidence of increasing brain trouble should become more pronounced at the end of two or three weeks, it would point to abscess. Cerebritis following trauma is manifested by symptoms beginning three or four days after the injury, and it is improbable that it can continue long in association with septic conditions without ending in suppuration. When the inflammation is due to trauma and pathogenic micro-organisms are absent, it may continue for an indefinite time without resulting in abscess. There is little difficulty in distinguishing a disintegrating infective sinus thrombosis or meningitis from abscess when one of these morbid processes exists alone. Meningitis is attended with a continued elevation of temperature considerably above the normal, and usually symptoms of intracranial nerve irritation or retraction of the head. An infective condition of a sinus is marked by repeated chills, followed by high temperature and well-pronounced remissions; the pulse is rapid and weak, and tenderness is often present over the mastoid region and superior posterior triangle of the neck, and over the course of the jugular vein. The difficulty in diagnosis arises from the fact that one or both of these complications may be present in a case of abscess. The symptoms of infective thrombosis are so obtrusive as in a great measure to overshadow those of abscess. The affection of the sinus demands the first attention, as a cure would be impossible, though the contents of the abscess were evacuated, so long as the condition of the sinus is allowed to go unrelieved; and the abscess should receive attention later if it can be diagnosticated. The presence of basilar meningitis in the posterior fossa will be evidenced by retraction of the head, and in the middle fossa by involvement of the cranial nerves.

In the vast majority of instances the long duration of chronic abscess would serve to distinguish it from tubercular meningitis, but occasionally the latter may run a course of several months' duration with symptoms simulating abscess.[1] When the terminal stage begins gradually, the diagnosis will be between tumor and abscess; injury may apparently be the cause of either, and if the traumatism dates back more than a year and was not attended by any evidence of suppuration at the time, the chances would be in favor of tumor rather than abscess. Optic neuritis, while a symptom of either, is more commonly attended with a greater swelling of the disks, and is more frequently followed by com-

[1] See Prof. Murri's Case V., *Lancet*, Jan. 12, 1895.

plete blindness in the former than in the latter. A steady but gradual increase of symptoms, with invasion of a more extensive area of the brain, the development of one nerve-root symptom after another, and prolonged periods during which the disease makes but little progress, are more common in tumor than in abscess. A chronic abscess may be so situated as to cause hemiplegia. In such a case, besides tumor, a vascular lesion would have to be excluded. In the absence of optic neuritis the presence of severe headache or a temperature higher on the paralyzed side several weeks after the hemiplegic symptoms were manifest would point to an irritative lesion in the brain, which is usually not of a vascular nature.[1] In the absence of a history or of optic neuritis, if the symptoms of the terminal stage develop suddenly and give rise to a condition simulating that of a large hemorrhage in the brain, it may be impossible to make a diagnosis.

Prognosis.—A few cases of superficial abscess of the brain have resulted in recovery by the pus finding an exit from the brain by a pathological opening through the membranes and bone, but usually such superficial abscesses prove fatal, unless operative procedure is instituted, from exhaustion resulting from prolonged suppuration. In exceedingly rare instances an abscess becomes encapsulated, its contents partially absorbed and changed, so that it may remain in the brain for a number of years without causing much inconvenience to the patient, but sooner or later œdema and softening of the surrounding brain substance take place, and death is the result. Macewen states that the contents of a very small abscess may be absorbed and recovery result. This must be a very rare termination. Practically, the only relief for a person suffering from cerebral abscess is through surgical means. Of 25 abscesses of the brain, details of which are given by Macewen,[2] 19 were operated upon, with 18 recoveries. This is a marvellous advance in dealing with what was formerly considered one of the most fatal diseases of the brain. Such results can only be obtained by a master in surgery, and the operation must be performed early and guided by a careful and exhaustive study of each individual case. Cases complicated by sinus thrombosis are the least hopeful even when surgical interference is instituted early; those in which meningitis exists give but little better results; while the uncomplicated ones, if the abscess or abscesses are reached, afford a sanguine prognosis. The motor and mental disturbances caused by the presence of an abscess in the brain usually completely pass away in a comparatively short time after the pus has been evacuated and the cavity cleansed.

Treatment.—One of the most important measures in the treatment of cerebral suppuration is in the prevention of infective foci, or in the event that any have formed the removal of these, if possible, before the brain substance has become affected. Wounds of the neck, face, scalp, bones, membranes, and brain should be rendered thoroughly aseptic at the earliest possible period, and kept so. All source of infection should be removed, whether it be in the nose, mouth, throat, or ear. Middle-ear disease, which is too commonly neglected, should receive

[1] This does not hold good in vascular lesions which are followed by an area of softening, and which cause some irritation of the adjacent brain substance.

[2] *Pyogenic Infective Diseases of the Brain and Spinal Cord.*

especial attention. In speaking of infective material in the mastoid antrum and cells Macewen tersely and forcibly says: "A person might as well have a charge of dynamite in the mastoid antrum and cells, as one cannot know the moment when accidental circumstances may arise which may cause the infective matter to become widely disseminated all over the cerebro-spinal system." If sinus phlebitis or thrombosis or local meningitis develops, such a menace to the integrity of the brain should be removed by operative procedure as early as possible. During the initiatory stage of abscess formation in the brain the measures should consist of those most likely to lessen suppuration, but as soon as the evidences of pus in the brain are apparent and its location reasonably certain, an operation for its removal should not be delayed. The symptoms of the initiatory stage are best combated by attention to the kidneys, bowels, and stomach, by the application of an ice poultice[1] to the head, by counter-irritation applied to the parts just below the occiput and over the mastoid bones, by relief of pain, and by the administration of nutritious, digestible, but not too stimulating food.

ATROPHY OF THE BRAIN.

ATROPHY of the brain may occur in utero or be acquired after birth. Children born with arrested cerebral development usually have small crania. Some have contended that the small cerebral mass is due to early union of the sutures of the skull, and in consequence general compression of the brain is supposed to occur. To give the brain room to enlarge craniectomy has been performed, but with indifferent results. The probabilities are in favor of the small crania being the result of deficient development of the brain, instead of its cause. The arrested development may involve the entire brain or only certain regions. Usually, however, when a considerable portion of the brain is small, other portions intimately associated with it by commissural fibres are imperfectly developed. In atrophy of one lateral lobe of the cerebellum the opposite corpus striatum and olivary body may be affected, while the cerebral hemispheres may be normal. Atrophy of one cerebral hemisphere is usually associated with atrophy of the opposite lobe of the cerebellum. It is rare for one entire cerebral hemisphere to be equally affected, but certain portions show greater changes from the normal than others. The possible causes of atrophy of the brain—or better termed arrested development—are too numerous to be considered in this article.[2] Partial atrophy of a hemisphere takes place more commonly after birth, but it may occur during intra-uterine life, when it is probably due to the same cause, meningeal hemorrhage, that seems to be the principal agent in producing partial atrophy after birth. This form of brain atrophy oftener affects the convex surface of the cerebrum, and especially the motor region. It may be bilateral or unilateral. Meningitis and hydrocephalus may cause the brain to waste.

[1] Consisting of equal parts of bran and pounded ice in a rubber bag.

[2] The entire subject of arrested development of the brain, malformations, etc. has been most ably discussed by N. E. Brill: *Nervous Diseases by American Authors.*

The symptoms will vary with the degree of atrophy or arrested brain development. In children that present an extreme degree of microcephalia at birth mental defects amounting to idiocy, arrested growth of the body, and imperfect special senses will be prominent symptoms during life. Convulsions and other disordered movements are not rare. When the atrophy has been due to meningeal hemorrhage, especially over the motor cortex, epileptic convulsions, monoplegia, hemiplegia, paraplegia, or diplegia (rarely), with more or less mental disturbance, will be common.

The brain in persons of an advanced age is smaller, the substance firmer, and the ventricles and subarachnoidean space contain more fluid than in early life. It is usually very difficult to determine whether these conditions are within the natural involutionary changes of the degenerative period, or whether they are the result of pathological processes.

TUMORS OF THE BRAIN.

In this section I shall make no attempt to write exhaustively on intracranial tumors, especially in regard to their pathology, but will endeavor to treat the subject from a clinical standpoint.

Definition.—Tumors of the brain, as described in this section, embrace the morbid growths found within the cavity of the cranium, including localized meningeal thickenings or deposits, usually due to syphilis or tubercle, which give rise to clinical symptoms similar to those of a more sharply defined growth, and cysts of the brain, simple or parasitic, but not false cysts that occur as the result of hemorrhage or softening from occlusion of a vessel. Large aneurysms might be included among the intracranial tumors, but these will be described in the section on the Diseases of the Cerebral Vessels.

Etiology.—The same degree of uncertainty that is met with in trying to determine the etiology of tumors in other portions of the body is encountered in studying the causes of growths in the brain. If we accept the theory that all tumors have their origin in perverted cell formation and proliferation, and attribute this perverted cellular focal process to the influence of an irritant acting locally, we are no nearer the solution of the causation of intracranial growths than we were before beginning to theorize. Definite etiolgical knowledge in respect to tumor formation seems, as yet, impossible of attainment. All that we can do in regard to the etiology of tumors of the brain at present is to note the apparent causes and their probable results. By a careful study of numerous cases some general etiological data seem to be obtained. These indicate that certain conditions predispose to the formation of growths within the cranium; that others are apparently capable of acting as exciting causes; and that two forms at least, the tubercular and syphilitic, are due to diathetic influences. The parasitic tumors are caused by the present of echinococci or cysticerci in the body.

Predisposing Causes.—Age.—No time of life seems to be exempt

from tumors of the brain. Steffen[1] has reported a case in an infant four weeks old. Cerebral growths are rare in extreme old age. The third decade gives the greatest number of cases, about 20 per cent.; the first and fourth, 18.5 per cent. each; and the second and fifth, 14 per cent. each.[2] The age of the patient has considerable influence in determining the character of the tumor. Tubercular and cystic growths largely predominate in childhood and early youth, although the gliomatous and sarcomatous varieties are comparatively frequent during these periods; the gliomatous, sarcomatous, glio-sarcomatous, and syphilitic are most common in young and middle-aged adults; and the carcinomatous, as in other portions of the body, are met with more often during the degenerative period. In an interesting table compiled by Starr[3] from various sources, in which 600 cases of tumors of the brain are analyzed, 300 in children and the same number in adults, the relative frequency of various forms of tumor to age is shown as follows: Tuberculous: children 152, adults 41; gliomatous: children 37, adults 54; sarcomatous: children 34, adults 86; glio-sarcomatous: children 5, adults 25; cystic: children 30, adults 2; carcinomatous: children 10, adults 33; gummatous: children 2, adults 20; not stated: children 30, adults 41. The frequency of carcinomatous growths in children, as shown by Starr's statistics, strikes one as remarkable in view of the statement made by Gowers that in 650 cases of various intracranial growths only 2 diagnosticated carcinomatous occurred during the first twenty years of life. It is probable that some cases of sarcoma have been labelled carcinoma without a careful microscopic examination.

It is supposed to be possible for a gumma to occur in children from hereditary influences, but the great infrequency of such an apparent result, while hereditary syphilis in children is so common, and the ease with which the virus of syphilis may find access to children after birth, especially through their wet-nurses, leaves the subject open to doubt.

Sex.—Males suffer nearly, if not quite, twice as frequently as females, and the preponderance of the liability in favor of the male sex seems to be as great in children as in adults, so that none of the theories that have been advanced to account for the difference in sexual liability are satisfactory. After the age of fifty the two sexes suffer with almost equal frequency (Gowers). It would seem, then, that the cause for the increased liability of the male sex to tumor of the brain must be sought in conditions connected with the developmental and active periods of sexual life.

Hereditary influences may play some part in determining the character of a cerebral growth when other causes that are capable of exciting a tumor are active, but its importance seems to be slight. Whether worry, anxiety, alcoholic indulgence, cerebral congestion, and depressed states of the central nervous system are factors favoring the formation of cerebral growths we are almost totally ignorant.

Exciting Causes.—Injury is almost the only exciting cause of tumor of the brain that has been studied, and the importance of this agent is easily overestimated, as persons who suffer from organic brain disease

[1] Knapp: *Intracranial Growths*, p. 2.
[2] Gowers: *A Manual of Diseases of the Nervous System*, 2d ed., vol. ii. p. 489.
[3] M. Allen Starr: *Brain Surgery*, p. 202.

are prone to attribute the cause to a blow on the head, notwithstanding the supposed injury may have been trivial and insignificant. But, after excluding all the improbable cases attributed to this cause, there remain a few, noted by nearly every observer, in which the first symptoms of the growth followed so closely upon the injury to the head that there seemed to be a direct relation between the trauma and the development of the cerebral tumor. In persons who are the subject of syphilis or tuberculosis an injury to the head appears to be sufficient, in many instances, to determine a local manifestation of disease in the brain : the result may be a growth or one of the other forms in which these diseases may become active in the cerebral structures. If this is true of persons suffering from these diathetic influences, it is probable that injury to the head in those who are suffering from a growth in other portions of the body will have a tendency to cause a growth in the brain.

Diathetic Influences.—Syphilitic and tubercular growths are due to diathetic influences. Whether other tumors have a similar cause we are not in a position to decide. Some writers have called attention to the possibility of certain intracranial growths having their origin in micro-organisms.[1]

Pathology.—In this section only those features in relation to pathology of especial interest to the general clinician will receive attention. For a full and detailed description of this subject the reader is referred to works devoted to special pathology.

Intracranial tumors are by no means of rare occurrence. They may develop from the cranial bones, from the membranes of the brain, or from the brain itself. Almost any form of tumor may arise within the cranial cavity, but a very few varieties, the tubercular, syphilitic, gliomatous, and sarcomatous, constitute more than nine tenths of all the tumors of the brain ordinarily met with clinically. It is impossible to determine with any degree of accuracy the frequency with which syphilitic growths of the brain occur, as a large proportion of these cases, presenting the classical symptoms of tumor of the brain, yield temporarily or permanently to antisyphilitic treatment, and are lost sight of by the observer. Gowers states that "the two groups, tubercular and sarcomatous" (including gliomatous), constitute together about four fifths of non-syphilitic tumors of the brain. Carcinomatous tumors are unusual except as secondary growths, and occur especially in advanced life. Parasitic tumors seem to be comparatively common in some portions of the world, but they are rarely met with in the United States. Fibromata, osteomata, cholesteatomata, angiomata, psammomata, lipomata, and neuromata are of infrequent occurrence in the cranial cavity. Lipomata and neuromata, according to Gowers, are the rarest of all forms of tumors of the brain. The gliomata are found only in the central nervous system and in the retina, and occur far more often in the brain than in the cord. The neuroglioma ganglionare is primarily a tumor of nerve-tissue origin.

The frequency with which tumors occur in different portions of the brain varies in childhood from adult age. Starr's table, already referred to, consisting of 300 cases in children collected from numerous authors, shows that nearly one half of the cases of tumor of the brain occurring in childhood are found in the cerebellum, pons, and medulla, while in

[1] Bramwell : *Intracranial Tumors*, p. 3.

the same number in the adult only a little more than one fifth were located in these portions of the brain. The cerebellum in childhood seems to be a little more than twice as often the seat of tumor as in adult life, but the cortex of the cerebrum in the adult is the seat of tumor six times as often as in childhood. Taking all of the available statistics of tumors of the brain occurring at all times of life, it is found that about one third of the cases occur in the cerebellum, pons, and medulla. In the adult the order of frequency regarding the seat of the tumor is, cortex cerebri, cerebellum, centrum ovale, pons, central ganglia, medulla, corpora quadrigemina, and crura; in childhood, cerebellum, pons, centrum ovale, central ganglia and lateral ventricles, cortex cerebri, base, medulla, and fourth ventricle. It will be seen that inaccessible portions of the brain are much more commonly the seat of tumor in children than the cerebral cortex. It is rare for a tumor to be so situated in the brain of a child as to be accessible to the surgeon's knife.

Tubercular Growths.—The tubercular is the most common form of brain tumor, especially in children, in whom it is nearly as common as all other forms of tumor combined. Tuberculosis of the meninges, with considerable thickening of the pia and unattended by the acute symptoms of tubercular meningitis, may give rise to the symptoms of a circumscribed growth. My experience leads me to believe that tuberculosis of the meninges, especially of the pia (not tubercular meningitis), occurs more often in the adult than in childhood, although it may take place at any time of life.[1] Its most common seat is at the base, involving the optic chiasm and adjacent parts and the anterior portion of the pons and medulla. The tubercular growths, or "solitary tubercles," as they are sometimes incorrectly termed, may take place on the surface or deep in the substance of the brain. These tumors, as well as tuberculosis of the membranes, are nearly always secondary to tubercle in other portions of the body. The bloodvessels and lymphatics are the channels through which the bacilli find access to the brain and its membranes from distant portions of the body. If the lymphatics have been the route through which the infecting germs have found an entrance into the cranial cavity, the membranes, especially the pia, rather than the brain substance, are likely to suffer most. The most usual sites of tubercular growths are the cerebellum, the cerebrum (cortex and centrum ovale), the pons, the central ganglia, the corpora quadrigemina and crura cerebri, and the medulla, nearly in the order named. The growths are non-vascular or nearly so, and appear on ocular inspection and palpation as firm rounded or irregular masses. On section the centre is found yellowish-white in color, and the consistence varies from semi-liquid to cheesy or firm material, which in rare instances is calcified. The peripheral parts are softer than the centre, and consist of granulation tissue, within which miliary tubercles may often be found. The size varies from a nodule a few millimetres in diameter to one four or five centimetres in diameter (from the size of a pea to that of a turkey egg). In more than one half the cases there are two or more tumors, and in some instances as many as ten or twelve have been found. Bloodvessels invaded by the tubercular process may be

[1] This is especially true in Colorado, where such great numbers of chronic tuberculous subjects are found.

occluded by infiltration of their walls with tubercular inflammation, which results in thrombosis. Sometimes tubercles enter the affected vessels and are thus carried to other portions of the brain, which they infect. The brain substance adjacent to the tubercular growths is usually atrophied or softened, but not infiltrated with tubercles. In rare instances a small collection of pus is found in the softened brain tissue adjacent to the tumor. If the mass ceases to grow, it may become partially encapsulated by a fibroid process at its periphery, and its interior may become calcified.

Syphilitic Tumor.—Syphilis, like tuberculosis, within the cranium gives rise to thickening of the meninges, especially at the base in the region of the optic chiasm, and may cause symptoms that are difficult to distinguish clinically from a more circumscribed growth. Syphilitic growths of the brain are of more common occurrence than autopsies reveal, as they not seldom disappear under timely and appropriate treatment. This form of cerebral tumor is almost always the result of acquired syphilis, hence its extreme infrequency in children. Bramwell states that "this is the most common form of new growth in adults between the ages of twenty-five and fifty." He is probably correct if we include among the syphilitic growths of the brain the meningeal thickening which gives rise to symptoms of a tumor. Tumors of the brain of syphilitic origin usually occur from three or four to ten or fifteen years, or even longer, after the initial lesion, although they may develop within a year or even a few months from the time of infection. Not infrequently a gumma of the brain takes place in persons in whom the secondary symptoms were so slight as to be entirely overlooked. Syphilitic growths are usually found on or near the surface of the brain connected with the membranes, the convex surface in or adjacent to the Rolandic region, the base, and the pons being the regions most commonly affected. They are rare in the cerebellum and the central ganglia. When they are situated deep in the substance of the brain it is probable that they are connected with a process of the pia or with the wall of a vessel. The growth is irregular in shape and presents two forms—the juicy, grayish-red, or jelly-like mass, which is fairly well supplied with capillaries, and may to some extent infiltrate the surrounding tissue; and the yellowish tumor, which is often quite firm, and apparently does not infiltrate the adjacent brain substance. The tumor varies in diameter from a few millimetres to several centimetres. On section the yellowish or rather firm growth presents irregular areas of caseous softening, fibroid induration, and even calcification in the older cases. Sometimes, as a result of treatment apparently, these growths are in an advanced stage of degeneration; they are shrunken and hard, and in some cases the most of the central portion of the tumor has disappeared and is replaced by a straw-colored watery fluid. The walls of the arteries adjacent to the growth are often thickened and the calibre of the vessel is narrowed; the surrounding brain tissue is softened and the overlying meninges are inflamed and thickened. The presence of fibrous tissue and irregular areas of caseation serve to distinguish the syphilitic from the tubercular growth.

Of the connective-tissue type of brain tumor the gliomata and sarcomata, on account of their common occurrence, are the only ones that

will receive attention in this section. Both varieties belong to the sarcomatous group of tumors.

Gliomata are less common in children than in adults, but are found as often in children as sarcomata. Excluding tubercular growths which preponderate in early life, gliomata and sarcomata are the most usual forms of brain tumor in children. In the adult, excepting syphilitic tumors, in point of numbers sarcomata stand first, gliomata second, and tubercular growths third. In children gliomata invade the cerebellum and pons in about two thirds of the cases; in the adult the cortex cerebri is the seat of the growth in more than one third; next come the centrum ovale and the central ganglia and lateral ventricles; and fourthly, the cerebellum. Glioma is only found in the central nervous system, and seldom in the retina. It arises from the neuroglia and never involves the meninges primarily. It never forms metastasis, and is probably only malignant on account of its situation, its infiltrating qualities, and its tendency to recur after removal. According to Gowers, it is single in nine cases out of ten. It varies greatly in size from a small tumor to one in which the dimensions may be greater than those of any other form of intracranial growth. Of all forms of brain tumor, this has the greatest tendency to infiltrate the surrounding tissues. The line of demarcation between the growth and the gray cortical substance is rarely distinct, and the tendency of the cells of the tumor to infiltrate without displacing the brain tissue renders it difficult, even in the centrum ovale, to determine where normal cerebral tissue begins. The consistence of glioma is frequently nearly that of healthy brain substance, so that on exposing during life a portion of the brain that is the seat of such a growth, only the yellowish and lustreless appearance of the exposed cortex, with increased cerebral pressure and the absence of pulsation, indicate the presence of a tumor. When the cortex which is the seat of glioma is exposed after death, it appears swollen, but usually retains the normal contour of the cerebral convolutions. In some cases the growth may present a nodular appearance on the surface of the cortex. Glioma is quite vascular, hemorrhage occurs, and thus a cyst may form in connection with the tumor. In some instances when the tumor is small and situated in the cortex, a hemorrhage may have destroyed all appearances of a growth. Under such circumstances microscopic examination of the surrounding brain tissue will reveal the typical cells, as was found in one case seen by me a few years ago. The brain tissue surrounding the growth is rarely softened, and still more infrequently does hemorrhage occur into the surrounding brain substance. On section the tumor presents a yellowish and grayish-red appearance, and often looks very much like congested cortical brain tissue. The terms fibro-glioma, when the tumor is quite firm, myxo-glioma, when mucoid in appearance and very soft in consistence, and glio-sarcoma, when it partakes of the cellular arrangement of both the glioma and sarcoma, have been applied to designate glioma varying from the ordinary form.

Sarcomata.—Sarcomata within the cranium may arise from the brain substance, from the meninges (pia or dura), or from the bone. They are probably about as common as gliomata, but the frequency of their occurrence is more influenced by age than in the latter. They occur

nearly three times as often in adults as in children, and the glio-sarcoma is found five times more often in adult than in early life. Gowers states that sarcomata occur more frequently without than within the brain substance. The cortex cerebri seems to be the portion of the brain most commonly invaded, the cerebellum and central ganglia being involved next in point of frequency, in the order named. Sarcomata do not usually infiltrate the brain tissue like gliomata, but they are surrounded by a zone of softening, and may often be separated from the brain tissue without difficulty. The tumor is usually single, especially when it arises within the brain substance, although it may be multiple. The growth may originate primarily within the cranium or develop secondarily from a similar growth in some other portion of the body, especially in the lungs; the melanotic variety, which is the most malignant, is usually multiple and secondary. The small round-cell sarcoma is more malignant than the spindle-cell. The latter is often the form when the growth springs from the dura. Sarcomata may grow rapidly or slowly, depending upon whether they are vascular and soft or poorly supplied with vessels and are hard. Sarcomata of the brain include several varieties, but limited space will not permit even a brief reference to them here. Sarcomata vary greatly in size: the larger, from five to ten centimetres in diameter, usually invade the convex surface of the brain—the smaller, the membranes at the base. The melanotic variety is usually small, and the numerous dark deposits may entirely change the normal appearance of the greater portion of the brain. The appearance of a sarcoma depends upon its variety, its seat, its size, its vascularity, and its consistence. When found in the cortex it is usually wedge-shaped. On section it is white or grayish, and hemorrhagic, cystic, or fatty degeneration may be found to have taken place.

Carcinomata.—Cancer of the brain, like cancer in other portions of the body, is found in the degenerative period of life. It is probable that statistics are misleading in showing an unwarranted number of this form of tumor in childhood. One cannot escape the suspicion that certain forms of sarcoma in children have been classed as cancer. Carcinomata of the brain may be primary or secondary, simple or multiple. They commonly arise from the dura, although they are sometimes found in the centrum ovale and seem to invade the central ganglia more often than other growths in proportion to their number. They are usually soft, and not infrequently quite vascular. The surrounding brain tissue is softened and infiltrated.

THE PATHOLOGIC CHANGES IN THE CRANIUM AND ITS CONTENTS RESULTING FROM INTRACRANIAL TUMORS.—Those who most thoroughly familiarize themselves with the possible changes likely to occur from the presence of intracranial tumors will be the best prepared to appreciate the symptoms of tumor of the brain. Intracranial tumors usually give rise to increased intracranial pressure, to irritation, and to destruction of tissue. It is also important to bear in mind, especially in the study of localization, that a growth in one portion of the brain may inhibit the function of a region of the brain to which the tumor has not extended. The changes within the cranium are modified by the seat of the growth, by its size, by its course (whether rapidly or slowly growing), by its effects upon adjacent structures (whether infiltrating or

encapsulated), and by the complications. Pathologic changes may take place in the brain substance, in the vessels, in the membranes, in the intracranial portions of the nerves, in the quantity of the cerebro-spinal fluid, and in the bones of the cranium.

Increased intracranial pressure results from enlargement of the parts in which the tumor is situated and from pressure on the afferent vessels, thus interfering with the return circulation from the brain. If the growth is situated above the tentorium, the parts below it are not subjected to much increased pressure, unless the growth is very large and unyielding, until late in the course of the morbid process, when general œdema of the brain may become manifest. On the contrary, subtentorial tumors not only press upon the cerebellum, pons, and medulla, but the circulation in the veins of Galen is often obstructed, and results in distention and dilatation of the ventricles with a watery fluid, which increases the entire intracranial pressure. On account of the falx cerebri, the pressure is greatest in the hemisphere which is the seat of the tumor. The greater the vascularity and infiltrating character of the growth the greater the increase of intracranial pressure, although there are exceptions to this, especially in glioma. Other things being equal, the pressure will be increased in proportion to the size of the tumor. If the morbid process is situated in the corpora quadrigemina or adjacent parts posteriorly to these, a small nodule, not more than a quarter or half an inch in diameter, may increase the pressure within the cranium more than a growth four times as large located in the centrum ovale. A slowly-growing tumor gives rise to less disturbance than one that runs a rapid course. An encapsulated or fibrous growth exerts less pressure than an infiltrating one. The principal complication that results in much increase of intracranial pressure is hydrops ventriculorum.

As a result of an intracranial growth irritation takes place both in the tissues immediately surrounding the tumor and in remote parts of the brain. Irritation of the cortex, especially in the Rolandic region, is often manifest by convulsive seizures. Such phenomena are much less common when the growth is situated in the white substance. It is probable that the irritation of the membranes accounts for much of the pain. Irritative phenomena are quite prominent when the growth is situated at the base or below the tentorium.

Destruction of tissue is an almost invariable result of a tumor within the cranium. This may occur from the infiltrating character of the growth or from softening caused by direct compression of the parts or interference with the blood supply. As a result of irritation there may be a temporary increase of manifest function, as evidenced by convulsions, etc., while destruction of a part is shown by lessened or abolished power (paresis or paralysis). Destruction of one portion of the cortex may, after a time, be compensated for by another cortical area performing its function. Destruction of white matter (containing conducting fibres) is never completely compensated for by another portion of the brain. So we are justified in saying that irritation is most resented by cortical gray matter, and destruction of tissue most nearly permanent when it occurs in the conducting paths. All the conditions that are found to modify the degree of intracranial pressure affect, to a greater or less extent, irritation and destruction of tissue.

The pathological changes occurring in the brain tissue as a result of tumor are—increase of the connective-tissue cells (Deiter's cells), most pronounced in the immediate region of the growth, enlargement of the parts, anæmia, passive congestion, hemorrhagic extravasation, and softening. The convolutions are often flattened. In the vessels we may have the walls inflamed and thickened and the calibre obliterated, or the vessels may be so weakened as to be unable to resist the blood pressure and hemorrhage may be the result. The membranes in the immediate vicinity of the tumor, especially if the latter approaches the surface of the brain, may be inflamed and thickened. The cranial nerves affected by a growth may be irritated, inflamed, and atrophied. In most cases of intracranial growths the quantity of the cerebro-spinal fluid is increased. This is most marked when the tumor is situated so as to exert pressure on the veins of Galen and on the parts in the neighborhood of the corpora quadrigemina. The cranial bones are sometimes rendered very thin from the pressure of intracranial growths. This may occur to any of the bones when the tumor is deep seated, resulting from increase of general intracranial pressure, but it is most marked in the cases in which the neoplasm is large and superficial, when the thinning of the bones takes place over the growth. Actual perforation of bone has been observed.[1]

Symptoms.—The symptoms of intracranial growths may be as variable as the possible pathological conditions that result from such tumors. It is probable that a growth within the cranium rarely, if ever, occurs without giving rise to symptoms, although they may be so slight as to be entirely overlooked, or if observed their significance may be totally misinterpreted. It is quite evident that the more thoroughly and systematically all affections of the brain are studied the less frequently will an unsuspected tumor of the brain be found at the autopsy, and it will be extremely rare for such a lesion to exist in cases in which no organic affection of the brain had been suspected; provided, however, that sufficient time and opportunity had been afforded the observer to study satisfactorily the history and symptoms. When it is remembered that the functions of many portions of the brain are comparatively unknown, and that slowly-growing non-vascular tumors may occur in regions of the brain the functions of which are best understood, without giving rise to any definite symptoms, the difficulty of determining the character of the lesion, and often the danger of mistaking an organic trouble for a functional one, will be appreciated. Fortunately, however, tumors of the brain in the vast majority of cases are attended by symptoms sufficiently distinct to point unmistakably to a lesion of an organic nature, and in many to its true character and its exact localization.

The usual symptoms may be classed as diffuse or general and focal or localizing. Generally, the former are the first to attract the attention of the patient, but in a few cases he dates the beginning of his illness from the manifestation of the latter. When the general symptoms are the first, the patient is annoyed by headache, intermittent or constant, but attended by periods of exacerbation. This may go on for weeks or months before dizziness, nausea, and apparently causeless vomiting are complained of. Not infrequently disturbances in vision or a general

[1] Knapp; Gowers; Bramwell.

convulsion occurs soon after the headache has become severe enough to interfere seriously with the comfort of the patient. In the few instances in which focal symptoms have been the first to attract attention convulsive movements, limited to a group of muscles, to one limb, or to one side of the body, or more often affections of speech, determine the sufferer to seek medical advice. As the disease advances the early symptoms become more marked, and numerous others are added, much to the discomfort and incapacity of the patient. Headache, if not severe before, soon becomes agonizing; vision gradually or rapidly lessens; nausea and vomiting become more troublesome, and the patient may emaciate; sustained mental effort is impossible, both on account of the headache which it usually augments and on account of the mental deterioration resulting from brain disturbance; walking may become difficult or impossible, either from paralysis or from interference with muscular co-ordination, usually due to a growth in the posterior cerebral fossa or in the region of the corpora quadrigemina; the special and general sensory phenomena may be marked by deafness, areas of hyperæsthesia or anæsthesia, or a condition of more or less complete hemianæsthesia. Various respiratory and circulatory disturbances may be present, and finally the unfortunate sufferer, bedridden and passing the discharges from the bowel and bladder into the bed, becomes oblivious to nearly everything except his suffering, gradually sinks into a stupor, then into coma, and death fortunately ends the distressing scene. In a few cases convulsions cause death before the patient dies exhausted from pain, from interference with brain function, or from inanition. It occasionally happens, especially in those cases attended by an inordinate appetite, that the body weight increases up to a short time before death.

For convenience of study it seems desirable to divide the symptoms into general and localizing. It must be remembered, however, that there is scarcely a general symptom that at times is not an aid in localizing the lesion, and some of the focal or localizing symptoms are often the strongest evidences, not only of an organic lesion, but of its being a tumor.

General Symptoms.—Among these, three are pre-eminent in importance, and on this account have been styled the classical symptoms of tumor of the brain, especially of the cerebellum. They are headache, double optic neuritis, and vomiting.

Headache is often the earliest symptom, and is usually one of the most constant and distressing. If all cases of tumor of the brain were carefully observed from their earliest manifestation to their termination, it would be exceedingly infrequent that one would be found in which during some portion of it course this symptom, due to the pressure of the growth, was entirely absent. If we are guided by published statistics, we shall be misled in regard to the frequency of headache. In the excellent article on tumors of the brain by Mary Putnam-Jacoby it was present in 423 cases out of a total of 568, or in about 74 per cent.,[1] while Mills and Lloyd in 100 cases found it recorded as absent in only 5 per cent.[2] Of 50 cases personally observed, and in which the diagnosis was verified by autopsies, this symptom was appar-

[1] *Reference Hand-Book of the Med. Sci.*, vol. i. p. 668.
[2] *A System of Practical Medicine, edited by Pepper*, vol. v. p. 1033.

ently absent in only 2, and in both of these there was slight and unusual headache, for them, on one or two occasions during the course of the disease. In one of these the pain lasted two weeks. The tumors were both gliomatous in structure. The pain is often constant, with periods of exacerbation; occasionally it intermits for weeks or months, especially if the patient is taking large doses of potassium iodide, even when the growth is not syphilitic. In a few instances it presents a periodicity that simulates malarial headache. The distress caused by this symptom is often most agonizing for weeks or months, and would probably lead the sufferer to commit suicide if he were not relieved by anodynes. Fortunately, the severe exacerbations are not often of long duration. In many, while the suffering may interfere with sleep and mental exertion, it is much less intense than in the few cases, and in a number the pain ordinarily amounts to little more than an uncomfortable cephalic fulness or tightness, with an occasional exacerbation. The pain may be lancinating, rending, stabbing, dull, heavy, or boring in character. It is often most severe when the tumor is rapidly growing when situated in the cortex, on the convex surface, in the mid-brain, or in the cerebellum; least distressing in slowly-growing tumors, especially when they are in the centrum ovale. Personal observation leads me to the conclusion that subtentorial growths, especially when situated so as to exert pressure on the middle lobe of the cerebellum, and directly or indirectly interfere with the circulation in the veins of Galen, are attended with the most constant and often the severest headache. It is increased by everything that augments the quantity of blood in the cranial cavity, such as coughing, sneezing, stooping, and mental or physical exertion. It may be diffuse, felt equally in all portions of the head, unilateral, or limited to a small area; not infrequently it is found in more than one region of the head, when it is most commonly frontal and occipital. Its location is no positive indication of the seat of the disease. It is rare for a tumor in the frontal region to give rise to pain in the occipital, but growths in the latter region are not infrequently attended with pain in the former. When the pain is persistently referred to the lower occipital region, the tumor is usually located below the tentorium, and is frequently associated with pain radiating down the posterior cervical region. A tumor in one cerebral hemisphere may give rise to pain on the opposite side of the head, and nowhere else, but unilateral occipital headache usually corresponds to the side on which the growth is situated. Gowers states that "when the growth is in the white substance the pain is often frontal, whether the disease is in the frontal or parietal lobe."[1] When the tumor is superficial, especially when the membranes are involved, the seat of the pain generally corresponds with that of the disease, and under such circumstances tenderness on gentle percussion may be found over the seat of the growth, and nowhere else. In many cases the headache is associated with a feeling of fulness, pressure, or confusion within the cranium, and sometimes with a sensation as if a band were drawn tightly across the forehead.

It is useless to speculate in regard to the cause of the headache. It is generally attributed to increased intracranial pressure, which is sup-

[1] *Loc. cit.*

posed to give rise to irritation of the dura and its sensitive nerve supply from the fifth cranial nerve.

Choked Disk, Optic Neuritis, and Optic-nerve Atrophy.—Among the symptoms of intracranial growths, choked disk, or a condition simulating it, is next to headache in frequency, but not less in importance. It is not, as a rule, a very early symptom, but it occurs during the course of the disease in more than three fourths of the cases. It begins acutely, and only a few days or weeks may elapse from the time of its first appearance until it has reached a degree of considerable intensity, when the disk may have increased to twice it normal diameter, and present an elevation in its centre that will give a difference in refraction between this and the fundus of two or more dioptres. Central vision may be normal, and the fields for light and bright objects but little impaired, until the atrophic stage sets in ; so that an ophthalmoscopic examination, often repeated, is the only means by which this important symptom of brain tumor may be detected. It is usually bilateral, although the morbid process is farther advanced in one eye than in the other. In rare instances it is unilateral, when it probably indicates that the disease is anterior to the optic chiasm.[1] Knies states that "simple neuritis, terminating in atrophy, is found less often than choked disk in cerebral tumors. It happens particularly in tumors of the frontal lobes, in which the growth is situated comparatively close to the optic nerve."[2] After the stage of atrophy has been reached it may be difficult to determine whether this has followed choked disk or simple neuritis. Under such circumstances "only the sinuosity of the vessels near the previously swollen disk enables us to recognize that the atrophic discoloration (white atrophy) has followed a choked disk."[3] Choked disk seems to occur least frequently in tumors of the centrum ovale, especially in the middle and anterior portions of the brain, and most frequently when the growth is situated in the cerebellum and adjacent parts, especially when the corpora quadrigemina and great ganglia at the base are involved. In the section on Diagnosis the importance of choked disk will be further discussed (p. 463).

Vomiting is a frequent symptom of tumor of the brain, especially when the growth is located in the cerebellum near the middle lobe, just above the tentorium over this lobe, or in the region of the corpora quadrigemina. It occurs less frequently when the growth is situated in the Rolandic and prefrontal regions. It is probably found in nearly one half the cases of intracranial tumors. It is often associated with severe headaches, during the paroxysms of which it is most common. The vomiting may be projectile in character and not associated with taking food or with nausea. An attack is not infrequent in the early morning. When the tumor is situated in the cerebellum, adjacent parts, or in the corpora quadrigemina, vomiting is frequently produced by sudden movements of the patient's head, as in turning in bed and in rising from the recumbent to the semi-erect posture.

Vertigo in many cases is associated with vomiting, but it may be a most constant symptom, very annoying and entirely unassociated with vomiting. Like the latter it is most frequent in cases of tumor in the

[1] M. Allen Starr: *Nervous Diseases by American Authors*, art. "Tumors of the Brain," quoted from Knies. [2] Knies: *The Eye in General Diseases*, p. 147. [3] *Ibid.*

posterior fossa and adjacent parts. In compression of the pons vertigo is quite severe, but vomiting may be infrequent. True vertigo, according to Mendel of Berlin, is always associated with disturbance in the musculature of the eyes, and the inharmony of the movement of these muscles when of central origin is due to imperfect blood supply to the nuclei of the nerves innervating the eye muscles.[1]

General convulsions during some period of the disease are not of infrequent occurrence, and are probably found in one third of the cases of tumors of the brain in all situations. A seizure may precede the other symptoms by months. Only one attack may occur during the entire progress of the disease; usually, however, there are several, following each other at irregular intervals. In a few cases, especially of a syphilitic nature, several occur in one day. They may have all the characteristics of epileptic seizures, either of the grand or petit-mal variety. During the latter stage of the disease they may prove fatal, and their presence at any time indicates activity in the morbid process. As a general convulsion may result from a local spasm, the initial convulsive movements should be carefully studied.

Mental disturbance to a greater or less degree must be almost a constant attendant upon all tumors of the brain. It may be scarcely appreciable, amounting only to slight impairment of mental vigor, so that concentration and sustained efforts of the mind are irksome. As the disease advances apathy and failure of memory are noticeable. In a few instances maniacal excitement or mental depression, with hallucinations and delusions, is the most obtrusive mental symptom. Such patients have been committed to asylums, and the post-mortems have revealed the presence of unsuspected tumors that had been the cause of the mental derangement. Between the two extremes occur various phases of mental failure. Some are irritable, impatient, and vacillating; some are lethargic, somnolent, and extremely apathetic; and others present a degree of mental impairment amounting almost to dementia, especially late in the disease. The cases of mental disturbance approaching nearest to true dementia have usually been due to growths in the frontal lobes. Unless life is suddenly cut short by convulsions or by some other rapidly acting cause, stupor and coma precede death for several hours, and sometimes for days.

Insomnia is a very troublesome symptom in a few cases, and is apparently most common in syphilitic tumors. In such headache is probably the main cause of the sleeplessness.

Somnolence, recurring every few weeks and extending over periods of several days, has been observed. In one case observed by me each somnolent period was preceded by an apoplectiform seizure.[2]

Syncope has been witnessed in a few cases. Rarely do we meet with distinct apoplectic seizures due to hemorrhage. Slowness of speech is common in cases in which mental failure is pronounced, and disturbances in articulation are observed most often in tumors of the pons and medulla.

Few subjective *sensory disturbances*, except a feeling of fulness, pres-

[1] *Journ. of Nerv. and Ment. Dis.*, Sept., 1895, p. 593.

[2] "Tumor of the Cerebellum, with Bulimia and Recurrent Apoplectiform Seizures," *Boston Med. and Surg. Journ.*, Jan. 10, 1895.

sure, or confusion within the cranium, and sometimes a sensation likened to a band drawn tightly across the forehead, already mentioned in connection with headache, are found among the general symptoms. The surface temperature of the head has been carefully studied by a number of physicians. I have made several thousand surface temperature observations of the head in different forms of organic disease of the brain. Many tumors are attended with a heightened temperature of the head, some are not, while in a few the temperature over the seat of the growth is higher than over other portions of the head. The axillary temperature is usually nearly normal; in some cases it is elevated one or two degrees F. above normal, especially during attacks of apparent congestion or somnolent periods. Respiration may become embarrassed in tumor in any portion of the brain, and when the growth is not situated below the tentorium this symptom usually denotes general increased intracranial pressure. In such cases the pulse may be slow, labored, and intermittent. The deep reflexes are often increased on both sides of the body in tumor on one side of the brain, but usually to a greater extent on the side of the body most affected by the growth. Exceptions to this general rule are often observed in tumors of the pons and medulla, in which the knee jerks may be absent from time to time. The superficial reflexes may be lessened or lost on the side on which the deep reflexes are most exaggerated. Emaciation is frequently well marked in persons suffering from tuberculous, carcinomatous, and some other growths, but in sarcomatous and gliomatous growths nutrition may be nearly normal or increased if vomiting is not frequent enough to prevent the retention of food in the stomach. Albuminuria, polyuria, and diabetes have been observed in cases of tumor in various regions of the brain, but, as a rule, they are associated with marked increase of intracranial pressure, are more frequently found when the growth is at the base, and usually when it is beneath the tentorium. In the cases in which mental failure is not well marked voluntary control of the sphincters of the bladder and bowel is not seriously affected until the stage of stupor or coma is reached. Symptoms of a hysterical character are often blended with those of tumor of the brain, and in some cases these have been so prominent and obtrusive as to have led physicians of experience and undoubted skill to diagnosticate the case hysteria when a growth in the brain has been present.[1]

Focal or Localizing Symptoms.—By a careful study of these the seat of the growth can often be more or less accurately determined. They vary according to the location of the lesion, and may be direct and result from the invasion of a part by the tumor, or indirect and due to its interfering with the function of some portion of the brain more or less distant from the initial lesion. Both sets of symptoms are often present at the same time, and great care must be exercised in distinguishing the one from the other.

The Disturbance in Motility and Similar Symptoms that may Aid in Localizing the Growth.—Hemiplegia, usually gradual in its onset, is not infrequent when the tumor is situated in the centrum ovale, corpus striatum, crus, side of medulla, or in such a position so as to exert pressure on the motor fibres somewhere in their course from the cortex to

[1] Eskridge: *Journ. of Nerv. and Ment. Dis.*, vol. xii., Jan., 1885.

the cord. If the growth is in the centrum ovale and produces hemiplegia by pressure on the internal capsule, the leg may be involved more than the arm at first, the arm to a greater extent than the lower side of the face, the upper portion of the face escaping almost entirely. Under such circumstances paralysis will often be attended by hemianæsthesia and hemianopsia, and sensory aphasia if the lesion is on the left side of the brain. If the tumor affects the crus, pons, or medulla and causes hemiplegia, certain cranial nerves may be involved on the side of the lesion and the limbs on the opposite side of the body. Very infrequently hemiplegia comes on suddenly from tumor of the brain. In such cases the growth is usually situated in the centrum ovale, and the paralysis is the result of extensive softening or of hemorrhage. The presence of choked disk or other general symptoms of an intracranial growth will prevent mistaking the case for one due to a purely vascular lesion.[1]

Monoplegia, commonly paralysis of one arm, next of arm and one side of face, of face alone, least often of one leg, may result from a tumor in or below the motor cortex or in the crus, pons, or medulla. It is rare to have a lesion so limited in extent in the internal capsule as to give rise to paralysis of one limb alone. When the growth is cortical or subcortical the paralysis is usually limited, at first, at least to the distal portion of the affected limb, and this is not seldom the seat of convulsive movements equally limited in their onset. If the growth is in the crus, pons, or medulla, cranial-nerve symptoms on the side corresponding to the tumor, with paralysis of the limbs on the opposite side, will indicate the seat of the lesion. Bilateral paresis or paralysis may occur from bilateral tumors or from a single growth near the median line so situated as to affect the crura, pons, or medulla. If the bilateral symptoms are due to a lesion of the crura, the weakness of the extremities will probably be slight and both third cranial nerves may be affected; if to a growth of the corpora quadrigemina or cerebellum, the weakness is usually slight in degree and attended by other evidences of the exact situation of the tumor. If the lesion is in the lower pons or medulla, the legs will usually be involved to a greater extent than the arms, and cranial-nerve symptoms amounting almost to bulbar paralysis will be present.

Local convulsion, or *Jacksonian epilepsy*, in which the convulsive movements begin in the muscles of one portion of the body, and may for a time be limited to these, is more apt to be caused by a tumor of the brain than by any other lesion, and is often a most valuable localizing symptom. In the cases in which the convulsion is limited to one limb the muscles of the distal portion may alone be affected, and these are often weak or completely paralyzed for some minutes or longer after the convulsive movements have ceased. If the convulsion, after beginning locally, extends to other portions of the body, the muscles first affected will be the last to cease twitching, and usually will show temporary weakness. In some instances the convulsion may begin at one time in the hand, foot, or face, from the irritation being greater at different times in the different cortical centres. In such cases the seat of

[1] Eskridge: "Tumor of the Brain simulating a Vascular Lesion," *The Med. News*, Mar. 10, 1894.

the growth is usually indicated by paresis or paralysis of certain muscles. Convulsive movements have been observed to begin in the hand when the lesion has been in the paracentral lobule or occipital lobe.[1] It may be stated that, as a general rule, convulsive movements that begin repeatedly in the face, hand, or foot will indicate a cortical lesion affecting the motor area that presides over the muscles involved in the initial convulsive movements. Local subjective sensory phenomena may form the commencement of the motor spasm and may take the place of it. These consist of tingling, "pins and needles," or painful sensations in the distal portion of the extremity, and have the same localizing value as the convulsive movements. Sometimes attacks of tetanic rigidity, affecting principally the trunk, neck, and often the arm muscles, with consciousness preserved, blurred, or lost, are witnessed from subtentorial tumors.[2]

Contracture occurring from tumor of the brain denotes an irritative lesion in the motor tract. It develops with the paralysis rather than follows it, as is the case from vascular lesions, and has little significance as a localizing symptom except in those cases in which it is limited to a distal portion of one limb or is associated with marked wasting in the paralyzed muscles, when it usually denotes an irritative lesion in the motor cortical centre or near it. A fine tremor on slight movement, becoming decidedly coarse and jerky on exertion of weakened muscles, may result from tumors involving any portion of the motor tracts or the cortex of the brain, and has little value as an aid in determining the more exact location of the lesion. I have seen one case of extensive sarcoma of the pia in which large portions of the convex surfaces of both cerebral hemispheres were slightly pressed upon, attended by intention tremor of the arms very similar to that of multiple sclerosis. Cases of tubercular growths of the pons have been observed in which a jerky intention tremor has been present.[3] It is doubtful whether choroid or athetoid movements have any localizing value, as they have been observed in tumors of the thalamus, parietal and frontal lobes. Unsteadiness of the legs, often called cerebellar titubation, or a drunken man's gait, with a tendency to fall in different directions, is observed in tumors affecting directly or indirectly the middle lobe of the cerebellum or the corpora quadrigemina.[4] In both the ocular muscles are usually affected. Bruns believes that ataxia preceding the ophthalmoplegia indicates cerebellar disease, while a reversal in this order of the appearance of these symptoms points to disease of the corpora quadrigemina.[5] In a case observed by me a large tumor of the hinder portion of the corpus callosum pressed upon the corpora quadrigemina and caused marked unsteadiness of the legs, with a tendency to fall backward, but no disturbance in the ocular muscles occurred. Forced positions of the entire body, with an involuntary turning to the right or left in walking, and sometimes turning toward the right or left side in lying, have been ob-

[1] Gowers: *q. v.*

[2] Eskridge: "Tumor and Large Cyst of the Cerebellum, with Symptoms extending over Several Years." *Med. Rec.*, Aug. 17, 1895.

[3] Gowers: *q. v.*

[4] Ataxia similar to the cerebellar type has been observed in tumor of the frontal lobe (Bruns: *Deut. med. Woch.*, Mar., 1892).

[5] *Annals of the Universal Medical Sciences*, vol. ii. A–40.

served in tumor involving the middle peduncle of the cerebellum. Conjugate deviation of the head and eyes may occur from a tumor in the cerebrum or pons. From a paralyzing lesion in one cerebral hemisphere the deviation is toward the side of the lesion, and from it in an irritative one, causing spasm. The condition is often reversed when the lesion is below the middle of the pons. The cranial nerves may be involved from tumor of the corpora quadrigemina, crura, pons, cerebellum, and medulla, but most of the symptoms thus produced may be studied to better advantage under the head of Localizing Diagnosis (p. 293), in which the symptoms from tumors of various regions of the brain will be considered.

Sensory Disturbances.—Hemianæsthesia extending up to the median line of the body, with loss or impairment of all the special senses on the same side, may result from a tumor destroying or damaging the posterior one third of the posterior limb of the internal capsule. Hemiplegia more or less complete will exist on the anæsthetic side. The disturbances in all the special senses except sight will be unilateral, but vision will be lost in that half of each retina which corresponds to the side on which the tumor is situated, producing homonymous hemianopsia (blind fields) on the anæsthetic side of the body.[1] A small subthalamic tumor may cause hemianæsthesia, but the special senses, with the probable exception of vision, will not be materially affected without producing hemiplegia, partial or complete. An extensive tumor in the centrum ovale at the junction of the occipital, temporo-sphenoidal, and parietal lobes may give rise to partial hemianæsthesia, but here the special senses, except sight, usually escape, even though hemiplegia exists. Sensory disturbance from the presence of tumors pressing upon or directly involving the pons and medulla would be attended by affections of the cranial nerves. Various sensory phenomena may occur from tumors in the motor cortical region. There may be pain in the distal portion of the affected limb, with partial loss of tactile sense and still greater impairment in the power of localization. Lesions in the posterior portion of the motor cortical area are attended by greater impairment of tactile sense than those involving the anterior margins of this region. The only peculiarity about the pain caused by tumors irritating the fifth cranial nerve is that when the first division of this nerve is involved and the second and third divisions are unaffected, the pain, instead of being neuralgic in character, is often complained of as headache.[2]

Hemianopsia and aphasia may be the result of tumors in several portions of the brain, and vary in character with the seat of the growth. Hemianopsia may be caused by a tumor in the occipital lobe involving the cuneus, by one in the centrum ovale at the junction of the occipital and temporo-sphenoidal lobes, by a small growth in the subthalamic region, by one in the posterior portion of the great ganglia, affecting the external geniculate body or the visual fibres from it, or by a growth invading one optic tract, the chiasma, or the lateral half of one optic nerve. If the hemianopsia is caused by a tumor posterior to the exter-

[1] The hemianopsia in such cases is probably not due to the involvement of the posterior portion of the internal capsule, but to pressure on the external geniculate body or on the visual fibres as they wind around the ganglia to reach the cuneus.

[2] Gowers: *q. v.*

nal geniculate body, it will be homonymous, and the lateral blind half field of each eye will be on the side opposite to that of the brain lesion, and the reflex of each iris will be unaffected; if by a growth affecting one external geniculate body or the optic tract, the blind fields will be similar in situation to those caused by a lesion posterior to the geniculate body, but the irides, normal on one side, will not respond to light when the rays are thrown into the eyes from the blind side, the hemiopic pupillary reflex of Wernicke. If the hemianopsia is due to a tumor in the occipital lobe involving the cuneus, there is neither motor nor sensory disturbance; if to one in the centrum ovale at the junction of the occipital, temporo-sphenoidal, and parietal lobes, the disturbance of vision is usually associated with some defect in muscular and localizing sensations, with very slight tactile-sense perversion, and not infrequently pronounced word-blindness when the lesion is on the left side in right-handed persons and on the right side in left-handed persons. A small growth, commonly syphilitic, in the subthalamic region, producing hemianopsia, causes some disturbance in general and special sensory phenomena on the side of the blind fields. A tumor situated in the posterior portion of the thalamus and pressing upon the sensory fibres of the internal capsule will cause, besides hemianopsia and hemianæsthesia, slight weakness and probably choroid movement on the blind side; while hemianopsia associated with hemiplegia, hemianæsthesia, and unilateral loss or impairment of the special senses would result from a tumor affecting the motor and sensory fibres of the internal capsule. Hemianopsia from a unilateral lesion in the tubercular quadrigemina differs from that produced by a tumor compressing one optic tract, in that the former is attended by an ataxic gait, while the latter would probably be associated with symptoms of basilar disease, but not with ataxia. Primary optic neuritis, loss of hemiopic pupillary reflex, and affection of the external ocular muscles would probably exist in each case. Hemianopsia produced by tumors pressing on any portion of the optic fibres and radiations posterior to the chiasma is usually homonymous and lateral, although sector-like defects in the fields of vision may possibly occur from tumors affecting only a small portion of the optic fibres and radiations of one side caudad of the chiasma; but they are usually due to a lesion in a portion of the cuneus. Hemianopsia is never homonymous from lesions in the chiasma or optic nerves.

Aphasia.—The faculty of speech is located in the left side of the brain in right-handed persons and in the right side in left-handed individuals. A tumor in the lower Rolandic region or in the centrum ovale of the temporo-sphenoidal lobe, or near the angular gyrus when in that hemisphere in which the faculty of speech mechanism is located, is usually attended by some form of aphasia. (See Affections of Speech as Localizing Symptoms in the section on Cerebral Localization, p. 309.)

Course and Duration.—The symptoms of tumor of the brain, as a rule, develop slowly, and are gradual but steady in their progress to a fatal end, unless relieved by the administration of medicines or by a surgical operation for the removal of the growth. In some cases the symptoms seem to develop rapidly; in others they are apparently

arrested for several months. Some tubercular growths may run their course and end in death in a few weeks, while others may extend over a period of many years. In some instances after the tumor has existed for several months, with a gradual increase of symptoms, death may suddenly take place with all the symptoms of an intracranial vascular lesion.[1] The average duration of tumor of the brain is about fifteen months, but the ordinary cases vary from a few months to two or three years. Death usually results from exhaustion, intracranial pressure, meningitis, especially in the tubercular variety, or from some intercurrent disease.

Diagnosis.—In the vast majority of cases of tumor of the brain a diagnosis can be made by a skilled diagnostician if a systematic and thorough method is pursued in the examination of the case and in the analysis of each individual symptom. Not infrequently the symptoms are so typical as to render the diagnosis apparently self-evident. It should be borne in mind that the most stupid blunders are usually made in the plainest cases on account of failure to observe the necessary precautions in drawing conclusions from observations. With the presence of the general symptoms—choked disk, headache, vomiting, dizziness, and convulsions, together with evidence of a focal lesion resulting in hemiplegia—the natural inference would be tumor of the brain, yet this formidable array of symptoms might be due to renal disease. None of the general symptoms and but few of the focal can be accepted unqualifiedly as evidence of tumor of the brain. The first problem that the physician should seek to determine in investigating suspected disease of the brain is whether the symptoms are due to an organic lesion or to functional disturbance. A multiplicity of symptoms of a functional nature is far less in importance in excluding organic disease than the presence of a single symptom that is due to organic trouble in diagnosticating a lesion of the latter character. A careful study of the manner and order of the development of symptoms and their association in an individual case, together with a complete history and a thorough examination, is an important aid in arriving at a logical conclusion.

Are the symptoms due to organic intracranial disease? So closely do the functional diseases of the brain at times simulate those of an organic nature that very few symptoms when taken by themselves can be positively asserted to be due to an organic lesion. Symptoms that are usually regarded as organic when caused by functional disturbance are, as a rule, temporary and fleeting in character, and the opposite, while true in the vast majority of cases, find a notable exception in multiple sclerosis of the brain. Marked muscular wasting in the distal portion of a paralyzed limb of cerebral origin, associated with flexor contracture and decided increase of the deep reflexes over those of a corresponding limb of the opposite side; wasting of the muscles of the face with reaction of degeneration not due to extracranial lesions; most cases of crossed paralysis or pronounced trophic disturbance in one or both eyes of cerebral origin; more than transient hemianopsia or sensory aphasia,—may be regarded in the vast majority of cases as of organic origin, although only one of these conditions exists. There are

[1] Eskridge: "Tumor of the Brain simulating a Vascular Lesion," *Med. News*, Mar. 10, 1894.

other and more frequent symptoms which are always very strong evidences of organic brain disease, but not positive proof of it. Among these the most prominent is optic neuritis or choked disk, which is usually double. While this symptom, when present, is always in favor of organic disease of the brain, and especially of an intracranial tumor, yet we must always bear in mind the possibility that it may be due to kidney disease, lead-encephalopathy, or profound anæmia, and, further, that a decided hyperæmia of the optic disks may be apparent in some hyperopic eyes. It is probable that the cases of optic neuritis reported in persons suffering from certain menstrual disorders are due to internal hydrocephalus or localized basilar meningitis. The subsequent history of some of these cases, especially the permanent blindness, lends support to this view.[1] When we call to mind the fact that kidney disease and lead-encephalopathy may be attended with optic neuritis, headache, vomiting, and convulsions, and that, in addition to these symptoms, the renal trouble may be attended with hemiplegia, the danger of mistaking this condition for organic disease of the brain is apparent.[2]

The swelling of the optic disks from kidney disease is not usually very pronounced, and the albuminuric retinitis, the albumin and tube casts in the urine, the hypertrophied left ventricle of the heart, the increased resistance of the pulse, and the general appearance of the patient would render the diagnosis of renal trouble comparatively easy in the vast majority of cases. Occasionally the choked-disk appearance from kidney disease is identical with that caused by tumor of the brain.[3] I have observed but one such case, but the difficulty in diagnosis was only apparent, because of the presence of numerous other symptoms of kidney affection. The form of Bright's disease commonly associated with optic neuritis is the chronic cirrhotic variety, in which no albumin may be detected for a time, and the tube casts may be so few as to be easily overlooked, but even in such cases the increased quantity of urine, its low specific gravity, and the condition of the heart, pulse, and vessels should make one cautious, and defer a positive opinion until further time for observation is afforded. It must be remembered that tumor of the brain has occurred in persons the subject of kidney disease. The optic neuritis from anæmia is less marked and usually develops more slowly than that from intracranial tumor, but this difference does not hold good in distinguishing it from the optic neuritis due to certain other organic lesions of the brain. Here, however, the extreme poverty of the blood, the pronounced general symptoms of anæmia, and the condition usually occurring in a young female should prevent a hasty diagnosis. In one case of optic neuritis associated with profound anæmia in a young female coming under my care the headache was located for weeks in the left temporal region, and was felt in no other portion of the head. Iron, quinine, arsenic, and a generous diet soon relieved the anæmia and the localized headache and optic neuritis disappeared. Lead-encephalopathy, attended with optic neuritis, is dis-

[1] *Intracranial Tumors*, Bramwell, p. 131.

[2] Prof. J. M. DaCosta has observed choked disk and symptoms of brain tumor from the chloral habit: *Univ. Med. Mag.*, Mar., 1896, p. 430.

[3] And a brain tumor may be attended by an optic neuritis, with an appearance considered characteristic of albuminuric retinitis.—Geo. E. deSchweinitz: *Univ. Med. Mag.*, Mar., 1896, p. 420.

tinguished from organic disease of the brain by the presence of symptoms of lead-poisoning, such as the blue line on the gums, anæmia, intestinal colic, constipation, and the presence of lead in the urine. The hyperæmia of the optic disks sometimes seen associated with errors of refraction is stationary and never very marked. If one is on his guard, a mistake is almost impossible from this cause.

Having discussed the difficulties of a diagnosis in cases in which optic neuritis is present, we shall often encounter greater ones in trying to determine the nature of the disease in those cases in which it is absent. Persistent headache and obstinate vomiting, so common in certain organic diseases of the brain, especially when this is due to tumor, are rarely present without changes in the eye-grounds. Is the hemiplegia, monoplegia, spasm, contracture, or hemianæsthesia due to organic disease or to hysteria? In cases of organic disease attended with unilateral motor disturbance the deep reflexes are increased in the affected limbs to a far greater extent than we find in hysteria. They may be increased on both sides of the body in hysteria and in hemiplegia of an organic nature, but the marked difference between the deep reflexes of the affected and non-affected side observed in organic disease is not found in hysteria. Hemianæsthesia, including the special senses, as observed by Gowers, "is one of the rarest effects of cerebral tumor, and is absolutely unknown from this cause unless associated with loss of motor power." Whether it occurs from tumor or other forms of organic disease affecting the posterior portion of the internal capsule, the hemianopsia, homonymous in character, will differ from the "crossed amblyopia" sometimes seen in hysterical subjects. An extended differential diagnosis between hysteria and organic disease of the brain is impossible in this connection, and for a full account of the former disease the article on Hysteria must be consulted. It is probable that distinct forms of persistent sensory aphasia never occur from hysteria, but temporary and variable aphasia, both sensory and motor, may be due to hysteria. Most of the cases of hysterical aphasia have been of the motor variety, partaking of the character of aphonia rather than true motor aphasia. The occurrence of the symptoms of cranial nerve lesions, if these were present, would aid in excluding hysteria. No case should be diagnosticated hysteria until it has been very carefully and minutely studied without detecting any positive evidence of organic disease. The fact that hysterical symptoms are present should not influence the diagnosis in cases in which one positive symptom of an organic nature can be detected.

In some cases it is extremely difficult, and even impossible, to determine whether convulsions, when these are the only symptoms present, are due to so-called idiopathic epilepsy or to organic disease of the brain. If the convulsions are general and unattended by any other symptoms, a diagnosis will be impossible. If they have a local commencement, the case should be treated as though it were organic, and an operation for its relief instituted if the supposed lesion is in an accessible portion of the brain. By following this rule I have seen no harm result, and in three cases a permanent cure has been effected.

It is necessary to bear in mind that a febrile condition, simulating

in its periodicity malaria, may occur in the course of organic disease of the brain, especially in connection with abscess and tumor.

Is the lesion due to tumor? If after careful analysis of the symptoms the conclusion is reached that the intracranial disease is organic, the next step in the diagnosis is to determine the character of the lesion, or, in this particular instance, is it a tumor? Organic diseases of the central nervous system may be divided in regard to the manner of their development into three classes—the sudden, acute, and chronic. The first class, developing suddenly, includes most of the vascular lesions, hemorrhage, thrombosis, and embolism; the second, acute and inflammatory in nature, requires time for the process to reach its height; while the third embraces all those diseases that are gradual in their development, and prominently among these is tumor of the brain.

It infrequently occurs, however, that a person with tumor of the brain has suffered from ill-defined brain symptoms for a considerable time, and is not seen by any physician regularly until sudden paralysis with stupor, deepening coma, and stertorous respiration, develops.[1] Under such circumstances the history, with the condition of the optic disks, would probably determine the nature of the lesion. In the absence of the previous history and with no changes in the optic disks a diagnosis would, in all probability, be impossible.

In the second class, or the diseases that develop acutely, meningitis is about the only one that is likely to be confounded with tumor of the brain. Acute meningitis, even of the tubercular variety, when it begins in the ordinary manner and runs a regular course, will not be difficult to distinguish from intracranial tumor. It begins more acutely, runs a more rapid course, produces more commonly one cranial nerve symptom after another, is attended with less swelling of the optic disks and greater febrile phenomena than a cerebral growth. But a tubercular condition of the membranes, with which there may exist small tubercular nodules or even a growth of considerable size, may lie dormant within the cranium for a considerable time until some disturbing cause, a blow to the head, mental or physical exhaustion, exposure to cold or the sun's rays, lights the latent condition into activity. Under such circumstances the symptoms will partake of the character of meningitis and intracranial tumor, those of the latter predominating when the growth is large, and those of the former when the deposits have been small. Every case must be studied with care, and each symptom analyzed in the light of the history of the development and progress of the disease. It is impossible to lay down safe rules for a differential diagnosis. In those cases in which a tumor has existed previously to the outbreak of acute cerebral symptoms not infrequently a history of attacks of apparently causeless vomiting, attended with severe headache, may be obtained. As a rule, the longer the duration of the disease beyond the first month the greater are the probabilities of a tumor; but it must not be forgotten that cases of tubercular meningitis, both in the child and in the adult, have run a course of six to eight months. Optic neuritis, followed by primary optic-nerve atrophy, is common in prolonged meningitis, whether the inflammation is limited to the base or to the ependyma of

[1] Eskridge: "Tumor of the Brain simulating a Vascular Lesion," *Med. News*, Mar. 10, 1894.

the lateral ventricles, while choked disk with secondary atrophy is usually associated with tumor. A febrile process is more common in meningitis than in cerebral growths, although numerous cases of the former have been reported with subnormal temperature, and of the latter with considerable rise of the body heat. Bilateral symptoms are more frequent in meningitis than in tumor, but here, again, by the occlusion of a vessel at the base, as I have observed in one case, or by localized thickening of the membranes over the motor region on one side, unilateral symptoms from meningitis may simulate those of tumor. Tubercular tumors occasionally give rise to meningitis.

Among the diseases of the third class, or those that are gradual in their development, aneurysm, chronic cerebritis, chronic hydrocephalus, chronic meningitis, paretic dementia, multiple and diffuse sclerosis, and chronic abscess may present symptoms similar to tumor of the brain.

Aneurysm, considered clinically, is an intracranial growth, and gives rise to all the symptoms of a more solid structure. The only absolute diagnostic sign is the detection of an aneurysmal bruit within the skull. As this is considered in connection with the diseases of the cerebral vessels, no further reference need be made to it in this connection.

Chronic cerebritis is a very rare disease. Its principal symptoms are headache, vomiting, and distinct optic neuritis, without localizing signs. It is evident that this condition cannot be distinguished from a cerebral tumor unattended by focal symptoms.

Chronic hydrocephalus may present symptoms similar to tumor, and the latter is not an infrequent cause of effusion into the ventricles. The course of ordinary hydrocephalus is slower and attended with fewer symptoms of irritation than tumor; the symptoms are bilateral usually, and the flexor spasm affects the legs more than the arms. In a case of effusion into one lateral ventricle from closure of the foramen of Monro in a lad seventeen years old, under my care, there were partial hemiplegia and unilateral choked disk. I mistook the case for one of tumor.

Chronic meningitis, especially when attended with great thickening of the membranes, may simulate tumor. It is rare except as a result of alcohol, syphilis, or a tubercular nodule. When due to the first, the convexity is involved and the symptoms are diffuse. They consist of slight or pronounced headache, impaired memory, mental dulness, sometimes of delirium and delusions, and are associated with alcoholic tremor. The optic neuritis partakes of the character of primary optic-nerve atrophy, rather than of choked disk. The history may aid, but it must not be forgotten that tumor of the brain, especially of the syphilitic or tubercular variety, is common in alcoholic subjects. A syphilitic meningitis or a nodular tuberculous condition of the meninges attended with a pronounced thickening of the membranes to such an extent as to give rise to distinct focal symptoms is clinically indistinguishable from an intracranial tumor.

Paretic dementia, on account of the convulsions, paresis, mental failure, and slow speech, may at first suggest tumor of the brain affecting the left frontal lobe near the motor speech centre. In both optic neuritis may be absent, and hesitancy of speech and a peculiar tremor of the lips and face muscles may be present. I have a case of tumor of

the brain under my care at present in which marked tremor of the lips, and sometimes of the face muscles, takes place in attempts to pronounce certain words; but the mental slowness rather than failure, the presence of agraphia, headache, and distinct tenderness on percussion over a small area just above and posterior to Broca's convolution, make the diagnosis easy, especially when the history, which is quite significant, is taken into account. One needs only to be familiar with the delusions of grandeur, the distinct mental failure, and other symptoms common to paretic dementia to prevent confounding it with tumor of the brain; and the same may be said of the other forms of insanity, especially mania.

Multiple sclerosis is attended with a bilateral jerky inco-ordination and frequently with headache, but choked disks and vomiting are absent. In the rare cases of tumor of the brain in which jerky inco-ordination has been present this symptom has always been unilateral, and the other symptoms of an intracranial growth have been quite distinct.

Diffuse sclerosis presents irregular symptoms of the character of hysteria and paretic dementia when it is limited to the cerebrum, but when the process involves the cerebellum the staggering gait, with a tendency to fall in certain directions, may be very pronounced, and suggest a growth in this portion of the brain. The absence of severe headache, optic neuritis, and vomiting, with these symptoms, will be decidedly against the presence of a tumor affecting the middle peduncles or the middle lobe of the cerebellum.

Chronic abscess of the brain occasionally presents symptoms that are very difficult, and sometimes impossible, to distinguish from those of a cerebral growth. A cause for the intracranial suppuration, such as otorrhœa, pus in a distant portion of the body, or traumatism, is often found, yet all the usual causes of abscess of the brain may exist and the patient die of tumor of the brain.[1] Chronic abscess is commonly situated in the frontal, temporo-sphenoidal, or occipital lobe, does not invade the cortex except by secondary softening, and in consequence is less frequently attended by focal symptoms than tumor. Abscess may remain more or less completely latent, with normal or more commonly subnormal temperature for months or years, and is followed by a terminal period during which chills and fever are common. A cerebral growth, on the other hand, is usually progressive. Marked swelling of the optic disks and great impairment of sight are rare in the former, while in the latter they are not infrequent. Rather rapid paralysis, taking place in one group of muscles after another, would be in favor of abscess. A steady but gradual increase of symptoms of organic disease of the brain, with the development of one nerve-root symptom after another, is in favor of tumor; so are prolonged periods during which the symptoms make but little progress.

If one is familiar with the charactistic symptoms of posterior spinal sclerosis, there will be no danger of mistaking tumor of the corpora quadrigemina or cerebellum for it on account of the presence of ataxia in each.

Evidence of the Location of Tumor.—If the conclusion is reached that a tumor is present, the next question to determine is its seat.

[1] Eskridge: "Diagnosis of Chronic Abscess of the Brain," *Amer. Journ. of the Med. Sci.*, Aug. and Sept., 1896.

Notwithstanding that it will necessitate a repetition of much that has already been considered under the head of Cerebral Localization (p. 293), it will make this article more complete and enable the reader to find readily the desired information if the principal symptoms produced by a tumor in the different regions of the brain are given here in a condensed form.

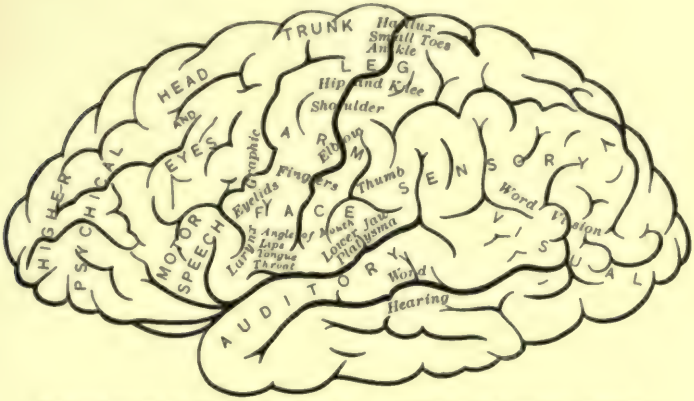
Tumors in the premotor region of the frontal lobe are as frequently localized by negative as by positive focal symptoms. The general symptoms of tumor of the brain, especially headache, vomiting, and optic neuritis, may be present, but the vomiting is less constant than in tumor in the posterior portion of the brain, and the headache is rarely so agonizing. In tumor of this portion of the brain I have found choked disk absent more frequently than present. The mental symptoms are usually the most constant. These vary in different cases. The patients may be apathetic, disregard the ordinary proprieties of life, become filthy and partially demented as the disease progresses; some exhibit a childishness foreign to their nature, and talk much of trivial things, especially when these relate to themselves; others are irritable, impatient, and at times may show a maniacal tendency; while nearly all manifest a lessened power of sustained mental attention and concentration, with absent-mindedness and lack of judgment. Amnesia is rarely complete unless both frontal lobes are involved. From the mental and general symptoms it is impossible to determine on which side of the brain the growth is located. When the tumor is situated in the posterior portion of the lobe or irritates the anterior border of the motor region by pressure or otherwise, definite localizing symptoms may develop, consisting of weakness in one limb or one side of the face, and often of spasm of the affected muscles if the cortex is affected. If the tumor affects the third frontal convolution or the foot of the second frontal on the left side in right-handed persons, or on the right side in left-handed individuals, an incomplete form of motor aphasia and agraphia will be observed. By a careful study of the initial motor symptoms and their gradual increase as the disease progresses in a case of tumor of the brain situated either in the anterior temporo-sphenoidal region or in the posterior portion of the frontal lobe, the direction from which the pressure and irritation come may be determined and the growth thus localized. The tumor may be situated at the lower part of the lobe, and cause unilateral disturbance or loss of smell by pressure on the olfactory bulb, or unilateral affection of sight by pressing upon one optic nerve, or temporal or horizontal hemianopsia by involving the chiasma. Bruns in 1892[1] pointed out that lesions of the frontal lobe are sometimes attended by ataxia of the cerebellar type. Starr[2] gives Bruns' differential points between frontal and cerebellar ataxia as follows: "Frontal ataxia is often associated with hemiparesis or with monoparesis; there is usually a localized tenderness to percussion or to pressure over the frontal region; optic neuritis occurs late in the course of the disease, and mental dulness is an early symptom. In cerebellar ataxia there is rarely hemiplegia; there is no tenderness over the frontal region; optic neuritis

[1] *Deut. med. Woch.*, 1892.

[2] M. Allen Starr: "A Contribution to Brain Surgery, with Special Reference to Brain Tumors," *Med. Rec.*, Feb., 1, 1896, p. 147.

with hemorrhage in the retina occurs very early in the course of the disease and is attended by blindness, and other symptoms of brain tumor, such as vertigo, vomiting, and slowness of pulse, develop early, mental symptoms, such as dulness and apathy, occur late." Notwithstanding the observations of Bruns have been confirmed by a few other investigators, it would be well to make a careful microscopic examination of the tubercular quadrigemina and cerebellum in all cases of gross

FIG. 57.

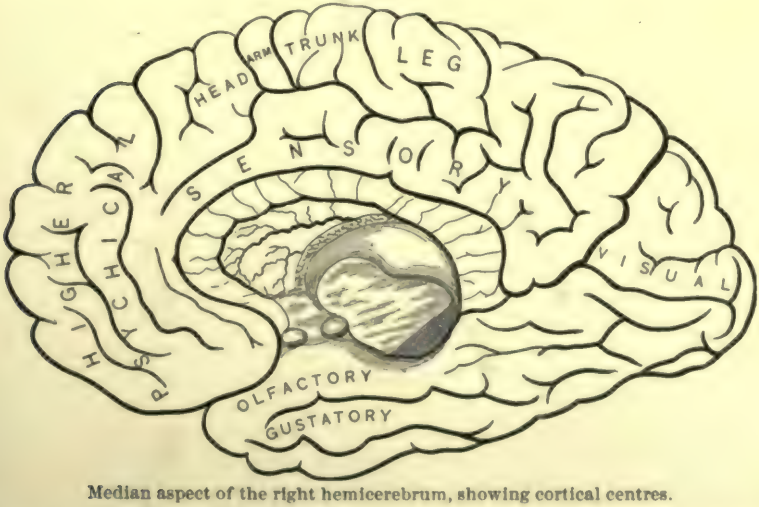


Lateral aspect of the left hemicerebrum, showing the cortical centres.

lesion of the frontal lobes in which ataxia of the cerebellar type has been present.

Tumors in the motor region are usually attended with symptoms suf-

FIG. 58.



Median aspect of the right hemicerebrum, showing cortical centres.

ficiently definite to render their more or less exact localization possible. Figs. 57 and 58 illustrate the cortical motor areas both on the external

and median surfaces of the brain. So far as I know, lesions in the cortical motor area on the median surface of the hemisphere, and limited to this region, do not give rise to very definite localizing symptoms in man. I have carefully observed two cases of tumor involving this region, and obtained an autopsy in each. In one the tumor, about one inch in diameter, was located in the supposed arm and trunk centres. The symptoms were optic neuritis, headache, mental weakness amounting almost to dementia, and very slight muscular weakness, with a little ataxic movement of the opposite side. In the other the growth was about one and a half inches in diameter and situated in the paracentral lobule. The symptoms were optic neuritis, agonizing headache, and unsteadiness of gait, without apparent muscular weakness on either side of the body. When the tumor involves the motor region of the external surface the symptoms are fairly uniform and usually correspond to the seat of the lesion. It should be borne in mind that the early symptoms of a growth in the cortical motor region are often the most reliable for localizing purposes, because the pressure and irritative symptoms produced by extension of the lesion may mask the earlier symptoms. Fig. 57, *L* (leg), *A* (arm), and *F* (face), shows the seat of the lesion when the symptoms are first manifested in leg, arm, face, and tongue, respectively. When the extreme lower portion of the ascending frontal convolution is the seat of a tumor, the tongue and lip muscles will be affected, while most of the face muscles may escape, but if the lesion is on the left side, there will be a partial degree of motor aphasia and awkwardness in writing. It is probable that a small growth involving the foot of the second frontal convolution on the left side will be attended by agraphia [1] and a condition known as paramotor aphasia, especially when the irritative lesion extends to the white fibres below the cortex. When a cortical centre of one limb is first affected, the focal symptoms may at first be limited to the distal portion of the extremity. If the tumor begins in the cortex, convulsive symptoms, usually limited, precede the weakness and paralysis, but when the growth is situated in the subcortical motor area and invades the cortex, weakness and paralysis more commonly precede the convulsive movements.[2] It is important to study the initial phenomena and the order in which one group of muscles after another is involved by the convulsion, as these afford a most important aid in localizing the primary seat of the lesion, especially early in the history of the disease. In some cases, probably those in which the irritation is limited to the cortex, the seizure may for a time consist of pain, numb or tingling sensations limited to the distal portion of an extremity, or these may immediately precede a convulsive movement which always begins in the part in which the sensory phenomena are felt. They have the same localizing value as the muscular spasm. If the spasm begins in the face, the arm will be affected before the leg; if it begins in the leg, the arm will be affected before the face. It is not uncommon to have slight twitching in the muscles of the face at the same time that the hand is affected. The convulsive movements usually cease last in the part first affected, and the muscles that are the seat of the initial spasm are often weak or paralyzed for several minutes after the convulsion ceases.

[1] Anorthographia (inability to write from inability to spell).
[2] Hughlings-Jackson.

When the convulsion begins in the lower face on either side, but more commonly on the right, temporary motor aphasia often follows the attack. Consciousness may not be lost unless the convulsion becomes general. Some objective sensory disturbance is not infrequently found in the distal portion of the affected limb when the lesion is a tumor in the motor cortex. It is usually slight and most pronounced when the region posterior to the Rolandic fissure is involved. Dana seems to be correct in the statement that the sense of localization is most affected of all the sensory phenomena when the sensory disturbance is only slight. The muscular weakness and paralysis come on gradually as a rule, and are often accompanied by contracture and marked increase of the myotatic irritability. In those cases in which muscular contracture is well developed very decided atrophy may be quite marked in some of the muscles of the distal portion of the extremity. It is more than can be explained by disuse, and it nearly all passes away on removal of the growth, although the paralysis remains. It is probably due to an irritative lesion. Unlike the atrophy of spinal or nerve-trunk origin, the electrical reactions of the most wasted muscles are normal.

Tumors of the parietal region, if small, may give rise to no focal symptoms, but in some cases the symptoms from tumor in this region are exceedingly complicated and interesting. Muscular sense is thought to be affected when the supramarginal convolution is involved on either side. On the left side in right-handed persons and on the right side in left-handed individuals a growth affecting the angular gyrus and inferior parietal lobule produces word-blindness. When a large tumor is situated in the lower portion of the parietal lobe marked sensory disturbance may be observed on the opposite side of the body, and if it extends far back, bilateral homonymous hemianopsia will be observed. It is probable that the latter symptom is due to involvement of the optic radiations. The view held by some that ptosis and "crossed amblyopia" are caused by a lesion in the lower parietal lobule seems to have been negatived by a case of large tumor recently observed by me in this region.

Tumors of the occipital lobe affecting the cuneus or the optic radiations will cause bilateral homonymous hemianopsia, the blind fields being on the side opposite to that of the lesion. Mind-blindness, a condition in which an object may be seen, but not recognized by the sense of sight, has been observed in connection with growths in this region of the brain on the left side.

Tumors of the temporo-sphenoidal lobe may be attended with no definite symptoms, especially if the growths are small and not infiltrating. If the extreme anterior portion of this lobe is affected, disturbances in smell and taste may occur, either in the form of an aura of a gustatory or olfactory character or loss of one of these senses, taste phenomena being noted on the side opposite to that of the tumor, and smell on the same side. Word-deafness will result from a tumor in the posterior portion of the first and second temporal convolutions—on the left side in right-handed persons and on the right side in left-handed ones. If unilateral deafness occurs from a lesion in one lobe, it is probably of brief duration. Adjacent regions are often affected by large tumors in the temporo-sphenoidal lobe, and from the

character of the additional symptoms caused by the invasion of centres outside of this lobe the exact location of the growth may be determined.

Tumors of the corpus callosum seem to be attended by no definite symptoms by which their exact location can be determined. In most cases that have been reported all the symptoms could be accounted for on the theory of irritation of adjacent structures and the general pressure symptoms from the presence of the tumor. Besides the general symptoms of tumor of the brain, the patient exhibits hemiparesis or bilateral weakness, the loss of power being more pronounced on one side than on the other, and usually most intense in the legs, with or without unsteadiness in walking, loss of mental power, stupidity, and finally coma. The ocular-motor and the bulbar cranial nerves are not affected except in rare instances. It is probable that a tumor in the posterior portion of the corpus callosum is attended with greater unsteadiness of gait than one in the anterior portion. In one in the former situation observed by me there was no absolute paralysis, but bilateral weakness with late rigidity of the muscles of the trunk and limbs, great mental weakness, pronounced choked disks, very slight and infrequent headache, occasional convulsions and vomiting, and throughout the active course of the disease a gait similar to that caused by disease of the cerebellum or corpora quadrigemina, with a marked tendency of the body to plunge backward when the patient was placed on his feet, were observed. Tumors of this region with ataxia are distinguished from those of the corpora quadrigemina by the absence of ocular motor symptoms until very late in the course of the disease, and from growths in the cerebellum from the cranial nerves of the bulb escaping.

Tumors of the great ganglia at the base produce no constant and reliable symptoms unless the internal capsule is affected. The symptoms caused by tumors in these ganglia differ from those resulting from vascular lesions in coming on more gradually and in being attended with more pronounced irritative phenomena. If the motor portion of the internal capsule is affected, the sensory escaping, there will be more or less complete hemiplegia of the opposite side, including the lower portion of the face. If the lesion is on the left side, there will be transient defect of speech. On account of the position of the leg, arm, and face fibres in the capsule the tumor may invade one set before the other, and produce a monoplegia, but usually paralysis of one limb from a tumor in this region is attended by a paretic or spastic condition of the muscles of the other limb on the same side. If the posterior portion of the internal capsule is affected by the tumor, hemianæsthesia, partial or complete, will be found on the opposite or paralyzed side. Complete hemianæsthesia from tumor in the basal ganglia practically does not occur without motor paralysis. When the hemianæsthesia is complete, all the special senses may be lessened or lost on the anæsthetic side, the visual defect being homonymous hemianopsia with the blind fields on the side corresponding to the affected side of the body.

It is probable that the hemianopsia is not due to destruction of the sensory fibres in the internal capsule, but to pressure on the visual fibres in the optic radiations or to involvement of the primary visual ganglion, the external geniculate body. The visual fibres, according to Henschen,

do not pass through the internal capsule, but in the case of a tumor in this region they are probably injured in their winding course around the optic thalamus.[1]

Athetoid movements and marked inco-ordination, chiefly affecting the hand, have been observed in lesions of the thalamus. A tremor similar in character to that of multiple sclerosis has occurred from tumor in the thalamus. It is always unilateral, however.

Tumors involving the corpora quadrigemina are usually attended with well-marked general symptoms of tumor of the brain, especially headache, choked disk, vomiting, and the focal symptoms, ataxia and incomplete opthalmoplegia. The ataxia is similar to that observed in lesions of the middle lobe of the cerebellum. According to the observation of Bruns, the ophthalmoplegia will precede ataxia when the lesion is in the corpora quadrigemina, but the latter will precede the former when the tumor is in the cerebellum.[2]

Tumors of the crus produce "crossed paralysis," hemiplegia on the opposite side of the body, including the limbs and lower side of the face, and paralysis of the third nerve on the side of the tumor. If the fibres on the upper or posterior portion of the crus are involved, sensation will be affected on the hemiplegic side. If the optic tract is seriously affected, hemianopsia will be present, and the pupils will not react to light thrown into the eyes from the side of the blind fields—the hemiopic pupillary reflex of Wernicke.

Tumors of the pons, as a rule, produce very striking symptoms. These vary in individual cases according to the size and seat of the growth. Limited vascular lesions in this region after the irritative symptoms have subsided can be more accurately located than tumors, because the latter are attended by greater irritation throughout their course than the former. A tumor may be situated without the pons and cause decided pontile symptoms from pressure. In such cases the trunks of some of the cranial nerves will be irritated before the pons is involved, and the symptoms will vary accordingly. The pons is not infrequently displaced to one side by a unilateral growth in its lower portion. Such a displacement may give rise to indirect symptoms that are misleading if they are mistaken for the direct symptoms due to the presence of the growth.[3] If the tumor is situated in the upper portion of the pons on one side, the third cranial nerve will probably be paralyzed on the side of the lesion, and the body and lower portion of the face on the opposite side will be weak. During the stage of irritation to the sixth nerve the eyes will spasmodically deviate toward the side of the lesion, but when this nerve is paralyzed the power of conjugate deviation of the eyes toward the side of the lesion is lost. If the tumor is not large, the fifth nerve will be affected on the side opposite to that of the lesion, but, owing to the diffuse character of the irritative symptoms of pontile tumors, the fifth nerve may be involved on both sides even when the growth is situated high up in the pons.[4] The fibres of the inner two thirds of the reticular formation are often irritated or partially destroyed by a unilateral

[1] Cooke: *Lancet*, May 28, 1892. [2] *Ann. Univ. Med. Sci.*, 1895, vol. ii. A–40.
[3] F. W. Langdon; *Brain*, Part lxii., 1895, p. 551.
[4] Limited vascular lesions in this region probably affect the fifth nerve on the opposite side only.

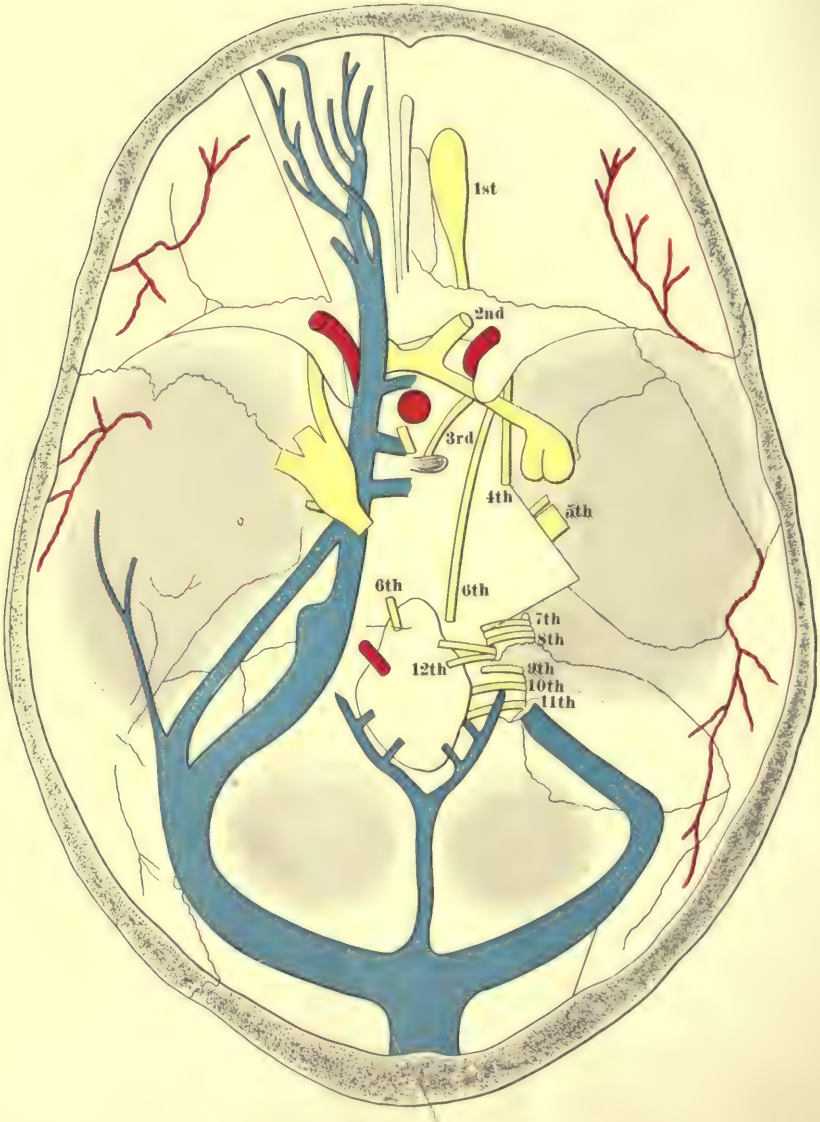
growth of the pons, and sensory symptoms in consequence will be observed in the limbs and trunks on the side corresponding to the motor paralysis. The symptoms from a unilateral tumor in the upper portion of the pons would probably be external strabismus, dilatation of the pupils, ptosis, sometimes cloudiness and ulceration of the cornea; pain, with hyperæsthesia and anæsthesia in the region of the distribution of the fifth nerve on the side corresponding to that of the tumor; and hemiplegia and hemianæsthesia of the opposite side of the body and face, often in the form of dissociation of sensory symptoms (loss of pain and temperature sensations, tactile preserved), and loss of conjugate movement of the eyes toward the side of the lesion. The fourth and the motor branch of the fifth cranial nerves might, and probably would, be affected. A unilateral tumor involving the lower half of the pons would cause crossed motor and sensory paralysis, the face, both the lower and upper portions, for motion on the same side. and the body and limbs on the opposite side to that of the lesion. Marked trophic disturbances would probably occur in the region of distribution of the affected fifth nerve, both in its motor and sensory branches. The fifth, sixth, seventh, and eighth cranial nerves may be involved. If the lesion extends to the lower portion of the pons, other cranial nerves may be affected, and articulation, deglutition, and respiration may be impaired, but these symptoms from unilateral tumors of the pons rarely become conspicuous until late in the disease.

Glycosuria and albuminuria have been observed from tumors in this region of the brain stem. Giddiness is often pronounced, and vomiting may be troublesome when the middle peduncle of the pons is involved. It is not infrequent for tumors to affect both sides of the pons, and then the symptoms will be bilateral, but they usually remain more pronounced on one side than on the other. The knee jerks are as frequently absent as present, and are extremely variable, sometimes increased with clonus and at others nearly normal or absent.

Tumors of the medulla usually give rise to bilateral symptoms, and on account of the numerous cranial nerves affected these are similar to those of bulbar paralysis, except that in lesions produced by tumors sensory as well as motor disturbances occur. Projectile vomiting, polyuria, glycosuria, retraction of the head, and local sweating may be occasionally observed.

Tumors of the cerebellum usually cause well-marked general symptoms of intracranial growth, such as choked disk, headache, vomiting, and, not infrequently, dizziness. General convulsions occur in a number of cases. Localizing symptoms may be present or entirely absent, depending upon the seat of the tumor and the structures irritated, compressed, or destroyed by it. Unless the middle lobe or the peduncles of the cerebellum are directly or indirectly interfered with, all symptoms pointing to the exact seat of the tumor are absent, so that a growth situated in one of the hemispheres, and sufficiently small so as not to compress the middle lobe, is unattended by any special localizing symptoms. The most pronounced and constant localizing symptoms of tumor of the middle lobe are the disturbed muscular movements, limited largely to the legs, but observed to a less extent in the trunk. The gait is reeling, the patient has a subjective sensation of insecurity in

PLATE VI.



The Cerebral Fossæ, Showing the Position of the Cranial Nerves at the Base of the Brain.

the upright position, and walks with short steps and feet widely separated. In standing, especially if the feet are held near together, the body sways to and fro, and in some cases there is a tendency to fall, apparently by forced movements in one direction, whenever the equilibrium is lost. Forced movement backward is said to be less frequent than forward or to one side. The gait is uncertain or zigzag, and very much resembles that of a drunken person, and has received the name of cerebellar titubation. When the patient is in a recumbent position the legs are moved fairly well, thus differing in this respect from the ataxia of posterior spinal sclerosis. The knee jerks may be normal, increased, or absent. They are often variable, and rarely present continuously unless the tumor is situated well forward and compresses the pyramidal tracts. A tremor is occasionally observed in the arms. When the tumor invades the peduncles or is situated in the anterior portion of the middle lobe cranial-nerve symptoms are usually present. Forced movement or staggering toward one side seems to be common in tumors of one middle peduncle, the turning of the body being more frequent from the side corresponding to the seat of the growth than toward it; but the cranial-nerve symptoms in such cases correspond in situation to the side of the tumor. Nystagmus occurs from some irritative lesions of the cerebellum, and is probably not infrequent in tumors of the superior peduncles. In tumors of the latter situation ophthalmoplegic symptoms are common. When the middle peduncles are the seat of the growth, the sixth, seventh, and eighth cranial nerves are not infrequently affected. Tumors of one peduncle usually involve one side of the middle lobe of the cerebellum. If the tumor is in the upper and anterior portion of the middle lobe, the symptoms are bilateral and pontile, and if it is principally on the under surface of this lobe, the medulla is affected, presenting symptoms of pseudo-bulbar paralysis, with sensory or motor phenomena and disturbance in articulation, respiration, deglutition, and the heart's action, together with polyuria and glycosuria in some cases. Starr states that it is impossible to differentiate a tumor of the superior peduncle of the cerebellum from one of the corpora quadrigemina, as both are attended by nystagmus and paralysis of the ocular motor muscles.[1] Bruns believes the ophthalmoplegia will precede the ataxia when the tumor is in the corpora quadrigemina, and the order of the appearance of these two symptoms will be reversed when the growth is in the cerebellum.[2]

Plate VI. shows the relation and position of the cranial nerves in the different fossæ. Tumors of the base strictly limited to the anterior fossa would only affect the olfactory nerves, but such growths not infrequently extend backward and compress the optic nerve or even the chiasma, producing unilateral loss of sight or bitemporal hemianopsia. Mental symptoms may result from compression of the frontal lobes. Tumors of the middle fossa, if situated near the sphenoidal fissure, may paralyze all the motor nerves of one eye, and compress the first division of the fifth nerve, causing ophthalmoplegia, anæsthesia, and pain in the region of the distribution of this division of the trigeminal nerve, together with trophic changes in the eye. In one such case recently observed by me

[1] *Nervous Diseases by American Authors*, p. 500.
[2] *Ann. Univ. Med. Sci.*, 1895, vol. ii. A-40.

the optic disk remained normal, and sight was only impaired from the trophic changes in the cornea up to the time of the patient's death, which occurred from a large intracranial hemorrhage in the opposite side of the brain. The Gasserian ganglion and all the divisions of the fifth nerve are favorably situated to be compressed by tumors in the middle fossa. Tumors of the posterior fossa injure the cranial nerves, pons, and medulla, and produce many of the symptoms of tumors in the medulla or pons—with these differences, that tumors in this fossa affect the nerves before they do the pons or medulla; in paralysis of the sixth nerve from injury to its trunk the conjugate fibres of the internal rectus of the other eye are not affected, as in nuclear paralysis of this nerve; and the seventh and eighth nerves are usually affected by the same lesion, as they lie near together at the base.

The DIAGNOSIS of multiple tumors is frequently impossible, as the growth that gives rise to the localizing symptoms, when such symptoms are present, is often the only one suspected during life. In cases in which no localizing symptoms are observed it is beyond human skill even to suspect more than one tumor, no matter how numerous and well marked are the general symptoms of an intracerebral growth. It is only in the few instances in which the two distinct sets of localizing symptoms develop that multiple tumors can be diagnosticated.

After satisfying ourselves of the seat of the tumor, the next question to answer is, What is its nature? In only a few cases is it possible to determine with any degree of certainty the exact nature of the growth. The presence of a growth in some other portion of the body, or the history of the removal of one, the pathological character of which is known to be cancerous or sarcomatous, in a person suffering from an intracranial tumor would be very strong evidence of the secondary growth being of the same nature as the primary one, but it must not be accepted as conclusive.[1]

Evidences of syphilis or tuberculosis in persons suffering from tumors of the brain suggest the nature of these growths. In adults the evidences of tuberculosis in other portions of the body are usually present when the cerebral growth is tubercular, but in children these are often absent.[2] Syphilitic cerebral growths are rarely if ever due to inherited syphilis.[3] It should be borne in mind that the secondary symptoms of syphilis have been frequently absent in persons who are the subjects of cerebral syphilis. The most common cerebral growth in children is the tubercular. The cerebellum is rarely the seat of syphilitic growth, but the tubercular and the gliomata, especially in children, commonly occur here. Growths in the cortex are usually syphilitic, tubercular, or sarcomatous: the last is usually on the surface of the brain. Gliomata and sarcomata frequently occur in the centrum ovale, and may subsequently involve the cortex. A very slowly growing tumor in the centrum ovale is probably a glioma

[1] In a report of "Three Cases of Abscess of the Brain," made by me, *Trans. of the Coll. of Phys.*, Phila., 3d series, vol. vi., 1883, p. 77, is described one which occurred in a patient beyond middle life who had had a cancerous tumor removed from the breast a year before the development of cerebral suppuration.

[2] Gowers: *q. v.*

[3] Bramwell: *q. v.*

or an encapsulated sarcoma. The effect of treatment, especially the antisyphilitic, is of little value in determining the nature of the growth unless the improvement is very great and can be maintained by such medication. It is probable that the symptoms in most forms of cerebral tumors in the early stages of these growths will be favorably modified by antisyphilitic treatment. I have seen marked improvement follow such medication in the case of cysts, sarcomata, and gliomata. Syphilitic tumors in their late stages will not respond much to the so-called specific agents.

Prognosis.—With rare exceptions the prognosis is absolutely hopeless in all forms of intracranial tumors, except in the tubercular and syphilitic varieties, provided surgical interference is of no avail. In children tubercular growths are more likely to become arrested than in the adult. If the tumor has attained much size, nothing more than a temporary arrest can be hoped for except in rare cases, and even the smaller growths rarely yield to treatment, although occasionally an apparent cure does take place. I have seen one such. A recurrence of the symptoms is always to be feared in such cases. While the syphilitic growths are the only ones that are probably amenable to direct medication, a recurrence of the tumor or the ravages of syphilis in another form are to be feared in all cases. It should be remembered that while the growth may be entirely absorbed, the lesions caused by its presence in the brain are non-specific and are uninfluenced by antisyphilitic medication; therefore, in cases in which paralysis has persisted for some time the removal of the tumor does not result in the cure of the secondary lesions. The earlier proper treatment is instituted in syphilitic tumors of the brain, and the more vigorously it is pushed, the better the prognosis. It is in the early stages of these lesions that an absolute cure may often be predicted, so far as the removal of the growth is concerned.

Treatment.—While in the vast majority of cases little can be done for the permanent relief of the patient, yet by judicious management much may be accomplished to make life less intolerable. We should guard against taking too gloomy a view of the prognosis, and resting satisfied as soon as the diagnosis of the tumor has been made and its location determined. The treatment should be different in the early stages of intracranial growths from that which must be adopted after they have attained considerable size and resulted in much damage to the brain. In the early course of the disease, unless we can positively say that the cerebral growth is not syphilitic—which is rarely possible—potassium iodide or mercury, or both, should be employed. The greater the evidence of the activity of the morbid process in syphilitic lesions the greater are the probabilities that mercury will accomplish more than potassium iodide, and the reverse has, in my experience, seemed to hold good, even in cases in which the initial lesion dates back only one or two years. If mercury is employed, the blue ointment should be thoroughly rubbed into the skin night and morning, and the parts kept covered by oiled silk or flannel. This is one of the most effective methods of bringing the system under the influence of the medicine. In giving mercury for constitutional effect the gums should be well rubbed with a stiff brush night and morning to prevent saliva-

tion occurring before the object of the treatment has been accomplished. If potassium iodide is employed, the dose to begin with should be about ten grains thrice daily in from four to six ounces of water an hour after meals. The practice of giving this agent before meals, in my experience, cannot be too severely condemned. It is more rapidly absorbed if given when the stomach is empty, but, unfortunately, gastritis is soon set up, and the patient in many cases becomes unable to take the drug. The dose of potassium iodide should be increased from one to five grains daily until the symptoms begin to yield or the tolerance of the patient is reached. If, however, the patient does not manifest some improvement in two weeks from one of these drugs pushed to the point of tolerance, it should be discontinued and the other tried. If neither alone gives the desired relief, they should both be employed at the same time.[1] Six weeks will be sufficient time in which to determine whether any good can be accomplished by antisyphilitic agents, and if at the end of that time no improvement has occurred, it is useless to subject the patient to further treatment of this kind. If benefit does occur from antisyphilitic agents, are we to conclude that the tumor is specific in character? By no means. In many forms of tumor the condition of the patient temporarily improves under the use of mercury or potassium iodide, or from a combination of both. If improvement should cease while giving antisyphilitic treatment, the quantity of the medicine should be increased for a week or two, and if there is no regression of the symptoms, it is fair to conclude that the growth is either not specific or, if it is, further treatment will accomplish nothing. In syphilitic subjects it may be necessary to improve the nutrition when anæmia is well marked before antisyphilitic medication will accomplish anything. In the late stages of brain tumor it is, to say the least, cruel to subject a patient to a prolonged course of antisyphilitic treatment. Even if there is every probability of the cerebral growth being syphilitic, potassium iodide and mercury should not be continued if improvement is not apparent soon after beginning these agents.

Persons suffering from tubercular growths do best under a generous and nutritious diet, general tonics consisting of cod-liver oil, iron, quinine, and arsenic, together with plenty of fresh air. Arsenic has been recommended in sarcomata of the brain.

In all classes of cases relief from special symptoms is often demanded. These are generally headache, vomiting, and convulsions. Headache may frequently be lessened or prevented by keeping the bowels open freely each day, by paying strict attention to the digestive organs, and by avoiding everything possible likely to cause an increased supply of blood to the brain. Cold to the head in the form of an ice poultice (equal parts of bran and pounded ice), with a mustard plaster at the nape of the neck, often relieves an annoying headache. Local abstraction of blood from the head by means of leeches gives relief in some cases. Cannabis indica, either alone or in combination with other agents, will often make the pain bearable. To be effective it must be given so as to obtain its physiological effects. Even when the

[1] In cases in which the initial lesion dates back several years, but in which there is more or less evidence of active irritative processes going on in the brain, mercury and potassium iodide should be employed from the first.

pain is great, ten to fifteen grains of antipyrine, phenacetine, antifebrine, and $\frac{1}{4}$ to $\frac{1}{6}$ grain of the English extract of cannabis indica or ten minims of the normal liquid, will sometimes give relief. The dose may be repeated every three or four hours for several doses, provided there are no contraindications and the precaution is taken to give caffeine citrate as a cardiac stimulant. If it becomes necessary to resort to any of the preparations of opium for the relief of headache, codeine or bimeconate of morphine should be given the preference. Codeine will not be effective in some cases. The bimeconate of morphine, in about the dose of the sulphate, should be employed. As a rule, as little opium or any of its preparations should be given as possible. Vomiting will often yield to cold applied to the head and a mustard plaster placed over the back of the neck and another over the pit of the stomach. Sometimes twenty or thirty grains of chloral hydrate given in a starch-water enema afford prompt relief both from vomiting and convulsions. In severe and intractable cases morphine hypodermically is necessary for the relief of the vomiting as well as of the convulsions.

When and in what cases should surgical interference, either for the removal of the growth or for the relief of pain, be recommended? A tumor situated in the cortex or immediately below it, over the entire upper and lateral regions of the brain, and in portions of the anterior and middle fossæ, is accessible to the surgeon, and it is probable that a growth could be removed with safety to the patient from any portion of this entire area with the possible exception of the cerebellum; and I should not exclude all parts of this were it possible to locate a growth in the posterior portion of the cerebellar hemispheres. If the tumor, supposing it to be syphilitic, or even tubercular, should not yield materially to the medication in six weeks or two months, it should be subjected to an operation for its removal if there are no contraindications. No one would think of endeavoring to remove a carcinoma or a melanotic sarcoma from the most accessible region of the brain if the diagnosis of the nature of the tumor was possible or strongly probable. Encapsulated sarcomata and cysts [1] afford the best prospects for relief by the knife, but one of the most brilliant results that it has been my good fortune to witness in brain surgery followed the removal, by Parkhill, of a large infiltrating glioma from the parietal and Rolandic regions of the brain in the person of a physician conjointly attended by us. If the growth is operable and will not respond to internal medication, it should be removed early to obtain the best possible results. Trephining for the relief of headache and intracranial pressure has been resorted to in a number of instances with apparent benefit. The button of bone should, if possible, be removed from the part that is nearest the seat of the intense pain.

[1] Since writing the above statement two of my patients, from whose brain cysts had been removed, have developed untoward symptoms several months after apparent recovery from the surgical operation. (See also Starr's remarks on cysts of the brain, *Med. Record*, Feb. 1, 1896, p. 147.)

FUNCTIONAL NERVOUS DISORDERS.

FUNCTIONAL NERVOUS DISORDERS.

EPILEPSY.

By FREDERICK PETERSON, M. D.

Synonyms.—Epilepsia; Falling sickness; Fallsucht (Ger.); Morbus sacer, l'Epilepsie (Fr.)

Definition.—Epilepsy may be defined as a chronic disorder of the cerebral cortex, usually functional, sometimes organic, characterized by periodical attacks of loss of consciousness with or without convulsions, and occasionally by mental excitement.

The view commonly entertained at present is that an epileptic or eclamptic attack is a katabolic discharge of nerve centres. The convulsions of infancy and the occasional convulsions of adult life are to be looked upon as a symptom of a great variety of pathological conditions, such as morbid states of the blood or vascular apparatus, trauma or neoplasms, giving rise to direct irritation of the epileptogenetic centres, or reflex causes acting indirectly. The periodical recurrence of such seizures constitutes epilepsy. Hence epilepsy should perhaps not be considered as in itself a disease, but rather as a symptom, and the more closely we look into the pathogeny of our cases of epilepsy the more restricted grows the class which we designate as idiopathic or essential epilepsy.

Idiopathic is a qualification applied to epilepsy in which the pathological basis is as yet unknown, and it still includes the majority of cases, for, although the syndrome of epilepsy has been described as a disease from the most ancient times, we are not yet very near a solution of its real pathology. We have been able to separate a small group of organic and toxæmic cases, but the essential or idiopathic form must be still regarded as a functional disorder until our untiring pathologists of the present day shall discover for us its true cause.

Epilepsy is a very common nervous disease, and it is estimated that it affects 1 in 500 persons.

Etiology.—Heredity plays a very important rôle in epilepsy. In an analysis of 1450 cases Gowers found a family history of epilepsy in two thirds, of insanity in one third, and of both in one tenth of the cases. There are other neuropathic conditions in the ancestors which may be forerunners of epilepsy in the descendants, such as neurasthenia, hysteria, and even chorea. Where there is a neuropathic basis it would seem that the females are more apt to suffer than the males. As many as 14 cases

of epilepsy in four generations of one family have been reported. An hereditary relation between phthisical or gouty diatheses and epilepsy, though once credited, has not been sustained. It is, however, different with alcoholism and syphilis. Alcoholic inebriety not only often leads to epilepsy in the individual, but, aside from that, is a frequent cause of the disease in the immediate descendants of an inebriate parent. Inherited syphilis is a probably not infrequent factor in the etiology of epilepsy. When instability of nervous organization is prepotent in a family consanguineous marriage may give rise to epilepsy in the children, as well as to other nervous or mental disorders.

It is a curious fact that while in infantile convulsions males are more frequently affected than females, in epilepsy the reverse is the case, afflicted females being slightly in excess of males.

As regards age of onset, all authors agree that by far the majority of cases of epilepsy begin before the age of twenty years. Gowers states that one eighth of his cases began during the first three years of life, and nearly one half between the ages of ten and twenty (especially around the age of puberty). The number gradually decreases after the age of twenty, though the disease may manifest itself in old age (over seventy). Late epilepsy, or epilepsia tarda, is the name given to epilepsy beginning after the age of forty.

Where there is an hereditary convulsive tendency in an individual one convulsive seizure seems in a manner to prepare the epileptogenetic centres for a repetition of an attack, until a true habit of discharge is acquired. The first convulsive seizure in an epileptic series is generally ascribed to some immediately exciting cause. It is doubtless true that the more carefully we study the etiology of our cases the more we come to separate the essential or idiopathic class from that in which the real cause is discovered. At the same time, the immediate causes are generally of less importance than the remote hereditary predisposition, particularly in the idiopathic class. An exciting cause will be found in perhaps 40 per cent. of the cases. The influence of infantile eclampsia in inducing epilepsy is considerable, for of cases beginning in infancy some three fourths are ascribed to ordinary infantile convulsions. These infantile convulsions are due to rickets in 30 to 40 per cent. of cases, to febrile and toxæmic causes, to anæmia and exhaustion, and to reflex causes (chiefly dentition and gastro-intestinal irritation). Often infantile convulsions are the result of meningeal hemorrhage also, occurring during labor, and the epilepsy developing from this may exist in conjunction with idiocy or with cerebral palsy, with hemianopsia, with a mere vestige of one of these organic conditions, or without a trace of the involvement of other than epileptogenetic centres.

Mental stress of one kind or another is a common exciting cause of epilepsy, and among such stresses figure largely excitement, anxiety, and fright. The latter is a particularly frequent cause in children and adolescents; which is not to be wondered at when we consider the marked effects of alarm on the nerve centres of the young.

Blows or falls upon the head are productive of epilepsy in two ways: firstly, by the mere effect of concussion in disarranging the delicate nerve centres in those predisposed to the disease; and, secondly, by direct organic lesion of the cerebral cortex (depressed fracture of the skull, neoplasms,

compression, and laceration of cortical tissue). In this connection, too, it is well to bear in mind that organic injury to any part of the cerebral cortex may induce epilepsy. It need not necessarily be of the Rolandic area, for lesions of the anterior or posterior parts of the brain may excite epileptic convulsions by the irradiation of discharge from such parts to the motor areas. Circumscribed injuries to the Rolandic area are apt to cause (but do not necessarily do so) local epilepsy or convulsions beginning locally.

Acute diseases give rise to epilepsy often by actual organic lesion (thrombosis or embolism), but more often probably by the irritation of some toxin in the blood. Among these acute diseases may be mentioned scarlet fever especially, and measles, typhoid fever, and cerebro-spinal meningitis. Aside from the toxin of acute disease, we have also among direct exciting factors in epilepsy the effects of the long-continued action of such poisons as alcohol, lead, antipyrine, and cocaine. Putrefactive or fermentative products in the gastro-intestinal tract, giving rise to auto-intoxication, doubtless induce epilepsy at times.

The rare cases of epilepsy due to diabetes (epilepsia acetonica) and renal disease (often without acute uræmic symptoms) deserve mention here. Inherited syphilis has already been mentioned as a cause; acquired syphilis is rarely a factor except by organic lesion (gumma or meningitis).

Sunstroke is sometimes given as an exciting cause, as are also masturbation, sexual excesses, and menstrual disturbances; but their influence as factors is open to discussion. Uterine and ovarian diseases are extremely doubtful exciting causes of epilepsy.

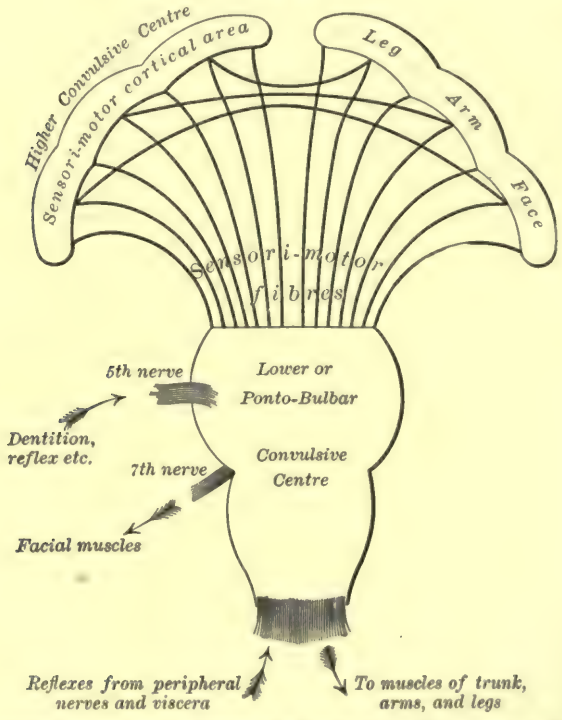
The so-called reflex epilepsies must be rare, for in over 1200 cases I recall but few which could be thus truly designated. In infants and children reflex eclampsia is of course a phenomenon of common observation, and such eclampsia may be a forerunner of true epilepsy. I have no doubt that there are, however, cases of this disease in which the exciting cause is of a reflex nature, due perhaps to intestinal worms, to old cicatrices, to genital disorders, to naso-pharyngeal affections, and to eye or ear troubles, for most authors mention these as possible factors.

There are causes which at times serve to induce individual seizures in a person afflicted with epilepsy, and among such are digestive disturbances due to overeating or the eating of improper food; emotional excitement; staying in an ill-ventilated and crowded room; sudden interruption of some course of treatment (bromides); and disturbances of the general health of the patient.

PATHOGENESIS.—I have already referred to the fact that epilepsy is probably a katabolic discharge of nerve-cells in the cortex of the brain. Jackson has taught that there are three levels from which such discharges may occur—from the cells of the ponto-bulbar region; from the Rolandic area; and from a level (purely speculative on his part) which he believes to exist in the frontal lobes, and to be the supreme controlling centre of sensory-motor functions. Whatever may be the merits of this author's interesting and suggestive theory, I feel that from the practical point of view we must consider epileptic convulsions as having their seat of origin in the cortex. The primary seat of the discharge which ultimately affects the motor areas may be an irritation in any part of the cortex,

whence it may extend by irradiation to the Rolandic areas. In psychic epilepsy probably it is the frontal cortex which is implicated. In cases of convulsive seizures, preceded by auræ of various kinds, doubtless the original site of the discharge is in the cortical centres for the functions represented in the particular premonitions in each case. Some authors are inclined to believe in a convulsive centre in the floor of the fourth ventricle. Jackson considers laryngismus stridulus a convulsive discharge from the ponto-bulbar region, as also the respiratory fits induced in animals by asphyxia, the attacks produced by convulsant poisons (urea, camphor, curare, absinthe, nitrous oxide, and the like), and such

FIG. 59.



Schematic representation of the two convulsive centres, one cortical, the other ponto-bulbar, which may be acted upon by direct lesions or by toxic blood states; or occasionally by reflex irritations conveyed along sensory fibres from distant parts of the body.

as result from injuries to the sciatic nerve and spinal cord in guinea-pigs. The following diagram (Fig. 59), from my article on "Infantile Convulsions" in the *American Text-book of Diseases of Children* (p. 742), illustrates the two convulsive centres—the ponto-bulbar and the cortical—together with their relations and connections, and shows how direct lesions may affect them, how reflex stimuli may be conveyed to them, and how they may be irritated by vascular or blood states.

Assuming that the cortex contains the epileptogenetic centres, the cause of the irritation inducing the attack in the majority of cases still remains to be discovered. Pathologists are making progress on many

lines of investigation, and already the term idiopathic or essential epilepsy is growing more and more restricted in application. There are auto-toxæmic and toxic conditions; reflex states; organic disease or injury; minute vestiges of old organic lesions (meningeal hemorrhage, etc.). But, leaving out cases of this kind, where we may discover or approximate to the true source of irritation, there is still a vast number of cases in which the real pathogeny remains to be ascertained. Marie has lately hinted that epilepsy may often be of infectious origin. Vasomotor spasm and consequent cerebral anæmia have been adduced, without, however, any very adequate basis, as a primary cause of idiopathic epilepsy. The theory of imperfect nutrition of the nerve cells, and over-action and discharge as an effect of this, is one that appeals strongly to the reason, and perhaps study along this line may be productive of valuable results in the near future.

Pathological Anatomy.—In many cases of general and local epilepsy, even in some cases supposed to be idiopathic epilepsy, real organic lesions have been found. Among these are tumors implicating the cortex, depressed fractures, wounds, circumscribed syphilitic pachymeningitis, localized tubercular meningitis, old atrophies, scleroses, and cysts (in infantile hemiplegic epilepsy), and the like. But the morbid anatomy of genuine idiopathic epilepsy is still an unknown territory. To the naked eye the brain in a case of this kind has every appearance of being normal, and even microscopic investigation has as yet revealed nothing very definite. One of the best of the older pathologists (Meynert) attached importance to sclerosis of the cornua ammonis, but this seems to exist in not more than 6 per cent. of the cases. More recently, further studies in this direction have been made. Féré has reported sclerotic conditions in parts of the cortex and in the olivary bodies analogous to that of the cornua ammonis. Chaslin in an interesting study describes in epileptic brains a condition of diffuse neurogliar sclerosis, a gliosis. Marie, Féré, and Chaslin look upon such gliosis as having great pathological importance in epilepsy, and believe that the sclerosis has its origin in some focal lesion. Bleuler, in order to test the results of Chaslin's studies, examined the brains of 26 chronic epileptics. All were more or less demented. He found over the whole cortex the neurogliar proliferation described by Chaslin, but none in the olivary bodies. But a nearly similar condition was present in the brains of 5 idiots, 3 paretics, 3 paranoiacs, and 3 senile dements examined by this author. We may reasonably, therefore, exclude diffuse gliosis as a cause of epilepsy. Such changes are probably secondary in nature, and cannot be regarded as anything more than new and helpful pathways made into an unknown region.

From the point of view of epilepsy as a nutritional disorder of the cell protoplasm it would seem that the best results will be obtained from studies of the conditions of the ganglion cells in the cortex; and this should be done as speedily as possible after autopsy. Some very instructive work in this direction has been carried on by Bevan Lewis, who finds changes in the small, irregularly-shaped nerve cells in the second layer of the cortex. A degenerative change affects first the nucleus and afterward the cell protoplasm. The centre of the nucleus is occupied by a bright, highly-refractile globule of fat, which increases in size

until it fills the nucleus, or the nucleus may be filled with two or three such globules. The whole cell ultimately becomes vacuolated, and may break down and disappear, as seems to be indicated by the paucity of elements in this second layer as compared with the same region in healthy brains. Somewhat analogous degenerations are observed in other layers at times, but not nearly to the same extent as in the second. Van Gieson has followed Bevan Lewis in this species of investigation by selecting for study fresh cortical tissue from two patients of Starr's who were trephined by McBurney. One was a case in which there was traumatic epilepsy of four years' standing, and a splinter of bone was removed from the brain. There was a plate of connective tissue projecting into the depressed part of the brain. The brain tissue seemed softened about it, and a portion of the softened area was cut out. In the ganglion cells degenerative changes were found in the protoplasm, which in some led to the entire disappearance of the cell body. The early stages of degeneration were marked by the appearance of hollow-looking vesicles in the cell body. In a later stage these vesicles coalesce, reduce the cells in volume, deform their contours, and cause the loss of their processes. There was a hyperplasia of neurogliar tissue, and in the vicinity of the small pyramids were clusters of young neuroglia cells. In the second case, likewise carefully studied by Van Gieson, there was a cortical cyst beneath a depressed fracture of the skull of some ten years' standing, giving rise to a hemiplegia and epilepsy. An analogous condition of things was found in both cases.

The work of Van Gieson differs from that of the other authorities just mentioned in that his cases were instances of marked organic disease from injury to the brain, and the degenerations found were doubtless due to the effects of the trauma upon the tissues in the immediate neighborhood, such as vascular disturbance, softening, and the like. Similar cell and neurogliar changes may possibly underlie many cases of idiopathic epilepsy, but it is more likely that the molecular cell changes are less gross than this in the majority of cases of essential epilepsy. At any rate, the fresh method of study of brain sections and investigations from the standpoint of physiological chemistry are doubtless the avenues which we must follow to gain access to the mysterious domain of the true pathology of epilepsy.

Symptoms.—The chief symptoms of epilepsy centre about the seizure, and the intervals between attacks often offer no indications of great moment to aid in the recognition of the disorder. We shall attempt a sort of classification of the symptoms for purposes of better study into the following groups: viz. pre-epileptic or premonitory symptoms; symptoms during the seizure; post-epileptic symptoms; interparoxysmal symptoms. Many cases will be observed that present no *premonitory symptoms*, the patients falling into their attacks as if struck by lightning. But in a majority of the cases there is some subjective warning of the approaching seizure, mental or physical in its nature, and to this premonition the name *aura* has been given.

Aurœ.—The *aurœ* are exceedingly diverse in character. They may be of an intellectual nature, may arise in the domains of the special senses, of common sensibility, or of visceral sensibility, or they may be cephalic, motor, or vasomotor. The duration of the precursory symp-

toms is variable. Usually lasting but a few moments or minutes, there are sometimes premonitions for hours or days. It is rare for any but the psychic auræ to be of any long duration.

The *intellectual aura* varies greatly in its manifestations. It may consist merely of a feeling of unusal quiet, of absent-mindedness, or the patient may become bewildered, anxious, restless, and irritable. Instinctive desires may be the form of premonition, such as unnatural hunger or a strong tendency to run. The psychic aura may take the form of a dreamy state, an emotion of fear, a particular idea, a series of rapid reminiscences, or a sense of things being strange or wrong. Sometimes, though rarely, it amounts to a veritable insanity, the so-called pre-epileptic insanity.

The *special-sense auræ* are particularly interesting. Visual auræ are more common than auditory, while olfactory and gustatory auræ are only exceptionally met with. As regards the first named, the premonitions are usually of the nature of flashes of light or color or sudden blindness. Color spectra may be particolored or consist of one particular color, commonly blue or red. There are sometimes visual hallucinations, elaborate or simple, pleasant or disagreeable, such as a scene, an animal, a face, or a figure. The objects seen are generally stationary, though sometimes mobile, and appear to approach or recede. I have called attention elsewhere[1] to the fact that the loss of sight may be hemiopic, and that there may be hemiachromatopia or homonymous hemiopic hallucinations.

In an analogous manner the auditory apparatus may be the seat of the premonition, and this, too, may be simple or elaborate, disagreeable or pleasing. Loss of hearing is not frequent. The auditory aura is usually a simple sound, a roaring, crashing, hissing, or whistling. Sometimes there is a sound of talking or delightful music or a tumult of noises.

Olfactory premonitions, when they occur, consist, as a rule, of disagreeable odors.

Gustatory sensations are the rarest of special-sense auræ. They implicate some one of the four primary sensations of taste—salt, bitter, sour, or sweet—usually one of the first three.

The *auræ of common sensibility* usually consist of various paræsthesiæ, such as numbness, tickling, tingling in an extremity, with a tendency to movement along the sensory pathway toward the head. While the sensation is generally of this simple nature, it may be painful or burning in character or a sensation of movement in the part (without actual motion). It may begin in the thumb, fingers, palm, wrist, or leg, and, while commonly centripetal in its progress, it may pass either up or down a limb, from one limb to another on the same side, or across the trunk to the opposite extremities.

The *auræ of visceral sensibility* are not uncommon. Indeed, one of the most frequent premonitions is the so-called "epigastric" aura. It varies from an obscure sense of discomfort to severe pain. It may begin lower down in the abdomen, in the middle, or to the left or right, and often progresses upward toward the head. Reaching the throat, it

[1] "Homonymous Hemiopic Hallucinations," *N. Y. Med. Journ.*, Aug. 30, 1890, and Jan. 31, 1891.

induces a choking sensation analogous to the hysterical globus and to the œsophageal constriction often complained of by melancholiacs. Sometimes the aura consists of this choking sensation only. The epigastric aura may be associated with a feeling of nausea. Occasionally the seat of the premonition is in the præcordial region, and may be painful or consist of palpitation.

Cephalic auræ consist of vertigo, more or less headache, a sensation of weight or pressure or of a feeling of sleepiness. Sensations of giddiness are among the commonest premonitions.

Motor auræ may present themselves as simple twitchings in the extremities, trunk, neck, or face, as general tremor, or as palpitation, spasm of the glottis, strabismus, or retching. Sometimes the twitching in an extremity is the real beginning of the general spasm, as in the case of local epilepsy, when it is well called a "signal" symptom. Loss of power in the extremities, a veritable paresis or paralysis, occurs as a warning, but is rare. The execution of elaborate movements, such as running, turning round, jumping, or tossing the arms, belongs to the category of motor premonitions.

Under the head of *vasomotor auræ* are comprised such symptoms as general chilliness, general sensation of heat, pallor, and coldness, or congestion and sweating of the extremities.

While the above constitute, in the main, all of the warnings, it is to be remembered that there are greater or less degrees in their manifestations, and that combinations of various auræ are met with in endless variety. They are as various as the functions represented in the cortex of the brain, for, while not perhaps actually demonstrated, it is more than probable that all of these auræ are central in origin and indicate the seat of the beginning of the katabolic discharge in the cortex.

The Paroxysms. —The *symptoms manifested during the paroxysm* itself vary much. Attacks are commonly divided into two great classes—the severe (*grand* or *haut mal*, or *epilepsia gravior*) and light (*petit mal*, *epilepsia mitior*).

Severe or major attacks often begin with an initial wild cry (the epileptic cry) immediately before or coincident with loss of consciousness. The patient falls with his whole voluntary muscular system in a condition of tonic rigidity. The posture of the head and limbs varies at this stage. The head may be drawn backward, but is usually rotated to one side. The arms may be thrown above the head, but are commonly abducted at the side, with the elbows, wrists, and metacarpophalangeal joints flexed, the thumbs drawn into the palms, and the finger-joints extended. The legs are extended. Sometimes the arms are extended, all the fingers flexed (hands elevated), and the hips and knees flexed. The spine is curved backward. The tonic spasm affects the respiratory muscles, and the face becomes cyanotic. The jaw is strongly locked. Sometimes the tonic spasm is so strong as to dislocate the shoulder. I have known of a case of dislocated shoulder occurring inexplicably on one or two occasions at night, which was finally recognized as nocturnal epilepsy. Lately a patient has been under my observation in whom dislocation of the jaw accompanies every paroxysm.

In a few moments twitching begins in the muscles of the eyes, face, tongue, jaws, neck, and extremities. The head is beaten against the

floor, the eyes roll upward and to one side, the facial muscles twitch, the jaw opens and shuts rapidly, often catching the protruding tongue between the teeth, and the limbs are affected with clonic spasm of coarse or fine character. As a rule, the clonic spasm is not equal on the two sides, the movements being stronger on one than the other. Sometimes the paroxysm consists simply of the unconsciousness, falling, and a tonic spasm of short duration. When the tongue is bitten a bloody froth is apt to appear at the mouth. The pupils are widely dilated during an attack, and do not react to light. The corneal and skin reflexes are lost. The pulse is quickened slightly, but the heart's action is always regular. Respiration is hurried. The temperature does not change except in long-continued convulsions or status epilepticus, and then the increase is usually not great, except in the latter state, when it may rise to 105° or even 107° F. Involuntary passage of urine and occasionally of fæces is not uncommon. The surface of the body often becomes covered with sweat during the seizure. All of these symptoms then gradually abate, and consciousness either returns very shortly or, as is generally the case, the exhausted patient sinks into a natural and deep sleep, lasting from a few minutes to an hour or more. Such, in brief, is the attack of *grand mal*.

Petit mal, or minor epilepsy, manifests itself in various degrees and ways. Often it is so slight that it is unrecognized by the patient or his friends. In other cases it is described as merely spells of fainting or giddiness. It is, in reality, an abortive or rudimentary form of epilepsy. In its lightest form the attack is a vertigo lasting but a moment and without apparent loss of consciousness. A less mild degree is one in which there is a momentary loss of consciousness, but without other noticeable symptoms. In such cases the patient suddenly fixes his eyes in a staring manner, and may for a moment pause in anything he is doing or saying, resuming it immediately afterward, or he may continue during the brief interval in an automatic manner whatever he may have been doing. Sometimes the head drops a moment and the eyelids quiver. The French term this *absence*. There are cases in which the automatic, or subconscious, state persists for an indefinite period, in which elaborate intentions are carried out, such as journeys, transaction of business, etc.; and this constitutes what is known as ambulatory automatism, epilepsia larvata, masked epilepsy. In other cases, a degree more severe, in addition to the loss of consciousness there may be twitchings of the arms, legs, or head, slight spasm in the limb or limbs, general tremor, choking in the throat, an epileptic cry, or a sudden fall to the floor, which is immediately recovered from. In still others the arms may be thrown up in a peculiar manner, or the hands rubbed together, or the patient walks or runs forward or backward (epilepsia procursiva), or turns around rapidly, or walks in a circle, or begins to undress himself. It is not uncommon for quite complex and elaborate movements to be executed, some of them seemingly purposive, such as pocketing an object at hand or making an attack upon any one near by. As an instance, one patient of mine, whose attack an injudicious friend attempted to arrest by squeezing his wrist, unconsciously took the friend by the collar and unceremoniously kicked him out of the house. Such cases as these just mentioned are not infrequently brought before the

courts on criminal charges of one kind or another—stealing, violence, or even murder. Involuntary evacuation of urine or sudden pallor may accompany minor attacks. Auræ analogous to those of the severe type may be present in epilepsia mitior. Hysterical phenomena are sometimes superimposed upon true epilepsy, and are especially likely to be added to the minor attacks, giving rise to the condition known as *hystero-epilepsy*. Hysterical convulsions should therefore always be carefully investigated for the presence of actual epilepsy.

Epileptic seizures, whether minor or major in character, are sometimes replaced by states of mental excitement or depression, and they are then designated as *psychic epilepsy* or psychical "equivalents" of epilepsy. In such conditions, which may be momentary or endure for an hour or more, great violence, or even homicidal tendencies, may be manifested. In very rare instances the form of sleep known as narcolepsy may take the place of a paroxysm.

The *post-epileptic symptoms* also present much variety. The attack may pass off without sequelæ of any kind. Generally there is a tendency to sleep, especially after the major form. Sometimes headache, more or less severe, supervenes. Immediately after a severe paroxysm the reflexes may be absent or diminished, but very soon there is a hyperexcitability, so that the knee jerks and wrist jerks are exaggerated, and ankle clonus even appears. There may be a transient loss of power in one or more limbs after an attack (hemiplegia or hemiparesis), or paræsthesiæ in the limbs or even temporary aphasia or hemianopia. Occasionally there is concentric limitation of the visual field. Hysterical and automatic phenomena may succeed any kind of a fit, though more common after the lighter variety. Vomiting sometimes takes place after an attack. Great hunger is not infrequently observed. In a considerable proportion of cases there is a tendency to turn over upon the face after a seizure, and this is a not uncommon cause of death, from the asphyxia that ensues while the patient is still in a deep sleep. Attacks of insanity, excited or depressed in character, or trance states, may follow epileptic seizures.

The *interparoxysmal symptoms* are few and not particularly distinctive. This fact renders diagnosis during the intervals between attacks difficult, and indeed generally impossible, unless we depend altogether upon the history as given us by the patient or his friends. The epileptic interval therefore demands much further study than it has had. Usually there are presented indications of uncertain character only, such as the mental hebetude of chronic and severe cases, evidences of a bitten tongue, or the marks of former treatment (bromide eruption, belladonna pupils, etc.). The general health of the patient may be perfect in every respect, and in the majority the mental condition is normal during the interval (unless deterioration has been brought about by the overuse of drugs and by neglect of education and occupation). While a manifest or latent inequality of the pupils (anisocoria) may be observed in many cases, it is doubtful if it is particularly more frequent in epilepsy than in other cerebral disorders, or, for that matter, in health. A certain amount of gastro-intestinal disturbance is present in some cases. Ecchymoses in the conjunctivæ and skin of the face and neck have some significance, especially if not explicable in any other way.

But these, with soreness of the limbs and headache, are symptoms present only immediately after attacks. Epileptics are apt to have thick lips and bad teeth, and the majority of them certainly are brunettes. We will not uncommonly find among them anatomical and physiological stigmata of degeneration.

The *frequency and time of attacks* present much variation. There are cases that have but a few seizures in the whole course of their lives, while others, again, suffer from them once a month, once or twice a week, or many times a day, without any particular regularity. Sometimes a series of attacks in one day or in a week may be followed by an interval of weeks or months of freedom. Where major seizures follow each other so rapidly as to show no very great interval it is termed the *status epilepticus*. Death not infrequently occurs in this condition. The petit-mal attacks are apt to be very frequent, often taking place every few minutes. Occasionally in women one meets with cases where the paroxysm or series of paroxysms takes place with considerable regularity about the period of menstruation. As a rule, epileptic seizures may occur at any hour of the day or night, but sometimes they are distinctively diurnal or nocturnal.

COURSE AND TERMINATION OF EPILEPSY.—The disease is chronic, and when once the habit of epileptic discharges is established, the tendency is rather to an increase in frequency of seizures than to diminution or cessation. This is more true of the disorder when it begins in early life than of epilepsia tarda. When the onset is before the period of puberty there is apt to be an increase at this physiological epoch. Insanity develops in about 10 per cent. of all cases, and a certain amount of mental deterioration (usually induced by the too free use of injurious drugs and by the absolute neglect of mental training, occupation, amusement, education) takes place in a somewhat larger percentage. The majority of cases, properly treated and properly cared for, present no particular alteration in the mental faculties, and epilepsy may be coincident with the highest mental development, even genius. In such as exhibit signs of mental deterioration outside of insanity proper we observe chiefly a greater or less degree of enfeeblement of the faculties of attention and memory and an irritability of disposition. The tendency to mental change is greatest where epilepsy begins in infancy or childhood, and is usually, though not always, directly proportional to the frequency of attacks.

As a rule, patients developing epilepsy continue to have the seizures during the remainder of their lives, though a small percentage is curable, and in some rare instances there is spontaneous and inexplicable arrest of the disease.

The occurrence of paroxysms is sometimes, but not always, prevented for a time by intercurrent acute diseases, and rarely by pregnancy; and they are not infrequently interrupted for considerable periods by startling emotion (fright), by accidents, by surgical operations of one kind or another, and by sudden changes of treatment.

The disorder itself does not ordinarily cause death, although it may occasionally do so by the production of cerebral or meningeal hemorrhage, by exhaustion in the status epilepticus, or by suffocation or other accident during a paroxysm. Patients usually die from intercurrent

affections. The average length of life is, however, shortened by epilepsy.

Epileptic Insanity.—There is a tendency on the part of many physicians, especially such as are brought into contact with epilepsy in asylums for the insane, to class all epileptics among the insane. It is true that epilepsy is frequently associated with insanity, and that the proportion of insane patients afflicted with epilepsy in asylums is somewhere about 9 or 10 per cent. But it is equally true that in any population it is found necessary to commit but 10 per cent. of all epileptics in a community to asylums, and even this has been excessive, for the reason that a considerable proportion of sane epileptics have been committed to institutions for the insane because no other form of hospital receives such cases. The vast majority of epileptics, under favorable conditions, are not, and do not become, insane. But, taking the small percentage of cases of epilepsy associated with insanity, not all of these owe their mental aberration to the epilepsy. It must not be forgotten that the same hereditary instability of the nervous system underlying the epilepsy is coincidentally effective in producing the insanity. This is true of no inconsiderable proportion of cases.

There are examples, too, in which epilepsy is caused by insanity, and others in which both insanity and epilepsy are simultaneously produced by one cause (such as a blow on the head or alcoholism).

Epileptic insanity is manifested in protean forms, so that no definite clinical syndrome can be described under that name. The mental conditions may present themselves in forms of idiocy, mania, melancholia, dementia, delusional insanity, hypochondriasis, and automatic or dream states; and the attacks may be acute, subacute, chronic, or periodical. As regards relation to the paroxysms, the insane outbreaks may be pre-epileptic, may take the place of an epileptic attack, may be post-epileptic, or the insane condition may be chronic, with or without exacerbations at the times of seizures. A sort of classification of the more common psychic phenomena of epileptic insanity may be made as follows:

I. General enfeeblement of all of the mental faculties of varying degree (epileptic dementia).

II. The so-called subconscious, dreamy, or automatic states.

III. Enfeeblement and retardation of mental development in early childhood (epileptic idiocy or imbecility).

IV. Outbreaks of great excitement, with violent, destructive, suicidal, or homicidal tendencies (epileptic mania).

V. Conditions of periodical emotional irritability: Patients may be moody, irascible, peevish, obstinate, suspicious, morose, or have outbursts of uncontrollable laughter and other hysterical manifestations.

VI. Conditions affecting the ethical nature: Disposition to intrigue, steal, or lie; strong religious proclivities.

Diagnosis.—In the majority of cases of epilepsy there is no difficulty in diagnosis if the recurrent attacks have been observed. In a diagnosis of genuine epilepsy we must exclude as far as possible organic and toxic causes (brain disease, uræmia, acetonæmia, etc.). One or two or three attacks are not usually sufficient to establish the presence of epilepsy. They may be symptomatic convulsions. Interparoxysmal

symptoms aid us very little. Seizures which occur at night solely may escape observation, and even the knowledge of the patient himself, for years. Cases which have every indication of being genuine epilepsy may on careful examination show vestiges of an old monoplegia or hemiplegia, such as unilateral increase in reflexes, traces of paresis, slight retardation of growth of extremities on one side, mild athetoid movements, or a certain amount of rigidity. Organic epilepsy, too, tends to exhibit a unilateral character in the spasm, though in old cases of hemiplegic epilepsy this unilateral character is usually lost. The milder grades of petit mal often go unrecognized as epilepsy, and are designated as fainting or dizzy spells. The differential diagnosis between epilepsy and attacks of syncope should be clear from an examination of their distinctive symptoms, which may be paralleled as follows:

Epileptic Seizures.	*Fainting Attacks.*
Absence of exciting causes.	Exciting causes in the way of hot rooms, bad air, emotional strains.
Aura of brief duration or absent.	Premonitions for some time before loss of consciousness.
Sudden loss of consciousness.	Gradual loss of consciousness.
Pulse normal.	Pulse weak, often scarcely perceptible.
Pupils dilated and light reflex lost.	Pupils small or unchanged.
Spasm tonic or clonic.	No spasm.
Often involuntary micturition and defecation.	Rarely or never.
Biting of the tongue.	No biting of the tongue.
Cyanosis.	Pallor.
Short duration of unconsciousness.	Duration of unconsciousness longer.
Automatism or stupor after attack.	Speedy recovery after attack.

Attacks of epilepsy of a vertiginous nature may closely resemble ordinary vertigo, but labyrinthine vertigo is especially capable of confusing the diagnosis. Remembering the above distinctive features of epilepsy, the common forms of vertigo should be readily differentiated, and in the labyrinthine variety this is commonly persistent, besides rotary movements, tinnitus aurium, some deafness, and some interparoxysmal giddiness. The two disorders may, however, be associated.

In attacks of epilepsy, with hysterical symptoms superadded, it is often difficult to distinguish the condition from seizures purely hysterical in character. The following parallel will serve to illustrate the differentiating features:

Epileptic Seizures.	*Hysteroid Attacks.*
No exciting cause.	Emotional cause.
Aura or no premonition.	Globus, palpitation, malaise.
Epileptic cry.	Crying, talking, screaming during attack.
Sudden and complete loss of consciousness.	Loss of consciousness incomplete.
Pupils dilated.	Pupils unaltered.
Tonic followed by clonic spasm.	Rigidity, opisthotonos, struggling and tossing movements.
Biting of the tongue.	Biting at self or others and objects at hand.
Involuntary micturition and defecation.	Never.
Duration brief.	Duration often for long periods.

Malingering is not so frequent in this country as abroad, where, for instance, freedom from military service may be purchased by successful simulation of epilepsy. We meet with some cases of feigned epilepsy, however, particularly in the so-called "dummy-chuckers," who have a fit in order to collect a crowd in which their pick-pocket associates may operate, or for the purpose of securing the comfortable care of a hospital for a time. Very rarely the disorder is simulated by criminals. Seizures may be accurately imitated, even to slight biting of the tongue and foam at the mouth (made by soap). The conclusive evidence of simulation will be found in the normal pupils. In true epilepsy the dilated pupils fail to respond to light.

PROGNOSIS.—The prognosis of epilepsy is very unfavorable, no matter whether a cause, organic, toxæmic, reflex, or other, be discovered or not. Under present methods of treatment perhaps somewhere between 2 and 6 per cent. of the cases of epilepsy are cured. Spontaneous cessation of the attacks is occasionally met with. Treatment may relieve patients from seizures for long periods of time, even as long as a year or two, after which the paroxysms return as before. It is unwise, therefore, to pronounce any case as cured until after the lapse of years. When an interval of two years has passed without relapse we may be ordinarily justified in adding it to our statistics of cured cases.

The dangers which lie before the epileptic are mental impairment, death in the status epilepticus from exhaustion, death by asphyxia (turning on the face in bed), death from asphyxia or pneumonia from vomiting after a seizure, and death by accidentally falling from heights or into the water, etc.

The prospects of cure are better in cases beginning in adult life than in childhood. The shorter the duration of the disease the better the prognosis. Browning has recently made the question of sex in the prognosis of epilepsy the subject of a careful study, and concludes that, while there is a fair chance of cure in the most favorable male cases, little is to be expected in female cases.

In post-hemiplegic epilepsy the outlook is much less favorable than in genuine epilepsy, not only as regards cure, but also as regards diminishing the number of seizures.

TREATMENT.—In undertaking the treatment of epilepsy we must be guided by the indications in each particular case. Our first duty is to discover, if we can, any apparent cause, and treat that if possible. As has been already intimated, there are many cases in which a probable exciting factor may be found, traumatic, reflex, toxæmic, or other. But in the great majority of cases of epilepsy no evidence will be elicited of any particular cause, and in these our treatment must be of two kinds—viz. general treatment for the purpose of improving the mental and physical conditions, and special treatment, always more or less empirical, directed to diminishing and aborting the convulsive attacks.

It is not too much to say that moral treatment, upon which so much may depend, is almost never referred to in our text-books. The unfortunate epileptic has been dismissed with a prescription or two and a few general directions. His infirmity has isolated him from companions and debarred him from the schools, recreations, and the occupations of

his more fortunate fellows. No hospitals are open to him. Left thus to his own resources, he has grown up ostracised as it were, and in ignorance, neglect, and idleness. Our authors have written pages upon the treatment with countless varieties of drugs of a class of cases confessedly incurable or almost so, yet scarcely a word concerning the alleviation of a lifetime of distress. Now, however, this state of things is undergoing change. This moral and general treatment of the epileptic is really, in the present state of our pathological knowledge, of paramount importance. In order to ameliorate his condition the first requisite is to overcome these depressing and morally degenerating conditions. The epileptic must be placed in surroundings where the sense of isolation from the rest of mankind will not harass him; where his education will be accomplished in ordinary and industrial schools; where he may be provided with occupation of any kind for which he may show a proclivity, especially out-of-door pursuits; and, finally, where he may enjoy the advantages of social intercourse and various recreations. To this end residence in a village community or colony especially adapted to these cases is advisable. There are now a number of such colonies abroad, and Craig Colony at Sonyea, N. Y., established a few years ago, is serving as a model for a number of similar communities just being founded in other States.

In regard to general physical health, besides regular occupations and interests, and a life chiefly out of doors, simplicity of diet, the care of the bowels, and continuous hydrotherapy are important. A diet chiefly of milk, fruit, cereals, and vegetables is best in most cases. Usually meat may be allowed once daily. Cold rain-baths, cold sponge-baths, or cold half-baths, once or even twice daily, are beneficial (temperature ordinarily 60° to 70° F.). Some cases require general up-building, and conditions of impaired nutrition, anæmia, and nervous depression may be met, in epilepsy as in other diseases, by iron, arsenic, quinine, strychnine, and the like when indicated.

As to special treatment, this must be to a great extent empirical, as there is no specific remedy for epilepsy. The drugs which have been found the most useful and are generally employed are the bromides. The bromides of potassium, sodium, ammonium, strontium, camphor, nickel, and rubidium-ammonium bromide, have been extolled by various authorities, either singly or in combination. But it is probably best to make use of but one salt, and that the bromide of potassium, which has the least irritating effect upon the gastric mucosa. It is unfortunate that it cannot often be obtained in absolutely pure form, for usually it contains some 6 per cent. of chlorate of potassium. Ordinarily it is given in 10–15-grain doses three times daily, in plenty of water, after eating, though there are cases in which it acts better when the daily total is given in one dose, at bedtime, in a full glass of water, or, according to the German method, in a cup of valerian tea. As a rule, it is pushed to the verge of brominization until severe acne appears and the throat reflex is lost. In some cases it is well to adjust the giving of the bromides as nearly as possible to the period of expected seizures, so that large amounts are taken a few hours before their appearance. The most that can be hoped for from the bromide is a diminution in the number and severity of the paroxysms. Cures are effected by it with extreme

rarity. In perhaps 90 per cent. of cases a favorable action is obtained. In the remainder it avails little or acts as a poison. It is unwise to continue in all cases the unlimited use of bromides, for in too many instances the stupor or dementia of epileptics has been caused or precipitated by such use. When a cure has apparently been produced by bromide or any other drug, the drug needs to be continued for a year or more after the cessation of attacks. In old cases of epilepsy in which undoubtedly the bromide treatment has already been employed for years, it is just as well to begin by the exhibition of some other agent or a combination of the bromide with some other remedy. Arsenic, and sometimes chloral, in conjunction with the bromide, prevents the development of acne.

Of late, the method advocated by Flechsig, the opium-bromide treatment, has had considerable test of its efficacy. Opium and its alkaloids had often been employed before in epilepsy, and doubtless frequently in some sort of unsystematic association with the bromides. Flechsig's method consists in beginning with one grain of opium three times daily, gradually increasing the doses to five grains three times daily during six weeks, and then suddenly substituting bromide in doses of thirty grains four times daily, this being reduced after a time to five or ten grains a day. My experience has been that in chronic cases, where no other form of treatment has been efficacious, the opium-bromide method is often remarkably successful in causing cessation of attacks for considerable periods of time.

Bechterew has introduced a new treatment of epilepsy by means of bromides in conjunction with adonis vernalis. The latter drug, as is well known, is very similar to digitalis in its effects. It is a means well worth trying in obstinate cases.

Such cases as do not do well on bromides, and such as have evidently been too freely brominized, may be placed upon any one of a number of other agents which have from time to time won the praise of various authorities. Belladonna is occasionally useful, particularly in nocturnal epilepsy. The tincture may be employed in five- to ten-drop doses, gradually increased. Atropine, hyoscine, or duboisine can be substituted for belladonna. Borax is sometimes of service, though its long-continued exhibition irritates the gastro-intestinal tract and injures the kidneys. The dose is fifteen to twenty grains three times daily, in water, after meals. I have seen very favorable results from tincture of simulo, ʒj–ʒij three times daily, and in petit mal pure nitroglycerine in doses of $\frac{1}{100}$ of a grain. Lactate of zinc has been well spoken of. Solanum carolinense has been exploited recently, but I have had no results from its use. There are numberless other medicinal agents which have received commendation from many sources, but the list would be too long for enumeration here.

In the few instances in which a possible cause is discovered for the convulsive seizures the appropriate measures or remedies are to be applied. We should treat any noteworthy eye, ear, mouth, and nose defects or diseases which may be present, although the cases in which any of these will enter as factors in the causation of epilepsy will be rare indeed. The same is true of cicatrices and genital irritation. As regards operations for any of these conditions, it must not be forgotten

that surgical treatment of any kind may prevent seizures for months, whether it be of the nature of a simple circumcision, injudicious tampering with eye-muscles, malpractice upon the female genital organs, or what not.

Gastro-intestinal irritation in some cases demands attention, as do also any marked symptoms of auto-toxæmia from fermentative or putrefactive changes in the alimentary tract. In instances of the latter the normal ratio of the preformed to the ethereal sulphates in the urine (10–1) is altered, so that the ethereal sulphates become excessive in proportion (7–1, 5–1, or even 3–1), according to Herter, and indigo-blue or phenol will be found in the urine in quantity. Treatment is then directed to diminishing sepsis in the alimentary canal, and intestinal antiseptics, such as salol, beta-naphthol, salicylate of soda, oil of peppermint, etc., are employed, together with copious drinking of hot water and care in the diet. In the regulation of the diet it is best to avoid, as far as possible, fermentable foods, and to restrict the diet to beef, milk, and green vegetables.

The treatment of the attack itself consists, where there is an aura, of the patient's inhaling nitrite of amyl from amyl pearls broken in his handkerchief or from a glass-stopped bottle containing some cotton saturated with amyl nitrite. Where attacks begin by a sensory or motor aura in a hand or foot they may sometimes be interrupted by a ligature about the wrist or ankle.

Certain hydrotherapeutic measures are serviceable at times in an ordinary attack or in the status epilepticus. If the face is pale, warm wet compresses may be applied to the head and genitals, with friction of the trunk upward and a low position of the head. Where there is turgescence of the vessels of the head and face cold compresses to the head, neck, and genitals, high position of the head, and strong wet beating of the feet are indicated. In the status epilepticus chloral per rectum is usually the best remedy. In all cases during attacks the patient should lie prone, the head upon a pillow, the clothing freed about the neck, and a wooden peg or a corner of a towel be put between the teeth when necessary to prevent biting of the tongue.

Some sort of register of the daily, weekly, monthly, and yearly number of attacks should be kept in most cases, so that the physician may note the effect of his treatment upon the malady. An ordinary calendar may be used for this purpose, but much better is the register for epileptics published by G. P. Putnam's Sons of New York at the instance of the writer.

The Surgical Treatment of Epilepsy.—Aside from counter-irritation of the neck and scalp, ligature of one or both vertebrals, and other procedures which in rare instances afford some relief, trephining the skull, and sometimes excision of diseased parts of the cortex, have been extensively tried, especially of late years. Probably not so much will be done hereafter as was done immediately after the new cerebral surgery came into vogue. Trephining for epilepsy is a very old operation. It is a question how far it is justifiable even in cases where the procedure has good grounds for trial, for it must be confessed that cures are almost never accomplished. The most suitable cases for trephining are such as originate from fracture of the skull and depression of bone,

particularly when the lesion is recent, is situated over motor areas, and gives rise to seizures beginning in some particular part—the arm, leg, or face. The excision of the cortical focus causes a paralysis of the muscles represented in the part, but, as Horsley and others have demonstrated, such paralysis lessens or disappears after a time. There is a fair probability of benefit in local organic cases of this nature, and a remote possibility even of cure. Usually any such operation has the effect of diminishing the frequency of attacks for a short period, whether it be trephining or a simpler surgical procedure. Trephining in itself is not a serious operation, the mortality being only 6 to 7 per cent., but I do not know of any class of cases of epilepsy in which trephining alone has any object. Where the membranes have to be opened and a focus of disease is removed from the cortex the operation becomes more serious. There is a disposition on the part of reporters of cases operated upon to announce recoveries at too early a period. Several months of remission may follow a circumcision or meddlesome interference with the eye muscles, and the probability of a remission in the symptoms is naturally greater after a more severe operation. If two years have elapsed after an operation without recurrence of the attacks, it is probable that the patient has been cured of his epilepsy. A fair argument against operation, even in many of the formidable cases, is that by trephining and removing a portion of the cortex a new cicatrix is merely substituted for the old focus of disease. In many cases of organic epilepsy operation is seldom or never justifiable at all. In this category I should include most cases of epilepsy following infantile cerebral hemiplegia, where the hemisphere presents atrophy, sclerosis, cyst, or porencephalus as a secondary condition from old hemorrhages or thrombosis. Among some 2000 cases of epilepsy that have come under my observation, I do not remember to have seen more than 15 or 18 that I should consider subjects for surgical interference. Of 12 or 13 operated upon I have never seen 1 cured.

INFANTILE CONVULSIONS; ECLAMPSIA.

By EDWARD D. FISHER, M. D.

DEFINITION.—By this term we designate those convulsions, epileptiform in character, which, however, cannot be included under the head of true epilepsy, as, although the convulsion itself may differ little if any from an epileptic seizure, it has not the tendency to become permanent, generally ceasing on the removal of the exciting cause. The term "eclampsia" is not necessarily applied to infantile convulsions only, but also to those occurring in the adult. Tuke gives a very comprehensive definition of eclampsia as follows: "A term generally applied to convulsions of an epileptiform character, due to some actual disturbances of the nervous centres, caused by anatomical lesion from injury or disease (differing thus from pure epilepsy), by a deficient supply of blood, by infection of poisonous matters into the blood (notably urea), or by reflex irritation (*e. g.* intestinal worms, teething, etc.). The term was formerly used as a designation of infantile and puerperal convulsions."

ETIOLOGY.—The causes lie external to the brain, and therefore exert their influence interruptedly. They may act directly on the nervous system, as in the case of its overwhelming in toxæmia, or, again, indirectly, as in reflex irritations. In the study of this form of convulsion I do not refer to established epileptic seizures, but, as said in the definition, rather to those which, although similar in many respects to the true seizures of epilepsy, generally present some external cause of cerebral irritation to which it is probable or possible to suppose the seizure owes its origin. These latter remarks have a special bearing on those cases which apparently have their origin in nasal, ocular, genital, or other sources of irritation; and I refer to them at this point in order to emphasize the fact that one must be careful in looking for the cause of this condition to distinguish between convulsions epileptiform in character and those occurring in true epilepsy, as the latter disease, in my opinion, while it may be affected by these causes of irritation, it can never be caused by them. One observes in many children of an unstable nervous organization or of the so-called neuropathic disposition that slight disturbances of temperature will produce convulsive seizures. Again, many of the acute exanthematous fevers are ushered in by convulsions. This may be due to the direct influence of the infection or to the rise of temperature accompanying these diseases. Acute spinal paralysis frequently commences with convulsions, although in most cases the lesion is very strictly confined to the spinal cord. Acute indigestion or overloading of the stomach with indigestible or undigested materials is a common cause, as also is intestinal indigestion. This latter cause is

more frequent than is usually supposed, and should be most carefully investigated. Convulsions in these cases may be even unilateral, leading to the supposition of the cause being central rather than peripheral, but this should not lead one away to an erroneous idea of the condition. Reflex irritation may arise from nasal obstruction and deviation of the septum or inflammation and hypertrophy of the turbinated bones, or even from foreign bodies in the nose. I have seen such a case where a child had forced a shoe-button up one nostril, relief being immediately obtained on its removal. Again, no doubt insufficiency of the ocular muscles plays an important rôle in producing convulsions, and often when this is relieved there is a cessation of the seizures. This subject has been under discussion for some years past, and probably most of the rancor and misunderstanding which has arisen in regard to these conditions as causes of epilepsy would not have occurred had there been the same definition of epilepsy accepted among those interested. Neurologists, as a whole, do not accept these causes as factors in epilepsy *per se*, while acknowledging them as every-day factors in the production of convulsive seizures. This latter position would seem to be sustained by experience, as the failures to cure by operative measures in this direction have occurred in just such selected cases in which epilepsy has been established as the diagnosis. Reflex disturbances also elsewhere, as from phimosis, rectal diseases, intestinal worms, teething, even scar tissue, operate in a like manner. A little patient under my care, suffering from herpes zoster, had as many as fifty convulsions daily while the acute stage of the neuritis continued, but on its subsidence the seizures ceased and did not return.

Irritative lesions of the brain, as meningitis, especially tubercular meningitis, tumors, injuries, etc., cause convulsions, general or local, which do not continue if the cause can be removed. In the majority of cases of infantile cerebral hemiplegia we have the condition introduced by epileptiform convulsions. These may take on the form of true epilepsy, or be such from the beginning, but in that case the explanation is manifest from the well-known permanent lesions of the brain cortex. The etiology is of great importance in our study of these cases, as otherwise a much more serious diagnosis than is warrantable might be made. Infantile convulsions very frequently have no influence in inducing true epilepsy. Lead and alcohol, as in the adult, can cause convulsions, but naturally these would rarely be a cause in children.

Pathological Anatomy.—There is little to be said of the pathology of this condition. I have spoken of it as a functional disturbance, as a symptom in other diseases, in contradiction to a fixed or permanent state. There appears, therefore, little outside of congestion, which is always an indefinite term expressing the inability to discover a fixed and constant lesion explanatory of the existing symptoms. Perhaps later more advanced investigations will reveal some changes in the motor-nerve cells of the cortex, which, by analogy from the cell exhaustion made out by Hodge in the spinal-cord cells, would seem probable. Or, again, one may expect to observe changes in the end brushes and processes of the cortex cells, as they probably, according to the latest teachings, have most to do with motor disturbances.

Symptoms.—The onset of the symptoms may be sudden, the child

losing consciousness, perhaps giving a cry at this time, although this is more rarely present than usually reported. The convulsion, though general, usually affects one side somewhat more distinctly than the other. The head is drawn to one side, the eyes are fixed and drawn upward. The hands are clenched with the thumb in the palm, and the whole body is in a condition of tonic contracture, and usually that of flexion. This lasts but a brief period, and is followed by a condition of clonic spasm. The conjunctival reflexes are lost and the pupils are usually dilated. There may be involuntary passing of the urine and fæces. This convulsion may be succeeded in rapid succession by others extending over a period of many hours, until complete exhaustion takes place, with even an appearance of paralysis of all four extremities, or, more strictly perhaps, limited to one side of the body. While the convulsion differs, thus, little if any from that of an epileptic seizure, it is characterized by the greater frequency of these seizures, which may be fifty or more in the twenty-four hours. There is usually, however, in the intervals between the seizures much less mental disturbance than in true epilepsy, the child returning to complete consciousness unless the cause has been due to some toxæmic condition, as in the various exanthematous fevers, to injury, or to hydrocephalus. This has been especially observed in cases in which the cause has been traced to some source of irritation, such as indigestion or teething, or some peripheral-nerve lesion. An important factor in differential diagnosis in convulsions, as distinguished from epileptic seizures, is that in the latter there is rarely found in the beginning of the disease such a large number of seizures; it is more usual to find a single convulsion followed by marked mental disturbance, especially a comatose condition, repeated at an interval of weeks or months, perhaps to be later followed by frequent daily seizures. In true epilepsy, in fact, the second convulsion frequently does not occur for six months or a year, whereas in convulsions of the character under consideration the seizures may persist for weeks or months without interruption. In a patient whose history was related to me there was a series of almost innumerable convulsions, occurring daily for weeks, with little mental impairment as a result, although there was a partial hemiplegia lasting for some weeks following the convulsions. In the intervals between the attacks, especially if as long a period as twenty-four hours intervened, the patient would be apparently as intelligent as before the attacks. Their nature seems to be entirely different, and, while due to cortical irritation, as in true epilepsy, they arise from external irritation carried to the cortex of the brain, which may, or may not—and in many instances certainly not—be pathologically unstable. In epilepsy proper the disease is situated in the cortex of the brain, and the seizures may therefore originate entirely from irritation of that area within itself or be secondarily excited by external irritation. Clinically, one may certainly say that where the convulsions have their onset marked by excessive frequency, continuing for several weeks or months, the prognosis is more favorable regarding the establishment of epilepsy than in those instances in which a single or only a few epileptic seizures usher in the disease, to be followed weeks or months afterward by a similar seizure, which may then again repeat itself at a later period. The

danger, therefore, to the patient in convulsions lies in the immediate effect or exhaustion experienced at the time, not in any future impairment of the mental faculties or establishment of a habit of convulsions or epilepsy. These remarks must necessarily apply only to convulsive states not dependent upon injuries, vascular lesions, or inflammatory cerebral conditions.

Complications and Sequelæ.—From the violence of the convulsion there may be some direct injury to the cortex of the brain, as a meningeal or capillary hemorrhage, leading to secondary degeneration in the brain substance. This would seem probable as a consequence of either the cerebral exhaustion or of the possible violence done to the cortex of the brain. It is difficult naturally, on microscopical examination—except, perhaps, where a well-defined meningeal hemorrhage is present—to separate the cause of the convulsion from its consequence, but I believe where the convulsions are of the character which I have described as due to some peripheral irritation, as indigestion, herpes zoster, etc., and where they have been marked, as is so frequently the case in this form, by their great frequency, that it is justifiable to consider the cerebral lesion as secondary to and caused by the convulsion. There may be, therefore, as stated, as a direct result of these convulsions, a hemiplegia or a diplegia. It is rare, however, that these conditions are at all extreme; certainly they should not be confounded with the ordinary forms of cerebral hemiplegia or diplegia which occur in early life or at birth, although they may have been preceded by convulsive seizures.

Diagnosis.—It is often impossible to make the differential diagnosis between a convulsive seizure and epilepsy *per se;* the various points already alluded to in describing the condition may aid. A convulsion, however, occurring in childhood, should always be regarded as serious where there is no existing condition, such as the exanthematous fevers or high temperature in general or any well-defined local irritation. Such a convulsion may possibly be the forerunner of permanent disease of the cortex of the brain.

Prognosis.—The prognosis, therefore, in convulsions is favorable when due to some reflex cause.

Treatment.—The hot bath is to be recommended, with the use of chloroform inhalations, to be followed later by the exhibition of chloral, usually administered by the rectum. Where this is not effective morphine hypodermically must be employed. The later continuance of bromide of potassium and chloral is recommended. Urethan has been successfully used in many instances. Great attention should also be paid to the removal of all local irritation, and I would especially lay emphasis on intestinal and gastric indigestion. In these cases, perhaps more than in any others, favorable results are obtained from the removal of any aural, nasal, or ocular disturbances. Until a thorough examination has been made for such causes of irritation no course of treatment can be followed out with success.

CHOREA AND CHOREIFORM AFFECTIONS.

By CHARLES L. DANA, A. M., M. D.

Classification.—Chorea and choreiform affections may be classified as—

1. Chorea minor;
2. Spasmodic ties, including—Habit spasms, Psychic tic, Progressive choreic tic.
3. Hereditary chorea;
4. Symptomatic choreas (post-hemiplegic, etc.).

The classification which I have given is essentially like that which I gave in 1888, in a lecture on "Chorea, Spasmodic Tic, and Hysterical and Spasmodic Disorders of Childhood," published in the *Archives of Pædiatrics* for March and April, 1888, and which I have since followed, in the main, in my *Text-Book* and subsequent writings.

Sydenham's chorea is undoubtedly a distinct malady, showing, however, varying degrees of permanence and intensity. Spasmodic tic often clinically shows itself in cousinly touch with common chorea, but it has its own individuality, and deserves it. Psychic tic verges closely on a mental disorder. In the symptomatic choreas, such as follow a gross lesion in the brain or a degeneration of the cortex cerebri, there is only a superficial connection with the ordinary chorea or even with tic, and the choreic movements are simply one of many morbid manifestations.

Chorea Minor; Chorea of Sydenham; St. Vitus' Dance.

Definition.—Chorea minor is a subacute disease, characterized by involuntary, irregular twitchings of the muscles and muscular groups of the body, accompanied with some motor weakness and often with slight mental disturbances. It is a disease that comes on rather slowly, increases in severity a few weeks, gradually declines, and runs its course in from two to four months. It affects the body, at first, unilaterally, the face, arm, and leg of one side being more involved than those of the other.

Etiology.—Chorea is essentially a disease of childhood, though it occurs at all periods of life and has even been seen in old age. The choreic age ranges from five to fifteen, the average age of 318 patients of mine being twelve. The years in which the disease is specially prevalent are the tenth, eleventh, and twelfth. It affects girls more than twice as often as boys. Direct hereditary influence is of little importance, but the patients are often of a neuropathic constitution or belong to somewhat nervous families. In 8 per cent. of my cases either a brother or sister of the choreic patient also had chorea.

The disease prevails much more in the temperate climates, and does not very much affect the inferior races, such as the negro or Indian.

The influence of season varies somewhat in different localities. In this country chorea occurs the most often in the spring months, next in the fall, next in the winter, and is least common in the summer-time. But the season is not the sole agent in producing these variations. In the summer the children are out of doors more, and less subject to the depressing influences which bring about the disease. In the spring, on the other hand, they are usually at the end of a long fall and winter's school-work, and the confinement and strain connected with this have no doubt something to do with causing the disease.

Rheumatism has been considered to be a potent factor in predisposing, and even in directly exciting, chorea, and its importance is considerable. Among 318 cases of which I have records, covering a period of thirteen years, I found a history of rheumatic pains or distinct rheumatism in 20 per cent. In most of these patients the rheumatic history was simply that of "growing pains," and the occurrence of a frank outbreak of acute inflammatory rheumatism preceding an attack has been noted in only about 5 per cent. of cases. The statistics from general medical clinics show a larger percentage of rheumatic histories.

My cases further show that endocarditis, which is also placed in close relation with chorea, was noted during the attack in only 10 per cent. of the patients. The last series of cases which I have studied have shown a smaller percentage of heart lesions than the earlier cases. Others have found cardiac lesions more often. Osler noted them in $57\frac{3}{7}$ per cent. of cases examined from two to sixteen years after the attack.

Chorea undoubtedly exists to a larger extent in cities than in the country, and more frequently among the poorer classes, who suffer the evils of badly-ventilated rooms and inferior food. Anæmia and malnutrition, from these and other causes, predispose to chorea.

The strain of school-life as a cause of chorea has been both exaggerated and misunderstood. It is not hard study, but bad methods of living at home, late hours, improper food, and unwise discipline, that cause chorea. Some of our alarmist educators would bring the general level of school-work down to a plane suitable for children with weak lungs, defective eyes, and impoverished blood. But because these classes get chorea from school-work, it does not follow that the school is all to blame.

Of all exciting causes, fright and shock, with or without injury, are the most frequent. The worry and strain connected wiih examinations are sometimes exciting causes, and chorea at times follows an attack of malarial or some other infective fever. Intestinal worms may perhaps excite chorea, though this agent of the disease has become much rarer in recent years.

Eye-strain has been put down as one of the causes of chorea. It sometimes excites habit spasm, but rarely ever a true Sydenham's chorea. The disease is also said to follow, at times, the irritation of nasal disease and sexual disorders. Pregnancy is the cause of chorea, generally in primiparæ and in women under the age of twenty-five.

A kind of pseudo-chorea is sometimes set up in a school or family by imitation. The true disease cannot be caused by this means alone, though an epidemic due to a common infection is possible.

Symptoms.—The disease begins with some slight twitching movements of the face or hand, one side being usually more affected than the other, and the right side being more often affected at first. The patient blinks the eyes, twitches the mouth, grimaces, and jerks the head. The hand starts involuntarily, and in conveying food or drink to the mouth the utensil is sometimes dropped. The trunk and shoulders are twisted about, and the child wriggles in its chair. In walking the feet catch, the legs give out, and the child stumbles or falls. In the course of a week both sides of the body become affected, but generally there is more disturbance on the side originally affected. The movements gradually become still more violent: the patient cannot sit quiet for a moment; the face is continually distorted; the tongue is thrust out; the eyes twitch; speech is indistinct; the head is jerked to and fro; the arms and legs are flung out like flails. A certain amount of muscular weakness accompanies it all, and, if the case is a severe one, in two or three weeks the patient is unable to dress or feed himself except with the greatest difficulty. The speech continues thick and indistinct, owing to the twitchings of the tongue and lips, and swallowing is difficult. The movements cease, almost invariably, during sleep, but in bad cases the twitchings are so severe that sleep is much disturbed. The child wakens with bad dreams, and in some cases an actual delirium occurs, during which the patient screams or talks inarticulately and incoherently, and betrays the same excitement that one sees in the delirium of fever. This state usually subsides toward morning. At all times the patient's mental condition is slightly affected. The child is more irritable and peevish. He cries at little things, and is difficult to please or to manage. There is, with this emotional disturbance, a good deal of actual hebetude and dulness. The appetite is less, and the child becomes pale and loses flesh. Nocturnal enuresis occasionally occurs. The urine contains an excess of urea and phosphates, and sometimes hæmato-porphyrin; the specific gravity is often high. The electrical irritability of muscles is, as a rule, increased, but there are no qualitative changes. The deep reflexes are somewhat lessened. The pulse is often rather rapid, sometimes running up to 120 or 130, and I have seen cases of actual tachycardia without any organic heart lesion. In a percentage of cases (10 to 20), as already stated, a blowing murmur is heard over the heart, and in some instances the evidences of a genuine endocarditis may be found, though usually the murmur is a functional one and eventually disappears. The child becomes pale and anæmic, and examination of the blood shows a diminution of red cells and increase of white.

The symptoms are worse in the morning, and improve in the afternoon. This is more especially the case when the disease has become somewhat chronic. The patient suffers little pain, and sensory disturbances are not often observed. Occasionally, however, rheumatic pains accompany the disease. The special senses are not affected, though the chorea may involve the motor muscles of the eyeball.

The disease reaches its height in about three or four weeks, and in most instances continues without very much change for a month or two, then gradually subsides. In very rare cases the movements are so violent and severe that the child cannot sleep, and can with difficulty

swallow food, and as a result the emaciation and weakness increase, so that the disease ends fatally.

Occasionally, one finds the malady running a very mild course, and affecting only one side of the body, and such cases have been described as illustrations of *hemichorea.*

In other instances the twitching movements are very slight, while the motor weakness is so great as to amount almost to a hemiplegia, and this rare type has been called *paralytic chorea.*

There are also, I am sure, many instances in which children have suffered from a progressive weakness, anæmia, mental hebetude and irritation, anorexia, and disturbed sleep—in fact, with all the symptoms of chorea except the spasmodic; and I believe that there is a type of chorea in which the disease affects the motor mechanisms but very slightly (*latent chorea*).

When chorea affects pregnant women the disease runs very much the same course as in the chorea of children. It begins in the early months of pregnancy, and does not, as a rule, end until parturition, although by suitable treatment it may sometimes be practically cured before that time. It usually affects primiparæ, and in many instances the patients have had chorea earlier in life.

When the psychic functions are particularly affected by the chorea, there results a great mental excitement, especially at night, with delirium, hallucinations, and illusions. After one or two weeks the excitement lessens, and the patient becomes pale and apathetic, then gradually passes into the stage of convalescence. This type is called *chorea insaniens* or *maniacal chorea,* and it affects older children (fifteen to twenty) and adult women. It sometimes ends fatally, and if so death occurs in about a fortnight.

Course and Prognosis.—Chorea usually runs a course, as already said, of two or three months. By vigorous treatment in mild cases the duration can be cut down to four or six weeks. If allowed to run its natural course, it subsides gradually, just as it came on. In a good many instances the child has a trace of the disease remaining in the form of an occasional twitching of the face or hand. If this continues for some months, a relapse is very likely to occur. Relapses, in fact, are extremely frequent in chorea, and amongst my own cases about one-third had more than one attack. These relapses are generally slightly less in intensity than the original attack, and they are apt to occur within a year of the first illness. The same child may have an attack of chorea every year for four or five years. The average number of relapses, however, is two. Among 55 cases of relapsing chorea, 26, or not quite half, had only a single relapse. These recurrences rarely take place after maturity is reached, except when the patient becomes pregnant.

Pathology and Pathological Anatomy.—A considerable amount of evidence is now being brought forward to show that chorea minor is usually a disease of infectious origin. Still, it cannot yet be said that this matter is an established scientific fact. If the process is not due to a microbe, we must certainly assume that there is a morbid diathetic condition—allied to that of rheumatism—and that this disturbing humor of the blood is poured out and irritates the nerves, precisely

as Sydenham suggested more than two hundred years ago. I cannot express my views upon the pathological anatomy of the disease in any different way to that which I have used before in my communications upon this subject. I believe that the seat of the lesions in chorea is in the brain, and is usually in the gray matter of the motor area of the cortex, involving also somewhat the pyramidal tract and basal ganglia. The lesions are in acute and severe cases of the nature of intense hyperæmia, with dilatation of vessels, small hemorrhages, and spots of softening. There is in these cases infiltration of the perivascular spaces with round cells and swelling and proliferation of the intima of the small arteries. In mild and chronic cases the evidence of active vascular irritation is less, but there are perivascular dilatations and increase of connective tissue. The process suggests a low grade or an initial stage of inflammation (*encephalitis subacuta non-suppurativa*), but more disseminated and less intense than in the ordinary types. The pathological examinations of cases of hereditary and chronic chorea (Oppenheim, Dana), as well as those of acute fatal chorea, give support to these views (Fig. 61). The hyperæmic process may not be confined to the meninges and motor areas of the brain and cord, but it is only from the disease in these parts that the symptoms of chorea arise.

The presence of points of irritation in the cortex and its meninges and in the deeper parts excites discharges of nerve force and produces the choreic movements. The interruption of the voluntary nerve impulses by diseased foci makes these movements irregular. The apparently special involvement of the basal ganglia may explain some of the inco-ordination. In paralytic chorea the pyramidal tract is probably more seriously injured by some single large focus of congestion, exudation, or hemorrhage. In maniacal chorea the meninges and cortex are more involved.

In a considerable proportion of cases, especially those of long duration, evidences of endocarditis are present.

Diagnosis.—The disease is to be distinguished from spasmodic tic, hysteria, and gross lesions of the brain leading to symptomatic choreic movements. It is differentiated from spasmodic tic by the fact that the twitching movements are wide and irregularly distributed, and do not simulate any purposive movements. In the spasmodic tic the child opens and shuts his mouth or distorts his features in a grimace, or he jumps up and down on the floor, or jerks his arm in some regular co-ordinate movement. In chorea the spasm usually begins unilaterally—is more pronounced on one side than on the other. In tic the movements are limited to a few groups of muscles, such as those of the eyes or larynx, or to the trunk or the legs, while in chorea the movements are unilateral or general. Chorea attacks children who have not any special marked neurotic taint and who are usually bright mentally. Spasmodic tic affects neurotic children, and is sometimes associated with decided mental deterioration. Chorea begins rather suddenly, in a comparatively short time reaches its height, and runs a subacute course. Tic may be said to be chronic from the start. Chorea is usually associated with physical symptoms of malnutrition, anæmia, general weakness, anorexia, and sometimes with rheu-

matism and cardiac trouble, while spasmodic tic may occur in persons who, physically, are otherwise quite well.

Hysterical spasms are readily distinguished by the fact that they are never simple inco-ordinate twitching movements, but are usually rhythmical, involving often the entire muscles of the body. Thus in hysterical spasm the child dances upon the floor, or has attacks of oscillatory spasm of the head, or goes through movements which are indicative of the mental excitement associated with it. Hysteria also usually attacks children of an older age during the period of adolescence or early adult life, and the emotional crises and the characteristic emotional mental state are often present. The physical stigmata of hysteria, such as contractures, palsies, anæsthesias, are also present and distinctive.

Prognosis.—The prognosis in chorea minor is almost invariably favorable. A great many patients, however, have a relapse, and in some instances the disease passes into a mild chronic type. This, in turn, may develop into a form of habit chorea or into some hysterical affection. Occasionally chorea seems to disappear, and epilepsy or a severe form of migraine takes its place as adolescence comes on. Death rarely occurs in children. I have seen only 1 case among nearly 400. Death from chorea in adults seems to be more frequent, particularly in *chorea insaniens*, but this is a form of the disease which is certainly rare in this locality. In England the mortality from chorea is about 2 per cent.

Treatment.—The child who is suffering from St. Vitus' dance should be taken from school, and, if the case is severe, from all studies or serious mental application. He should be put to bed early and made to have long hours of sleep. In severe and serious cases the child should be kept in bed all the time, and this treatment has been recommended as one to be universally followed. It is not, however, necessary in the majority of cases. The patient should be allowed to go out of doors, but not to indulge in any violent play or exercise. Every morning the nurse should sponge the back, neck, and chest thoroughly with cool water, splashing it on and rubbing the surface well afterward. In place of this, it is sometimes better to have the child sit on the edge of the bath-tub and then to pour water, at a temperature of about 70° F., from a pitcher so that it will strike the back. The child should then be well rubbed, and either put back to bed or dressed, according to the severity of the condition. The diet should be a plain one, excluding much meat, rich food, and sweets, but it is not necessary to put these patients upon a very rigid regimen.

As regards medicines, there is no doubt that arsenic has a good effect in shortening the disease and lessening the severity of the symptoms, and it is usually best to combine this with a preparation of iron and quinine. The common prescription which is used by myself personally and in my clinic is the following:

℞. Solutionis potassii arsenitis, ʒij ♏xl;
Vini ferri amari, q. s. ad ℥iv.—M.
Sig. ʒj *ter in die.*

If the child has any tubercular taint, I use, instead of the bitter wine of iron, the syrup of the iodide of iron, in the following prescription:

℞. Solutionis potassii arsenitis, ʒij ♏xl;
Syrupi ferri iodidi, ʒv;
Aquæ, q. s. ad ℥iv.—M.
Sig. ʒj *ter in die.*

The arsenic should be increased gradually, at the rate of two drops every second day, until the patient is taking from 10 to 15 drops of the Fowler's solution three times a day. It is rarely necessary to go above 12 drops at a dose, but at times one can advantageously increase it to 20 drops.

The use of zinc in chorea is sometimes beneficial, and it used to be strongly recommended by West. It is a much more disagreeable drug to administer, however, and I seldom use it now, except in cases where the disease has become somewhat chronic and is assuming, perhaps, the characteristics of a spasmodic tic. In such cases the following may be prescribed:

℞. Zinci bromidi, ʒj;
Aquæ, ℥j.—M.
Sig. ♏x *ter in die,* increased by ♏j to ♏xxx *t. i. d.*

Next to arsenic in absolute and demonstrable efficiency are the various coal-tar products—antipyrine, antifebrine, and exalgine. Of these I have found that exalgine is by far the most certain. In a series of cases which were treated with this drug at my clinic I found that in some instances the disease could be really aborted by large doses of exalgine; in others its course was shortened and the symptoms were ameliorated. The plan of giving it is to make powders containing each 2 grains of exalgine and 5 of the saccharated carbonate of iron. One of these is given three times a day the first day; then an additional powder is given each succeeding day until the patient is taking eight powders daily, the maximum daily dose being 15 to 18 grains in children of the age of ten or twelve. It is to be remembered, however, that exalgine is not an entirely safe drug. While I have never known any very serious results from it, I have seen a very alarming condition of cyanosis produced, and it is for this reason that I administer the iron in combination with the exalgine. I also insist that the patients who are taking this treatment shall be put to bed for at least a larger portion of the time, and be seen daily by the physician. While I believe that exalgine is a most effective remedy for chorea, I do not generally use it, for the reason that it may produce grave symptoms, and I feel that it may be, at times, a dangerous drug. The exalgine treatment, therefore, is only reserved for the more serious cases and those which can be kept under control in a hospital or under the close personal supervision of the physician.

There are a great many other drugs which have been recommended in chorea. Among them is quinine, the use of which has been advised by H. C. Wood. He states, however, that in order to get what may be called the "specific" effects it must be given in very large doses, and he reports a case in which the child received 1000 grains in four weeks or about 30 grains a day. I am quite sure that quinine has no special

effect on chorea in ordinary doses. The salicylate of soda has been recommended in chorea, and it probably has some utility in cases associated with decided arthritic manifestations.

THE SPASMODIC TICS.

INTRODUCTION.—It sometimes happens that chorea minor, when not completely cured, leaves as a residuum certain habit spasms which are usually mild in type and eventually curable, but which may perhaps be kept up by some reflex irritation or neuropathic constitution. These spasms involve generally one or two groups of muscles, such as a twitching of the eyes or sniffing of the nose and shrugging of the shoulders.

At other times one sees children develop precisely this same kind of twitching spasm without any previous chorea. The child of nervous habit is noticed, when he gets to be nine or ten years old, to distort his face, to twitch the hand occasionally or the shoulder, and the parents become alarmed at the possible onset of St. Vitus' dance. These spasms last perhaps for a few weeks and then gradually disappear, or they settle down into permanent habits and last for a number of years. These are cases which resemble abortive attacks of chorea on one hand, or incompletely aborted attacks, ending in a chronic condition, in other instances.

In still other cases the child may, without any previous symptoms, develop a peculiar spasm involving a single group of muscles, such as that of the diaphragm or those of the larynx or those of the neck or face. Here there is no particular evidence of a history of Sydenham's chorea; the twitchings are isolated and chronic and persistent.

The above spasmodic troubles have often been classed under the head of "chronic chorea." Some of them do hold a kinship to that disease, but, on the whole, they occupy a very distinct field, both on account of their pathology and their clinical course, and they are best grouped under the head of "spasmodic tic" or *tic convulsif*.

DEFINITION.—The spasmodic tics are chronic disorders showing themselves in the form of quick, electric-like spasms of single muscles or of muscular groups acting *expressively* for the same physiological purpose. The spasmodic movements are violent, and several rapid contractions succeed each other, after which there is a period of rest. They are variously distributed, and are at times associated with other symptoms. The disease is a degenerative one, and lasts for many months or even years. The malady takes various forms, and under it are included the following groups:

1st. *Habit spasm* or habit chorea.

2d. *Spasmodic tic proper*, including forms of wry-neck and mimic tic, retro-collic spasm, so-called chorea of the larynx and of the diaphragm.

3d. *Psychic tic*, including the so-called Tourette's disease, latah, and myriachit, the jumpers, etc.

Etiology.—The tics almost always begin in childhood, and the age at which they start is about the same as that of Sydenham's chorea—that is, between the fifth and fifteenth years. Some of the special forms of tic begin later, as will be shown under Symptoms (p. 513).

The disease attacks boys rather more often than girls, and there is always some hereditary neuropathic history, or, at any rate, the children themselves are of nervous temperament. In the psychic tics the influence is especially marked. Rheumatism has little to do with the malady. The exciting cause is sometimes an attack of ordinary chorea, leaving a habit spasm or a tic as a residuum. In other instances the cause is some excessive strain, overwork, fright, shock, or injury. Masturbation plays a part in psychic cases. Not infrequently the active cause of habit spasm is a reflex irritation, such as disturbance in the refraction of the eyes, nasal stenosis, stenosis of the lachrymal ducts, adenoma of the pharynx, genital irritations, and perhaps, at times, intestinal irritations. Tic proper, however, is not a reflex disease, and on the whole the influences of a neuropathic constitution and of nervous strains are the predominant ones in this group, distinguishing it in many respects from ordinary chorea.

Symptoms.—*Habit Spasm.*—The most common form of habit spasm is a twitching of the muscles of the face, particularly of the orbicular muscles of the mouth and eyes. The child is noticed frequently to close the lids of one or both eyes very tightly two or three times in succession, repeating this every few minutes. At other times he suddenly throws his mouth wide open, jerks the head back, and perhaps protrudes the tongue, or perpetually gnaws the lips. Another common form of habit spasm is a constant sniffing of the nose. This is often started, perhaps, by some chronic catarrhal condition that is kept up by the neuropathic tendencies of the patient.

Children with habit chorea often jerk the shoulders, and occasionally the arms and legs, or make some curious gesticulation or grotesque pantomime.

Spasmodic Tic Proper.—When the spasm gets definitely located upon one or two nerve centres it is more often spoken of as a *spasmodic tic*, and, clinically, we find names given to a number of different kinds of tic. A rather common type is that known as "chorea of the larynx" (laryngeal tic). In this the twitching spasm affects the muscles of respiration and of voice. As a result the patient makes at more or less regular intervals loud expiratory noises, like the bark of a dog or like the short, sharp cry that ushers in an epileptic spell. Sometimes the noise assumes the character of an articulated word, as in the case of a girl who was under my treatment, who continually repeated "Hoy, hoy." In these cases the noises may be kept up almost constantly throughout the waking hours at the rate of ten or twenty a minute, giving neither the patient nor friends any rest. The spasmodic movements increase when the patient is excited, and are much less when he is isolated. They cease during sleep. No amount of voluntary control or punishment affects this disturbance, except temporarily.

Spasmodic tic also affects, at times, the muscles of the trunk, causing the head and body to be suddenly pulled backward a little. As the patient sits in a chair the head and shoulders are pulled against its back,

and as the patient walks his gait is interrupted by this backward twitching of the body.

Wry-neck.—There are two forms of spasmodic tic which are seen in middle and late life, as well as during childhood and adolescence. These are *wry-neck* and *mimic facial tic*. The term wry-neck is applied also to a tonic spasm of the muscles supplied by the spinal accessory and cervical nerves, and this condition is due either to a congenital disorder, to muscular rheumatism, or to irritation of enlarged glands or cervical caries. Leaving out these cases of tonic and symptomatic wry-neck, however, there is a large number of cases of twitching spasm involving the neck muscles which belong properly under the head of spasmodic tics. They are really a kind of habit spasm; their seat is in the brain, and French writers (Brissaud) speak of them as *mental torticollis*. While in childhood and adolescence wry-neck is often only a kind of psychic or habit spasm, in later life it assumes the character of a more serious degenerative neurosis, and is perhaps associated with some organic change.

Facial Tic.—The above statements apply also to facial or mimic tic. This usually develops after the period of middle life, and is sometimes seen associated with *tic douloureux* or after an attack of hemiplegia. It is a rather frequent symptom in aged people, especially women, and there too is a mark of some real degenerative process. Facial tic occurring in adult life is sometimes, like the other tics, a reflex disturbance, but this is not often the case. The disease is generally described separately, as is torticollis, on account of the fact that, while the clinical symptoms are very uniform, the cause and pathology vary very much. It is my opinion that, both clinically and pathologically, mimic tic can be brought in closer relation with the group of spasmodic tics than with any other class.

Gilles de la Tourette's Disease (*Myospasmia; the Jumping Disease*).—There is a form of spasmodic tic which is characterized by sudden convulsive movements, accompanied with inarticulate, obscene, or profane ejaculations (coprolalia), and sometimes with echoing of words heard (echolalia) or with imitation of movements suggested (echokinesis). This malady, which is sometimes called "Gilles de la Tourette's disease," has been observed in various parts of the world, where it receives different names. The persons affected in Maine, Canada, and certain parts of the Northern and Central States are called "jumpers." In Siberia the affection is called "myriachit." In Java and in the East Indies, amongst the Malay population, it is called "latah." It is endemic, therefore, in these locations, but it is occasionally seen in the populous centres of Europe and of the United States. It begins, as a rule, in children between the ages of five and sixteen, and affects men oftener than women. There is a neurotic family history, and the disease is decidedly an hereditary as well as a family one. The exciting cause is probably, in most instances, very slight. Perhaps some powerful emotion may be sufficient or some acute disease. Tickling has been said, in the case of the "jumpers," to be an exciting cause; masturbation is also mentioned. The affection seems to prevail amongst rather inferior races or those portions of strong races that have a low grade of nervous vitality. It is a disease of a neurasthenic people.

It begins with attacks of sudden motor explosions affecting the head, face, and upper extremities first, then involving the body. The patient while sitting quietly will suddenly throw the arms out or the head back, jumping up from the chair high into the room, and perhaps uttering some cry. In the cases of latah and myriachit the patients will often go through a sudden act which they are told to do, such as taking off their clothes, getting up, or sitting down. They have to do this whether they are willing or not, and sometimes while they are loudly protesting against it. In mild cases, especially those seen in this region, the more violent movements are not often present, and the child simply has severe choreic twitchings of the face or arms, sometimes jumping movements, which in many respects resemble, so far as the motor phenomena are concerned, the ordinary spasmodic tic. Besides these motor phenomena, however, the patient utters explosive sounds, uses foul or profane words, sometimes repeating the same word half a dozen times; at other times uttering a phrase full of obscenities (coprolalia). The attacks occur at any moment when the child is at school or at table amongst his family or in strange surroundings, and may lead to very embarrassing situations. In place of the profane or obscene words the child sometimes simply repeats the last words or phrases of the person to whom he is talking. This is known as echolalia. Tourette records the case of a young woman who had retired to rest one night, when a dog began to bark under her window. The unfortunate patient echoed the bark, which the dog took up in turn, and against her will she was kept barking the greater part of the night.

Aside from the motor and mental symptoms just referred to, the patients are in fairly good health. Their sleep, appetite, and general nutrition are not seriously impaired. Their mental condition is also, as a rule, good, though they show evidences of nervousness and of neuropathic taint, and are in some instances backward morally and mentally. The disease is not specially associated with hysteria, and is more allied to a mental disorder than it is to the neuroses. The malady is a chronic one, beginning insidiously, lasting for years, sometimes for a long lifetime.

Prognosis.—The prognosis of habit spasm is almost always good unless the case is very much neglected. As a rule, children who have the mild types of habit spasm get well as they reach the age of fifteen or sixteen; if they pass that age, however, without being cured, it is apt to continue more or less through life, there being often long periods of comparative rest from it.

The prognosis of the spasmodic tics proper, such as affect the neck muscles, face, and trunk, is much more serious. When the malady comes on in children or in early youth the outlook is fairly good, the majority of cases getting well; but where it develops after maturity, as is often the case with tic of the spinal accessory or face or of the upper cervical nerves, the disease is extremely obstinate, though it is not absolutely incurable. It rarely shortens life. Psychic tics are rarely curable.

Pathology and Pathological Anatomy.—While chorea minor is essentially an irritative and, one may almost say, inflammatory disease, the result of some infective or humoral poison, the various forms of spasmodic tic are the expression of a degenerative process. In some

cases, such as those which are the sequel of chorea minor, this degeneration is simply a residuum of minute foci of irritation which caused the chorea. In other cases the degenerative process is congenital, and it means that the child was born with certain neurons defective in their functional work. The spasmodic tics are therefore to be classed with degenerative neuroses.

In the case of spasmodic tic the seat of the malady is, in the classical types of the disease, the cortex of the brain, and the trouble is essentially one involving the higher centres of the brain. The French observer, Brissaud, makes a very sharp distinction between spasmodic tic proper and that which he calls "spasm," but which, in English, would be represented by the phrase "twitching spasm." The symptomatic twitching spasm of chorea, he asserts, is always a reflex disturbance or at least an irritation of some part of the *spinal reflex arc*. The twitching spasms of tic, however, are due to an irritation of the *psychic reflex arc*, represented by the sensory neurons, associative neurons, and the two motor neurons. This view is correct for most cases, but there are certain kinds of twitching spasms which simulate tic clinically, such as the facial spasms of old people, and which may be due to lesions located lower down than the cortex.

As for the pathological anatomy, cases have been reported of tic due to sarcoma of the brain (Moos), to aneurysm of the vertebral artery at its junction with the basilar (Schültz), and to lesions of the convexity of the hemispheres, as in the cases of Chipault, De Brou, Berkeley. In the spasmodic tic which is sometimes associated with tic douloureux there is degenerative change in the trigeminal nerve or its ganglia or in the artery supplying it. The peripheral nerves, however, as a rule, are not involved, nor are the motor nuclei.

Diagnosis.—The characteristics of spasmodic tic have already been described under the head of Chorea. The essential point in their symptomatology, as was pointed out by Trousseau, is that the movements are quick, lightning-like, and expressive. Spasmodic tic is a purposeful twitch, while chorea is a purposeless, inco-ordinate twitch of a single muscle perhaps or of muscular groups. The distinguishing factors in etiology, course, and pathology have been dwelt upon sufficiently. Psychic tic is one which has distinguishing features so marked that it only requires the physician to be sure he is not dealing with a simulator or with hysteria.

Treatment.—The treatment of habit spasm is very much the same as that for chorea minor. The disease is often simply the expression of anæmia and malnutrition, overwork, or overtaxing of the eyes; hence measures to relieve these things should be taken. Such measures have already been indicated under the head of the Treatment of Chorea (p. 510). Sources of reflex disturbance from the nose, throat, and teeth should be investigated.

In the spasmodic tics, which comes on idiopathically without any previous history of chorea, the treatment has to be somewhat different. In these cases the neuropathic constitution of the patient has to be considered, and measures for strengthening the nervous system are indicated. There is, in these cases, often a very great need of moral influence, and the same measures which are indicated in hysteria are many times use-

ful; that is to say, the child needs to be isolated, or at least removed from its parents, and placed either with strangers or in an institution for a time. This applies particularly to cases of chorea of the larynx, facial tic, and irregular forms of tic occurring in the young. In wry-neck and retro-collic spasm I have found that patients do best when placed in a rest-cure, where they should be given massage, electricity, and the usual adjuvants of that treatment. As an adjuvant I have found the use of the Sayre apparatus for stretching the neck gives benefit. The patient should be placed in a chair and the apparatus adjusted to the neck and shoulders. He is then lifted from the ground and held suspended for, at first, one minute, the time being increased to three or four minutes daily. In other instances it is better simply to place the patient in a chair and pull upon the neck to the extent of about twenty-five pounds, the patient being kept in this position for five minutes. During this time gentle muscular movements are made.

In rare cases cure has been secured by the hypodermic injection of large does of atropine, beginning with $\frac{1}{120}$ of a grain, and increasing it gradually until a tenth of a grain or even more is taken once daily. Sometimes a pill containing $\frac{1}{100}$ of a grain of hyoscine hydrobromate is helpful. In cases where the disease is of long standing or the patient is in middle or advanced life the use of opium in moderate doses has been recommended, but it should be given very carefully. The fluid extract of conium has been used with some success, temporarily at least. The patient is given 5 drops of fluid extract of conium three times a day, and this dose is increased a drop at a time until 25 or 30 drops, or even more, are taken three times a day. Constitutional effects, such as drooping of the eyelids and great muscular weakness, should be closely watched for. Furthermore, the physician should see to it that his conium is a good article. In some instances I have found the preparations to be absolutely inert. The fluid extract of gelsemium may be used in the same way. The bromide of zinc, in doses of 2 grains increased to 10, three times a day, is sometimes useful in younger patients. This drug is apt to disagree with the stomach, and its administration must be carefully watched.

Too much importance cannot be laid upon the necessity of beginning treatment early in the disease. I must confess to having very little confidence in the use of drugs of any kind in this affection, especially when the patients are adults or the disease is of long standing.

It has long been considered very important to look out for reflex irritation in cases of spasmodic tic. In mild cases, in children, there is no doubt that much good can be obtained by examination of the eyes, correcting any deformities there, and by seeing that there are no serious disorders of the respiratory and lachrymal passages, no disease of the teeth, no irritation in the ears, no genital or gastric disturbances, and no cervical caries. In the older cases very little benefit results from these measures. In a facial spasm one can sometimes temporarily lessen the severity of the trouble by dropping a 2 per cent. solution of cocaine into the eye.

Mechanical measures have often been resorted to for the relief of cervical spasms, and some ingenious instruments have been devised, of which pictures can be found in the current catalogues of surgical instru-

ment-makers. I have, myself, never been able to get any benefit from such appliances, although occasionally such benefit is reported. Surgical measures have often been resorted to, and have been much-lauded in certain quarters. Stretching the nerves was at one time attempted, but this measure may be considered entirely useless. Resection of the nerve is only practicable in those forms of tic involving the spinal accessory and cervical nerves. In spasm affecting the spinal accessory the best results appear to have followed the resection of this nerve. The simple cutting or stretching of it does not do any good. In most cases, however, if one nerve is resected, the spasm soon involves other nerves, and the final result is unsatisfactory. The resection of the posterior branches of the upper third cervical nerves has been performed by Keen of Philadelphia, by R. H. Sayre of this city, Walton, and others with slight success.

Local applications sometimes produce temporary benefit; thus freezing the skin over the facial nerve and injecting cocaine over it will sometimes relieve the spasm for a time. The application of the galvanic current has certainly a palliative effect, but hardly anything more. A stabile current should be given directly over the affected muscles for a period of about ten minutes.

Progressive Choreic Tic.

There is a form of spasmodic disorder which starts in childhood, beginning usually with the symptoms of chorea; it gradually progresses, taking upon itself the character of spasmodic tic, involving generally, first, the neck muscles, then the face or arm, and finally the whole body. Associated with twitching spasms, tonic spasms also appear toward the end, when the patient may even suffer from opisthotonos. The disease is steadily progressive and eventually fatal.

Etiology.—The patients are most often boys, and the malady begins during the choreic age—*i. e.* between five and twelve. There is generally a neuropathic family and personal history. No special association with rheumatism has been noted. The exciting causes are much the same as those of chorea—that is to say, mental shock, injury, infectious disease, overstrain, and malnutrition.

Symptoms.—The disease begins with choreic twitchings in one side of the face, then in the neck, and then in the arm. These choreic twitchings extend slowly with some remissions. In four or five years both sides of the body are affected with the twitchings, and symptoms which resemble torticollis may be noticed. This torticollis at first involves the neck muscles supplied by one spinal accessory. After a time other cervical muscles are involved, and the head is at times twitched to one side, and again pulled back upon the neck or forced forward. In a few years more the choreic movements and the other tonic spasmodic movements have invaded the muscles of the trunk and all extremities. The patient is now never left at rest except during sleep. The severest movements, however, still centre about the neck muscles, and these cases are often classed as cases of chronic torticollis. But they have, besides the symptoms of torticollis, facial tic, violent choreic movements of all four extremities, with, at times, tonic spasm of the trunk muscles. On account of this wide extent

of the spasms, especially in cases where the general spasmodic movements are very marked, the diagnosis is sometimes made of chronic chorea. The disease having reached this point, the patients often remain without any great change for a number of years. They do not suffer in their general nutrition very much; they have no great amount of pain, but there is at times great fatigue incident to the continual motion. They may also have, at times, some actual muscular pains, which are perhaps the result of the constant strain put upon them. Mentally, these patients, so far as I have seen them, have been fully up to the average in intelligence, and they do not show the progressive deterioration of hereditary chorea. Occasionally they may suffer from hysterical or even epileptic convulsions.

As the presentation of this complexity of symptoms has not been made before, I venture to insert briefly the notes of a few of my cases:[1]

Case I.—A boy, Frederick M——, twelve years of age, of good family history, came to my clinic in the fall of 1895. He was well-developed, bright, and intelligent for his years. At the age of five years, without any cause that could be discovered, he began to have some choreic twitchings in the right side of the face, then in the neck, and then in the arm. These continued to increase gradually, though there were some remissions, and the progress was very slow. His speech was clear when seen by me, and his intelligence good; he had no signs of cardiac or other organic disease. He suffered from choreic movements which affected the fingers, especially of the right hand, but also, to some extent, the muscles of the arm and forearm and the facial muscles, especially the orbicularis. In addition to the chorea there were tonic spasms of the muscles of the neck, causing the head to be turned over toward the left side. In other words, the boy, starting with certain choreic movements in the face and arm, ended in developing symptoms of a spasmodic torticollis.

Case II.—A boy at the age of ten years had a severe attack of acute rheumatism. From this he recovered. At the age of fourteen he began to have chorea, the attack resembling apparently the ordinary chorea of Sydenham. The attacks of chorea were repeated every two or three years, but between them he was never entirely well, and with each return of the attack there was a gradual involvement of the muscles of the neck and trunk, so that with the chorea he developed symptoms of wry-neck, and at times spasms of the erector spinæ which produced almost an opisthotonos. The tonic and choreic spasms increased in violence, and he finally died. At the post-mortem I found a very marked meningeal thickening of the convexity of the brain, with degenerative changes in the pyramidal cells of the cortex, especially in those involving the legs, arms, and trunk. The case has been reported in detail elsewhere (Figs. 60, 61).

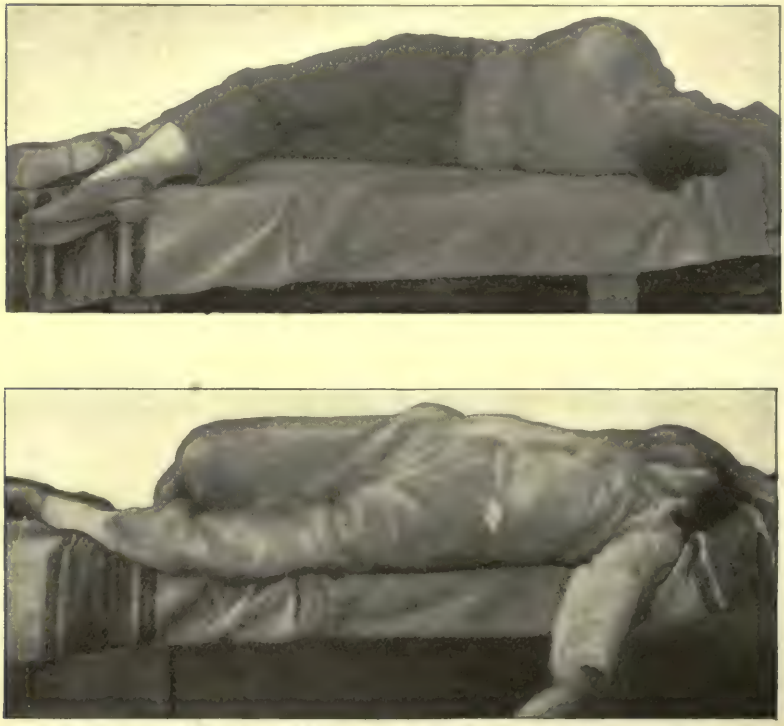
Case III.—Kate K——, aged thirteen, was attacked when ten years old with a severe pain in the left side and a sensation as though she were dying. She was intensely frightened, but had no spasmodic attack at that time or any unconsciousness. Two or three days later she began

[1] Sachs in his *Text-Book of Nervous Diseases of Children* describes a form of "hereditary chorea without mental deterioration" which resembles the above malady; but I have not found any direct hereditary influence, and the spasms are not entirely choreic.

to have choreic movements of the eye muscles, of the left side more particularly. These movements have continued, with very slight intermissions, ever since, and extended gradually, so that when seen by me, three years later, she had choreiform movements of the shoulders, neck, head, and face. These movements were accompanied with tonic contractions of the muscles of the neck, so that she presented the appearance of a person in the chronic stage of torticollis. In addition to that, she had a slight tremor involving the right hand. This became much more marked when she was excited. In this case also there seems to have developed a form of torticollis on the basis of an ordinary attack of chorea minor.

DIAGNOSIS.—These cases are to be distinguished from chronic chorea by the fact that they have not only the choreic spasms, but also the typical purposeful spasms of tic, such as are shown in wry-neck and in facial tic, and by the fact that they have also tonic spasms involving the muscles of the trunk, and, to some extent, those of the neck and extremities. The disease differs also from spasmodic tic proper, first in

FIG. 60.

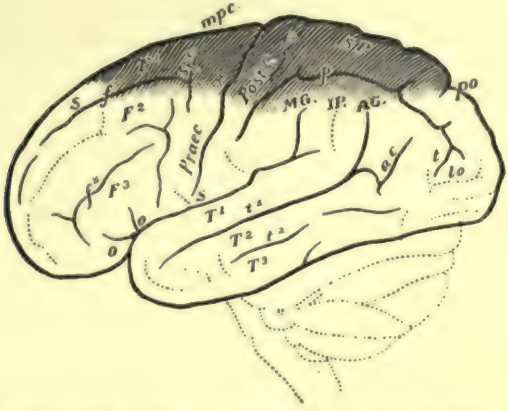


Choreic tic, showing attitudes assumed during the spasmodic movements.

the primary choreic nature of the symptoms, there being at the onset, in some instances, very little difference in the symptoms from an ordinary case of chronic chorea. In the second place, the spasms are mixed in character up to the end, involving the choreic twitchings, the true tic-like spasms, and tonic spasms.

PATHOLOGICAL ANATOMY.—In two cases in which I made an autopsy I found serious degenerative changes, chiefly in the motor area of the cortex of the brain. There was a thickening of the pia mater, and the nerve cells showed various stages of degeneration; the blood-vessels were less markedly diseased[1] (Fig. 61).

FIG. 61.



Progressive choreic tic, showing area of lepto-meningitis.

PROGNOSIS.—The patients may live many years despite their suffering. Two of my patients have died, one after twenty years, the other after ten years, of illness. After the severe symptoms have set in the disease steadily but slowly progresses. In the first few years there are long but not complete remissions.

TREATMENT.—In the early stages, rest and the ordinary measures applicable to chorea and spasmodic tic are indicated. Later, little can be given but symptomatic treatment.

FORMS OF CHOREA (SO CALLED) NOT DESERVING THAT NAME.

In the history of medicine there has been observed a number of different groups of symptoms to which ambitious authors have attached the name of *chorea* with some modifying adjective.

1. **Fibrillary chorea** is a term given by Morvan to a myoclonic affection characterized by fibrillary contractions, which appear first in the calves and the posterior part of the thigh, extending thence to the muscles of the trunk, and then to the upper limbs, but always respecting the muscles of the neck and face. Morvan tries to distinguish this from the myoclonus of Friedreich on account of its limitation to the limbs; also because no displacements of the body are caused by the contractions, and the attacks are not accompanied by sweating or vasomotor symptoms. Despite this, however, there is no doubt that fibrillary chorea is only one of the forms of paramyoclonus.

2. **Rhythmical chorea, malleatory chorea, saltatory** and **rotary chorea** are forms of rhythmical spasm which are characterized by certain definite co-ordinative acts performed rhythmically. They all belong to the group of hysterical affections, and not to chorea at all.

[1] *Journ. American Med. Sciences*, Jan., 1894.

3. "**Electric Chorea**" (*Henoch's, Dubigni's, Bergeron's Chorea*).—Under this name Henoch described cases which are now recognized as belonging properly to the group of spasmodic ties or else to myoclonus multiplex. The term is often used also loosely to indicate a very exaggerated active form of ordinary Sydenham's chorea. *Electric chorea* is also the name given by Dubigni, an Italian physician, to a form of chorea which occurs chiefly in the northern part of Italy. The descriptions of the malady show that it is really not a form of chorea, but some serious infection or toxic disorder causing, probably, organic changes in the nervous centres. It occurs in childhood as well as in advanced age. The chief symptoms consist of violent spasmodic movements of the muscles of the extremities, including the neck and head. These movements continue with great severity, and after some months there is wasting of the muscles with paralysis of some of the limbs. Epileptiform seizures occur. In the course of a year or more the patient may become paralyzed entirely and confined to bed, but the majority of cases die within a few weeks or months, and the disease is usually a fatal one. The patient during its course suffers from pain and some elevation of temperature.

The term *electric chorea* has also been used by M. Bergeron, a French physician, who in 1880 described a disorder which he called "electric chorea." It was characterized by violent and sudden rhythmical spasms; the disease ran a uniformly favorable course, and seems not to have any true relation to chorea.

Hereditary Chorea, or Huntington's Chorea.

Hereditary chorea is the name given to a very rare chronic disorder characterized by choreic movements and a progressive mental deterioration, ending eventually in dementia. It is always hereditary in character, and depends upon a progressive cortical degeneration of the brain. The disease was first described by an American physician, Waters of Long Island, in 1842. Later it was written about by Gorham and Lyon. In 1872, Huntington gave a more complete description of it, and his name has been connected with the disease.

Etiology.—The affection is always hereditary, and is transmitted from generation to generation; in many cases it can be traced back through five or six ascendants. It does not attack all members of the family, some escaping, and one generation may, to a large extent, escape altogether. It is transmitted oftenest through the mother.

The disease is found in this country chiefly in Long Island, New York, Connecticut, New Jersey, and Pennsylvania. Cases have been reported in Germany, France, and England. The symptoms rarely begin before the twenty-fifth or thirtieth year, though there are instances on record in which it occurred before the age of twenty. The males and females are about equally affected, and there seems to be no particular exciting cause.

Symptoms.—The disease is characterized by two sets of symptoms, motor and mental. The motor symptoms consist, first, in slight twitching or jerking movements of the facial muscles or of some extremity of the body. These movements suggest, to some extent, the choreic or

habit spasm, but approach more nearly the movements that characterize spasmodic tic than they do those of chorea. The patient makes grimaces or grotesque gestures, his gait is often "high-stepping" or loping, and he walks with a peculiar to-and-fro motion of the body. At times he makes awkward jerking movements of the hands and feet. With this there may be simple choreic movements of the facial muscles or eyes. In some cases the choreic movements are very slight. In all cases the most serious symptom of the disorder is the marked mental disturbance, the mental symptoms developing with the motor. Usually there is first a gradual loss of memory, which finally becomes so great that the patient is unable to follow his pursuit. This, after a time, is associated with attacks of excitement and some mental irritability, but rarely with any active mania or with homicidal or suicidal manifestations. In some instances the patients become acutely insane, even early in the disease. The general character of the mental symptoms is depressive. The patients are apathetic and inclined to sit by themselves, and show evidences of a mild type of

FIG. 62.

Physiognomy of hereditary chorea in the fifth year of the disease.[1]

melancholia (Fig. 62). There are occasional delusions, but none that are very fixed or persistent. The patient rarely suffers from any serious muscular weakness or sensory troubles, and his bodily condition is good, so that the disease is a very chronic one, and some patients who are attacked at the age of thirty have lived for twenty or more years. In most cases the duration of the malady is ten or fifteen years. This seems to vary somewhat in different families.

PATHOLOGY AND PATHOLOGICAL ANATOMY.—A sufficient num-

[1] Writer's case, from the *Journ. of Nerv. and Ment. Dis.*, Sept., 1895.

ber of autopsies have been made to enable one to speak with some certainty as to the nature of this curious disorder, which belongs in strictness to the province of teratology. Hereditary chorea depends upon a congenital defect in the structure of the brain. The patients are born with nerve cells which have not the capacity for living more than a certain number of years. A short time after adolescence these cells begin to degenerate and symptoms of chorea and dementia set in. Post-mortem examination shows that the seat of the disease is in the cortex of the cerebral hemispheres, and that here it especially affects the cortical motor centres. In a careful study of the relative thickness of the cortex of the brain, made in a case of my own,[1] I found the central convolutions on both sides very markedly wasted as compared with the convolutions of the frontal and occipital lobes. Microscopical examination showed that the degenerative process, though most marked in this area, was not evenly distributed, but occurred in patches throughout the affected parts. This explains, perhaps, the irregular motor symptoms, the twitchings, and the other spasmodic movements. The brain shows no signs of inflammation or proliferation of connective-tissue cells, no exudation or accumulation of leucocytes. The process seems to affect primarily certain groups of nerve cells, leading to their decay and death. In the case observed by myself the cells of the second and third layers were especially affected; they stained badly, the process had, in many instances, dropped off, the outlines were irregular, there were pigmentation and a gradual disintegration apparently of the cell body. These changes correspond, in general, with those found by other observers, but my own case represented a less advanced stage of the disease. In the last stage very much severer degenerative changes, with meningeal thickening, are observed.

Diagnosis.—The diagnosis is made very largely upon the family history of the case and the presence of progressive mental changes. The fact that the disease is never developed in childhood enables one to distinguish it more easily from ordinary chorea, and at once renders the observer suspicious of some more deep-seated malady. The peculiar expressive, purposeful character of the spasm, such as we see in tic, is characteristic also of the disease.

Prognosis.—The disease is steadily progressive and ultimately fatal. Nothing has yet been discovered which can long stay its course, although there is no doubt that under favorable circumstances the progress is made slower.

Treatment.—The treatment of this disease really belongs to the sphere of preventive medicine. Choreic families should not be allowed to marry, and, above all, not to intermarry. As for the treatment of the malady itself, we can only delay its progress by having the patient live a quiet life, removed from excitement and stimulating influences of all kinds. No drugs produce any special effect. In one case I trephined the patient, with the result that there was an improvement for three or four months, and then the patient relapsed to his former condition.

[1] *Journ. Nerv. and Ment. Dis.*, Sept., 1895.

TETANY.

By CHARLES L. DANA, A. M., M, D.

Definition.—Tetany is a functional nervous disease, usually subacute in course, and characterized by attacks of bilateral tonic spasms, which last for hours or days, and which have a tendency to recur. The attacks affect especially the extremities, but in rare cases involve the trunk also. They are accompanied almost uniformly with sensory disturbances, such as paræsthesias and anæsthesias, and at times with pain. The disease is one of toxic or infectious character. It occurs in infants and young children with especial frequency, but is often seen in adult life also. It is associated with stomach and intestinal disorders and pregnancy, and appears sometimes as a prodome or in the course of the acute infectious diseases.

Synonyms.—The synonyms for the disease are interesting, as showing, in a measure, its clinical features. It was called by Comte *tetanilla;* by Dance, *intermittent tetanus;* by Trousseau, *rheumatismal contracture of infants.* Other observers have spoken of it as *partial tetanus* (Cruveilhier), *an essential contracture*, a *tonic occupation cramp* (Benedikt) and *an idiopathic muscular spasm.*

History.—The disease was first described by Steinholm in 1830, and by Dance in 1831. Frankl-Hochwart cites a passage in Hippocrates which shows that symptoms resembling those of tetany were observed by the Greek physicians. References to it also are found in the medical writings of the seventeenth and eighteenth centuries. It is only in the last twenty years that thorough and scientific observations have been made upon the symptomatology and pathology of the affection. Such observations have been made chiefly by German and Austrian writers. Until recently there has not been much attention paid to it in this country. In 1894, Griffith, in an article on "Tetany in America," was able to find only 72 reported American cases. A good many have been added to this list since that time, especially by New York physicians, notably Lewis Smith, McAlister, Vaughn, Lewi, Peckham-Murray, and Kraus. The disease has been in the last few years rather frequently observed in this city.

Etiology.—Tetany occurs with especial frequency in the second, third, and fourth years of life. It is seen again at the time of puberty and adolescence. Its rate of frequency then decreases, and it practically never occurs after the fiftieth year. It affects males oftener than females in the young, and it occurs most often in the working-classes and in those whose conditions of life are such as tend to produce malnutrition. While rare in this country, it has of late been seen rather often amongst the Italians of New York City who live in the poorer quarters. Aside

from these predisposing causes, tetany is associated with four different conditions, which may be looked upon as the exciting causes:

1. *Infectious and Toxic.*—It attacks, in the form of epidemics, those who are apparently healthy. When it occurs in this way it affects especially the laboring classes, almost always men, and appears especially often in the winter and early spring. In Germany, at least, shoemakers, tailors, and cabinet-makers suffer from the disease especially, and it attacks these classes mostly between the ages of sixteen and twenty-five. It has been observed epidemically in asylums and prisons (Althaus). Tetany develops also as one of the advance symptoms of infection by an acute disease, such as cholera, typhus, measles, scarlet fever, erysipelas, rheumatism, malaria, and influenza. This is noted especially in children.

Tetany is produced by poisoning with ergot, alcohol, carbonic oxide, and chloroform.

2. *Gastro-intestinal and Auto-toxic.*—Tetany occurs in connection with disorders of the stomach and intestines. In children such disorders are generally diarrhœa, constipation, and the presence of worms; in adults, gastric disturbances, associated with dilatation of the stomach, are especially frequent causes. According to Bouveret and Devin, gastric dilatation and hyperacidity, combined with the ingestion of alcohol, favor the development of the disease.

Tetany has a tendency to occur in pregnancy, and here an auto-toxæmia may be suspected. In children malnutrition and rickets are very often present.

3. *Thyroid Tetany.*—Tetany may be artificially produced by removal of the thyroid gland. This has been observed both in man and animals. The disease has been known to complicate exophthalmic goitre.

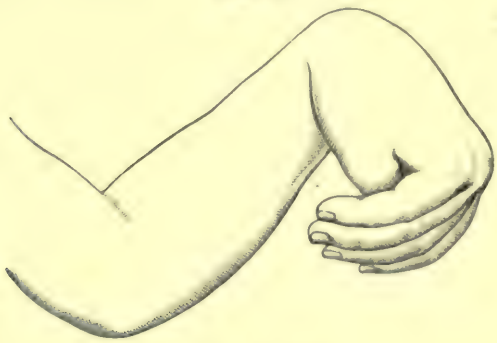
4. *Hysterical and Symptomatic Tetany.*—Tetany in some of its adult forms may be simulated by hysteria (Peckham-Murray), and some French writers have regarded the disease as an hysterical manifestation (Tourette). I have seen the disease in an hysterical woman who was addicted to the excessive use of nux vomica.

In general, it may be said that tetany in children is produced by malnutrition, rickets, digestive disturbances, and infections. In adults it is generally produced by infections and toxæmias of one kind or another, combined also with bad conditions of nutrition.

Symptoms.—The disease may begin rather suddenly with spasmodic contractions of the extremities, but usually these symptoms are preceded by prodromal sensations of prickling, numbness, and heaviness of the arms and legs; after which the muscular contractions set in. These contractions affect most often the ends of the upper extremities. The thumb is adducted into the palm, the fingers flexed at the metacarpo-phalangeal joints and extended at the other joints, producing a condition known as the "accoucheur's hand." This form of contraction, however, is not always present. Sometimes the fingers and thumbs are all tightly flexed, as shown in Fig. 63. The wrist is flexed upon the forearm and the forearm flexed somewhat on the arm. The upper arms are generally held tightly to the trunk. The thighs and knees are extended, the toes are flexed, and the foot is inverted. The contractions are tonic and the muscles are hard. Some pain may be felt in connection with the

contractions, but this is not always present. In mild cases the spasms affect only the arms, and perhaps the legs. In children spasm of the glottis almost always occurs. In severe cases the trunk may be involved and a condition of opisthotonos may be present; in fact, the whole body may be made for a time rigid, as in tetanus; even the muscles of the face and jaw may become contracted, and the patient in these severer types presents very much the appearance of a case of true tetanus; but trismus is rare, and occurs only late in the disease. The attacks last from a few minutes to several hours, or even for many days. They are not accompanied with fever or with any serious disturbance of the other bodily functions. The seizures remit for several days or weeks, and then return, and the patient may suffer from attacks of tetany in this

FIG. 63.



The hand in tetany.

way for months or even years, though this is most unusual, the duration being commonly only a few weeks. During the course of the disease epileptic attacks have been known to occur.

The characteristics of an attack are best understood by the following description of a case of tetany in pregnancy reported by H. M. Thomas:[1] "The attacks, the patient said, begin with a tired, aching sensation of her hands, which is soon followed by the fingers becoming stiff and drawing shut, the feet also becoming stiff and drawn. In a severe attack the pain is intense, and the fingers are so tightly closed that the nails cut though the skin, the arms being stiff and held close to the chest, and the hands blue and swollen. At times the spasm spreads to many other muscles; the whole of the body becomes stiff and the face and eyes are drawn, and even the muscles of the larnyx are affected, stopping respiration for a moment or two and causing her to feel as if she were going to strangle. The patient has never lost consciousness in an attack. The duration of the attack varies very greatly, from a few minutes to several hours, or even days, and she has never found anything that seemed to shorten them, although the pain can be controlled to a certain extent by repeated doses of morphine."

In another case, that of a man aged forty-eight, reported by J. P. C. Griffith, "the feet would be drawn into dorsal flexion, the knees

[1] *Johns Hopkins Bulletin.*

and hips extended and stiff, the thumbs bent into the palms and the fingers closed tightly over them, the wrists flexed, the elbows flexed and drawn to the sides, and the head held stiff. The jaws would be tightly closed, or sometimes could be opened and not shut again. The breathing would be painful and difficult, and the man feel as though each moment would be his last. The attack would come on slowly and increase in severity. At its height the spasms would be somewhat remittent, lasting some minutes, and then partially relaxing, while the greatest intensity of the pain shifted from one part of the body to another. The whole duration of an attack would be four or five hours."

In another case, that of a single woman of twenty-six, reported by Henry Hun, the patient after exposure to a rain-storm began to feel a stiffness in her right hand. This continued, and "two weeks later the muscles of the calf of the leg became cramped and hard, continuing so for about half an hour, during which time she was unable to stand. The next day the spasms returned, and subsequently they recurred more and more frequently and attacked the arms also, and finally involved all the muscles of the body. These spasms have, from the first, presented an intermittent character, but during the past six or eight months, even in the intervals of the spasms, the muscles do not completely relax, but continue stiff. At times, especially when in bed in the morning, she thinks that she is perfectly well, but very soon the rigidity comes on. The acute attacks of spasm are usually confined to her arms and legs, but on several occasions the muscles of respiration have been slightly involved. She has no pain in any part of her body, although the acute attacks of spasm are somewhat painful. She always feels very hot, and even in the coldest weather likes to sit without any extra clothing by an open window."

These individual records show better than any general description the peculiar clinical characteristics of the disease.

Tetany, however, has, in addition, certain special symptoms that are peculiar to it and which need separate notice.

The first of these is the peculiar irritability of the motor nerves. This is so marked that striking on a motor point brings out a muscular contraction which may remain for several seconds. When the motor point of the facial nerve is struck, for example, a spasm of the muscles of that side of the face is produced, which is known as the "facial phenomenon" or "Chvostek's symptom." Furthermore, by pressing upon the artery of the arm or leg, and sometimes by pressing upon one of the nerves of either extremity, a tonic contraction can be brought out in the muscles of that extremity. This is known as "Trousseau's symptom."

Second, the electrical irritability of the muscles and nerves is increased, especially to the galvanic current (known as "Erb's symptom"). A negative-pole closure contraction is brought out by a very weak current of a fraction of a milliampère, and if this current is made stronger, the contraction becomes tonic. The positive-pole opening may also be tonic, and there may be also an opening tetanus with the cathode. Thomas describes a peculiar electrical condition in which the cathode, when placed over the nerve, produced, first, fibrillary and then tonic contractions in the muscles supplied by the nerve, but when the anode was

substituted for the cathode, no contraction took place. On the contrary, even a strong current of five or seven milliampères could be applied without producing any tetanus. This peculiar reaction has also been noticed by Bechterew.

A third characteristic of tetany is the peculiar increase of irritability of the sensory nerves. This is shown by pressing upon them, when sensations of prickling and formication will be felt along their course (known as "Hoffmann's symptom"). There is an increase also in the electrical sensibility, shown in the passage of very weak galvanic cur-

FIG. 64.

Showing facial spasm on the right side, the result of percussing the cheek at the point of exit of the facial nerve (Chvostek's symptom) (Thompson).

rents. The auditory nerve also reacts much more easily to galvanic stimulation than normally.

PATHOLOGY AND PATHOLOGICAL ANATOMY.—There is no known anatomical basis to the disease, and it is only assumed that there is probably a congested condition of the gray matter of the spinal cord and medulla. There is hardly any doubt that underlying this there is some infection or some auto-toxæmia.

Post-mortem examination of one case showed the presence of a cysticercus in the right supramarginal gyrus. In another case an effusion of blood was found on the anterior surface of the cauda equina and the upper cervical cord. Other examinations have been negative, except as showing dilated stomach or evidence of some toxic or infected condition. In one case a syringomyelia was found.

The fact that tetany may be produced artificially by the removal of the thyroid gland would indicate that in some cases, at least, a defect in the activity of this gland may be a factor in the disease.

DIAGNOSIS.—The disease is usually easily recognized, because it closely resembles tetanus, and yet has not the persistence or serious character or extent of that disease. The spasms are usually limited to the extremities, and are of moderately short duration, and there is no violent or painful opisthotonos or trismus. The spasms in tetany also begin in the extremities and extend up to the trunk. There is no

trismus, and the neck and facial muscles are rarely affected. The absence of fever, absence of trauma or a wound, the long and intermittent course, and the presence of Trousseau's, Erb's, and Chvostek's symptoms, should be sufficient for the diagnosis.

From hysteria the disease is distinguished by the age of the patient, or, if the disease occurs in an adult, by the peculiar bilateral spasms and the remittent character, the absence of hysterical stigmata, and the presence of the peculiar forms of sensory and motor irritability described above.

PROGNOSIS.—The prognosis is favorable in most cases. In adults, however, when the disease is due to gastric dilatation and auto-toxæmia, a rapidly fatal issue has been often seen. The disease usually lasts a few weeks, but in adults it is liable to recur unless the conditions of life are changed.

TREATMENT.—The most important thing is to attend to the nutrition and digestive functions of the patient and rid him of any possible toxins that may be in the system. Rest, nourishing food, and tonics therefore are indicated. Symptomatically, the valerianate of zinc, in capsules containing 3 gr., may be given three times a day, combined with 20 gr. of bromide of potassium three times a day, and warm baths at a temperature of 93° F. for twenty minutes. During the attacks chloral, in doses of 20 gr., is indicated. In very severe seizures chloroform inhalations are recommended by Trousseau. Hypodermic injections of morphine may also be given then. The relation of the disease to thyroid toxæmia has suggested the use of thyroid extract, and in one case implantation of a piece of the thyroid gland in the abdominal cavity has been tried without result. Bramwell gave the thyroid extract in one case with success. Schultze and Masshner have used it in six cases with negative results. Hoche has recommended the use of curarine in doses of gr. $\frac{1}{320}$ to $\frac{1}{90}$. Vaughn saw excellent results from the use of quinine, but his results were not corroborated by Macalester, so that, after all, the later attempts at treating the disease have not added as yet to our therapeutical resources.

NEURALGIA; MIGRAINE.

By WILLIAM H. THOMSON, M. D., LL.D.

NEURALGIA.

Neuralgias are pains which are not due to inflammation or to pressure or to tension of nerves. Not only are we thus obliged to define neuralgia negatively, but so much confusion results both in diagnosis and in treatment from the vague use of terms that it is necessary still further to distinguish neuralgic from other varieties of pain by a brief statement of their respective contrasting features. Thus, the three great characteristics of an inflammatory pain are that it is increased by pressure or by handling of the part; that it is increased by movement of the part, not excepting its own proper functional movements; and that it is always accompanied by specific disturbance of the normal function of the inflamed part. With a neuralgic pain there is no objection to firm pressure, but usually the reverse; likewise none to movement, but here also often the reverse; and it is not accompanied by any characteristic disturbance of function in the part which it affects. On this account we would exclude most cases of so-called trigeminal, cervico-occipital, cervico-brachial, and intercostal neuralgia as no more entitled to this term than sciatica is, because they commonly partake of the characters of neuritis rather than of neuralgia, and hence are best treated as inflammations. Owing, however, to their ordinary designation as neuralgias, we shall treat of them in this article according to their symptomatology and nature. Pains due to pressure, as by tumors, aneurysms, etc., do not have the above-mentioned characters of inflammatory pains, and hence are often mistaken for neuralgias, especially when they radiate widely along the course of nerve trunks implicated in the pressure. It is useful, therefore, for diagnosis to note that they differ from neuralgia in the constancy of pain at the seat of the organic lesion which causes them. However extensively and changeably they may radiate from it, yet there always remains some uneasiness or pain at the primary focus, whereas true neuralgias are characterized by their tendency to shift from one nerve region to another or to intermit altogether.

Pains due to tension of nerves, as in the passage of a calculus through a narrow duct, or in sprains, are characterized by their sudden onset and by their tendency to cause nausea and faintness.

Pains which do not present the distinctive features of the above-mentioned varieties may then be termed neuralgic, and this term will be further found applicable because, though they are of numerous kinds, yet they possess certain general characters in common. The first of these has been already adverted to, in that they are all prone to wander about,

disappearing from one locality to develop with the same symptoms and severity in another part. They are also much less local, and are seldom referred to the skin, but rather to nerve trunks or their deeper distributions. Another marked peculiarity of neuralgic pains lies in the significant fact of intermittency. Neither an inflammatory nor a pressure nor a tension pain can intermit; such pains can only remit or else cease with the cessation of their cause. Neuralgic pains, on the other hand, may come and then go entirely, leaving the patients as free from them, temporarily at least, as if they had never experienced them. This fact of intermittency about many neuralgias is suggestive of their toxic origin, as it is in chronic toxæmia that we have similar cumulative effects leading to nervous explosions, so to speak, as uræmic convulsions or rigors from infections.

Though the above-mentioned characteristics are common to neuralgic pains in general, yet they differ so much from each other in their origin that a serviceable classification of the varieties of neuralgia ought to be based upon the causes rather than upon the symptoms of the complaint, more especially as the treatment should vary accordingly; for we can no more speak of the treatment of neuralgia than we could speak of the treatment of pain itself as if it were a single disease. Instead, therefore, of deferring the subject of treatment to the end of the article, we prefer to enumerate the various forms of neuralgia according to their etiology, and mention the remedial measures which on that account seem appropriate to each.

Toxic Neuralgias.

First in order come the neuralgias due to some form of toxæmia, of which the pains in the head, back, and limbs caused by the febrile infections are familiar illustrations. They are in no sense inflammatory, for they cause great restlessness, the patients tossing about in bed on their account, and not on account of the fever. This fact is of some import in diagnosis from like pains in meningitis, but which, being inflammatory, forbid movement of the body. Febrile neuralgias usually cease much before the fever ceases, except in the case of epidemic influenza, for these, like other sequelæ of this infection, are often prolonged and obstinate. The best remedy for febrile pains is aconite administered with ammonium bromide and phenacetine.

Periodical Neuralgias.—Among the non-febrile members of this class we have the truly periodical neuralgias, or those which come on at definite times and then subside, like an attack of ague. It is natural to surmise that they have some connection with malarial infection, but in many cases this is not plainly demonstrable, and, moreover, notwithstanding they may be very severe, they commonly are not accompanied by rise of temperature. They may attack the head, the sides of the chest or abdomen, or be located anywhere along the spine or in the pelvis.

I have not met with more violent cases of pain than in some of these patients whom I have seen both in my own practice and frequently in consultation. In the latter cases I have learned that quinine, Warburg's tincture, arsenic, and the coal-tar analgesics had been adminis-

tered, often in heroic doses, without effect toward preventing or mitigating the attacks; and hence I am glad to say that I consider ergot in full doses to be a true specific for periodical neuralgias, whatever their seat be. A drachm of the fluid extract should be taken every two hours with a drachm of elixir of cinchona, in water, to prevent nausea. If the stomach does not tolerate the ergot, it may be administered per rectum in two ounces of water. The first dose should be taken at the beginning of the onset of the pain, and if that suffices to arrest it, a second dose is not needed, but with some it requires three doses.

Migrainous headaches I regard as essentially toxic in their origin, and best treated accordingly (see Migraine).

Neuralgias with Tachycardia.—Patients with a persistent rapid action of the heart, without fever, and without cardiac or renal disease, and also without any affection of the brain or spinal cord to account for the tachycardia, are very commonly great sufferers from neuralgic pains in various parts of the body. This is well shown in many cases of Graves's disease both before and after the development of thyroid enlargement; but such neuralgias may be equally present in patients with such tachycardia who never show either exophthalmia or goitre. These pains are particularly apt to affect the ears, also the eyes and the tips of the fingers and toes.

Muscular pains are also common, which may be mistaken for rheumatism but for their tendency to shift often by mere change of position. These patients always give a history of chronic digestive troubles, both gastric and intestinal, long preceding the access of the pains. Here intestinal antiseptics, with frequent mercurial laxatives, perseveringly followed up by phenol- or naphthol-bismuth, bismuth and strontium salicylate, salol, etc., and total abstinence from butcher meat, taking milk instead, will cure both the neuralgias and the over-action of the heart. Many cases of gastralgia and of enteralgia are presumably due to like conditions in the alimentary canal, though not accompanied by tachycardia. The relief following upon the administration of $\frac{1}{20}$-grain doses of arsenious acid in gastralgia would seem to indicate its causation, for arsenic quickly arrests any form of fermentation.

Under the head of toxic neuralgias would come those occasioned by chronic metallic poisoning, especially that by lead, in which wandering pains occur which are apt to be mistaken for rheumatism, though undoubtedly they have more relationship to gouty neuralgias, as lead-poisoning is one of the recognized causes of the gouty state. On the other hand, the abdominal cramps are just as specific effects of plumbism as its special paralyses are. Gouty patients, especially women without pronounced articular symptoms, are very liable to neuralgias, which may long fail to be relieved until their relation to the blood state is detected. A high-tension pulse, with a history of frequent attacks of subacute ophthalmia or of bronchitis, or the presence of the "gouty beads" on the external ears, will give the clue to the proper treatment of the pains. Lastly, among the toxic neuralgias are those occurring in Bright's disease and in diabetes. Pain in the back of the head is common in Bright's disease, and is not necessarily of dangerous import. Not so, however, when general pains are complained of throughout the body, for these may presage a near fatal issue, as their frequent associa-

tion with convulsions shows. On the other hand, the local pains occurring in diabetes not infrequently are due to actual neuritis.

Anæmic Neuralgias.—Anæmia is often stated to be a cause of neuralgia. I would rather say that neuralgic pains are very common in anæmic persons; but that anæmia *per se* causes neuralgia I doubt, because of its frequent absence in many of the worst cases both of primary and secondary anæmias. Thus, I have met with instances of pernicious anæmia in which there was no neuralgia complained of from the beginning to the fatal end of the disease. On the other hand, blood impoverishment is often the direct result of the toxæmias which also cause toxic neuralgias, and hence I would paraphrase the saying, that "neuralgia is the cry of the nerve for more blood," into a cry for purer blood. Iron, quinine, cod-liver oil, etc. are good remedies in such cases by improving that general nutrition which is Nature's most effective antiseptic.

Non-toxic Neuralgias.

Reflex Neuralgias.—The second class of neuralgic pains is the non-toxic, under which head may come, first, reflex neuralgia, a good example of which I can cite of a true trigeminal neuralgia due to a necrosed piece of bone in the nares, and which was cured by its removal. Reflex neuralgias should be distinguished from the pains which radiate from an inflammatory irritation, as in toothache, or aching in the right shoulder in hepatitis, or at the head of the penis from calculus in the bladder, etc., or from pains radiating from a pressure focus, by the absence of these causes. When this is done the instances of true reflex neuralgia are found to be comparatively few, but they undoubtedly do occur, and commonly as a result of a traumatic injury of a nerve. Beginning with pain in the nerve first affected, it may pass up and implicate one nerve distribution after another through some intervening plexus or regional association. One of the severest cases in my experience followed upon treading barefoot on a piece of glass, which in time was accompanied by severe pain, not only extending up the leg, but finally involving equally the other leg. Ascending neuritis will account for some of these cases, but not for all of them, as many are explicable only on the theory of a central disturbance being set up by the peripheral lesion, but which becomes independent of it, as evidenced by the neuralgia persisting in some of these patients, though the nerve first implicated be cut. Sometimes the neuralgia, though originally due to a peripheral traumatism, affects by central transfer quite another territory, as in a case reported by Anstie of injury to the ulnar nerve causing neuralgia in the trigeminus.

The treatment of reflex neuralgias varies according to the accompanying conditions. Where there is a history of traumatism the most effective resource is a persevering use of the hot-water douche to the parts first affected. I have seen some striking results from this procedure, particularly if chronic muscular contractions accompany the pain. The douche should be kept up for an hour at a time three times a day, to be followed by hypodermics of morphine and atropine. In some of these cases the original irritation appears to induce an arterial anæmia of the

part by reflex vasomotor irritation, as shown by local coldness and wasting. Here nitroglycerin given every three hours is often of signal service. Where the pain has become chiefly central in its origin, general constitutional treatment is required, and, particularly, free out-door life.

Mental Strain.—Another form of neuralgia which is probably always of central origin appears to be caused by emotion, whether in the form of sudden mental shock or developing gradually under continued conditions of grief or anxiety, when the pain may ultimately become very severe. If following mental shock, no remedy is equal to opium given freely for a week and then stopped altogether, to be followed by ammonium bromide with spts. ammon. arom. and brandy. As the general health is usually much impaired in these patients, a strengthening regimen is indicated, while change of surroundings appears to be very effective by displacing morbid nervous habits.

Hysterical Neuralgias.—As the sensation of pain is itself a centric function, so it is natural to infer that its origin may in many cases be due to a primary disturbance of the pain centres themselves, without an antecedent peripheric irritation. Many writers hold that all true neuralgias are centric, and in support of this view adduce instances of central organic disease causing neuralgia at the periphery, as in a case of mine where a small intercranial sarcoma caused a severe pain in the foot. Also, neuralgic pains may occupy adjacent parts of different nerve distributions, or as in reflex neuralgia, where the pain may be felt in another place than the nerve irritated. The most conclusive examples of central neuralgias, however, are the numerous painful affections of hysterical patients. They are of every variety, cephalic, spinal, and visceral. About the head they do not often follow the course of a nerve, but are local, as in the so-called hysterical clavus, or they may be limited to special points on the spine. The visceral pains may be referred to the heart, stomach, bowels, bladder, ovaries, peritoneum, etc., and, as may be readily supposed, are more migratory than any other kinds of pain. Like other developments of hysteria, its neuralgias are often puzzling to the practitioner, especially when complained of about the joints or in the abdomen. As pain is such a subjective symptom in any case, and therefore must be gauged by the statement of the patient, the highly wrought accounts of suffering by hysterical patients might readily deceive but for a careful observation of the mental accompaniments of the pain. Thus, real objective pains are very concisely described with a few characteristic gestures and equally few words. Hysterical pains, on the other hand, are described with demonstrative gestures and with a copious variety of terms, which go to show how much the patients occupy their minds with dwelling upon them.

The TREATMENT of hysterical pains is on the lines of treatment for hysteria itself. Among single measures I have found much benefit from frequent recourse to mercurial and aloetic purgatives.

Degenerative Neuralgias.—One important form of neuralgic pains is that due to degenerative processes set up in sensory nerves and nerve roots, such as the lightning pains of tabes. The degenerative processes themselves may have been due originally to a toxic infection, such as syphilis, but this no longer operates as a cause of the pain. That these pains are not peripheric is shown by their contrast to the

peripheric pains of tic douloureux, for in the latter the peripheral nerves are exquisitely tender, while in tabes the skin area of distribution of the affected nerves is anæsthetic. The patients rub, seize, or grab the affected part as if they were desperately bent on arresting the pain in its flight. One of the drawbacks of naming diseases after common but not invariable symptoms is often illustrated in patients with tabes, who may suffer for months or years from the severe neuralgic pains of this disease without the cause being recognized, on account of the absence of the motor symptoms of ataxia. It is therefore always advisable in any case of obscure neuralgic pains to make a careful examination of the reflexes, particularly of the knee and of the pupil. The pains of tabes are commonly described as shooting or lancinating, and are very variable both as regards duration and seat. Sometimes, however, they are mainly visceral or very definitely localized, in which case they may be described as boring or compressing.

For the pains of tabes the coal-tar analgesics are often very effective, particularly acetanilid. Nitrate of silver also occasionally affords decided relief, and it is curious that this drug will sometimes unmistakably mitigate pressure pains caused by a tumor involving the roots of a spinal nerve, as I have had occasion to note in several instances.

True angina pectoris might come properly under the head of degenerative pains, though many anginose pains about the heart are of different causation (see Angina Pectoris, Vol. II. p. 503).

Herpetic Neuralgias.—Herpes zoster, or zona, is due to an organic change in special areas which are definitely associated with certain visceral nerves. It is probable that in the last case the visceral precedes and induces the special nervous lesion, for in the majority of patients neuralgic pains are felt for some time before the herpetic eruption makes its appearance, and not infrequently the pains continue, particularly in elderly persons, long after the eruption has disappeared. When zona attacks the eye the pain is not only very great, but the attack may destroy vision.

I have found ergot in full doses much the best remedy in the ocular attacks, both for the pain and for the accompanying inflammation. It is useful also in these pains when developed in other regions, and for the post-herpetic stage may be usefully combined with phenacetine. In elderly patients the pills of phosphorus, $\frac{1}{100}$–$\frac{1}{50}$ gr., with strychnine are often beneficial.

Neuritic Neuralgias.—Under this head is grouped an important class of pains generally termed neuralgias, but which, as remarked above, partake more of the characters of inflammatory pains in that they are usually permanently local and not shifting, as they may affect certain nerves or their branches without change for prolonged periods. The nerve trunks themselves become tender to pressure, as shown by the specific points named after Valleix, where the nerves in their course are superficial enough to be pressed against underlying bone. At their cutaneous expansion also they become exquisitely sensitive to pressure or to touch during the paroxysms. Likewise, movement may excite the worst paroxysms, as those of the muscles of mastication or of facial expression in trigeminal neuralgia, rendering chewing, or even opening the mouth for speaking, extremely painful. Finally, like neuritis in

general, trophic and vasomotor sequelæ are not uncommon in long-continued affections of the kind. They differ, however, from most forms of neuritis in the primary seat of the inflammation, which appears especially to implicate the nervi nervorum. These nerves are abundantly distributed to the fibrous sheaths of the nerves, ramifying and ending in them, without penetrating the interfascicular septa, and yet evidently entering into a close relationship with the fibres proper of the nerve, as the conduction of a local irritation of the trunk to its peripheral distribution shows. So inflammation of the nervi nervorum renders the trunk itself first tender, and then leads to its peripheral ends becoming hyperæsthetic as well. Their inflammation, however, is not likely to be attended with as much local effusion or infiltration as in ordinary neuritis, which explains why, though the skin may become highly sensitive to touch during the paroxysms, yet the patients often attempt to relieve the severity of the pain by firm pressure. Another difference from ordinary neuritis is, that the implicated nerves do not degenerate nor lose their reflex functions, nor does their affection cause muscular wasting.

Trigeminal Neuralgia.—By far the chief member of this class of neuralgias is the trigeminal, or tic douloureux, as it is an affection of the most important as well as the most exposed of the nerves of common sensibility of the body. One of the most frequent of its exciting causes, and which supports the view of its inflammatory character, is exposure of the face to cold winds, especially in cities, from the greater likelihood in them of sudden changes to persons from warm air in-doors. Another common cause is from the irritation of retained roots of decayed teeth, which may initiate a trouble which will persist, as in other instances of nerve irritation, after the original cause has been removed. One predisposing cause in elderly persons appears to be lowered resistance from the malnutrition following upon arterial degeneration. It is in them that it sometimes takes the form of Trousseau's epileptiform neuralgia, which is characterized by sudden extremely severe pains, which, though of short duration, may, however, succeed one another so often during the day that life becomes unbearable. In others the pain in the affected branch begins more gradually, then grows worse on movement of the facial muscles, and then comes on spontaneously in paroxysms which succeed one another like waves. The pains usually dart centrifugally, though when caused first by a peripheral irritation, such as a decayed tooth, the direction may be the reverse. Like inflammatory pains also, extensive radiations may occur from the face to the occiput, to the shoulder, or down the neck. Trigeminal neuralgia does not often affect all three of the main branches of the nerve at once, though not uncommonly two of them ; and it is rarely bilateral, which is another proof of its peripheral inflammatory origin. When attacking the supraorbital branch the pain radiates over the forehead to the eyelids and to the side of the nose, with special points of tenderness at the supraorbital notch, at the external angle of the bony orbit, and at the exit of the nasal branch at the lower edge of the nasal bone. Pains in the frontal sinuses and in the eyeball itself are not usually due to trigeminal neuralgia, but to other local causes, as their frequent bilateral development indicates. Infraorbital neuralgia is often very severe on the malar bone, but sometimes the pain is worst at the side of the nose or along the

gums of the upper teeth, and at the height of the paroxysm it may involve the whole cheek. The most extensive, however—and, in my experience, the severest—pains accompany affections of the third division of the trigeminus, radiating above up to the region just anterior to the tragus of the ear, or into the meatus itself, or to the temple, or to the parietal bone, with tender points corresponding; or it may dart into the tongue or be felt deep in the neck under the jaw, with severe wrenching pain radiating from the inferior dental foramen. When the paroxysms of pain occur in trigeminal neuralgia, vasomotor symptoms are very common, such as flushing, sweating, lachrymation, flow of nasal mucus, salivation, or even local nutritive changes, such as grayness or loss of hair or thickening of the periosteum. As might be expected from the inflammatory nature of the complaint, the foci of pain are very uniform in each particular case, corresponding to the special seat of the process.

Treatment.—Trigeminal neuralgia is best treated according to the principles of treatment of inflammation. The first indication, therefore, is absolute rest of the affected part. It is to neglect of this principle, in my opinion, that the intractable course of this affection is most commonly due. Unless the patient will agree to remain in bed, as he would with a fractured leg, and take special precautions to avoid all movement of the face as much as possible, as well as all exposure of it, every other measure of relief is apt to fail of its particular efficacy. In severe cases wearing a light mask of cotton will diminish both the frequency and the severity of the paroxysms. The food should be liquid, neither too hot nor too cold, and when the third branch is involved should be taken through a tube introduced on the unaffected side into the mouth. Medicinally, I would, with Trousseau, put opium in the first rank, but its specific operation will be secured only according to the mode of its administration. Occasional doses of the drug to relieve the agony of the paroxysms are of little avail. To be curative the opium (preferable to morphine) should be given continuously and at definite intervals by night as well as by day, in sufficient doses to suspend all returns of the pain, however slight. In other words, the patient should not be allowed to emerge from the effects of the drug throughout the twenty-four hours—a condition which, it must be admitted, becomes very irksome to the patients, who often beg that the continuous dosing be intermitted. This must not be allowed, however, for the success of the treatment depends upon its not being interrupted, even by ordinary sleep. I have never seen any injury result from this course, and on recovery the patients are only too glad to be rid of opium to ever take it again for anything but pain.

By this treatment I have cured cases of epileptiform tic which had resisted all other measures, in one case for five, in another for six, and in a third for nine, years. The duration of the opium course in these three patients averaged twenty-one days. The patients, however, should keep their beds for six weeks, and with infraorbital and inframaxillary affections should avoid chewing for two months, if not longer.

Aconitine has considerable reputation in the treatment of trigeminal neuralgia, beginning with doses of $\frac{1}{200}$ of a grain, and increasing until

decided physiological effects of the drug, such as lowering of the pulse to 60 or 50, are experienced. It requires to be kept up for prolonged periods, and acts best when the paroxysms seem by it to be gradually lessening and the pain changing into a continuous sense of soreness, when antipyrine may be further added, along with spts. ammon. arom. The fluid extract of gelsemium, in doses of 7 to 10 drops every four hours, may also be tried, particularly in superorbital neuralgia. In some patients a vasomotor condition is in time established in which the affected side becomes paler and cooler than its fellow, at least between the attacks. In such cases the administration of nitroglycerin every three hours is often followed by a marked relief from the neuralgia. Arsenic and phosphorus are very uncertain remedies in this complaint, but now and then seem to benefit individual cases. Gowers recommends a combination of arsenic, quinine, and Indian hemp. Dana[1] reports thirteen cases of severe tic douloureux treated by a prolonged course of rest in bed and hypodermic injections of nitrate of strychnine, given in a single dose daily. He begins with $\frac{1}{30}$ of a grain, and slowly increases till by the fifteenth or twentieth day $\frac{1}{6}$ to $\frac{1}{4}$ of a grain is administered. After reaching the maximum dose which the patient can bear, it is continued for a week or ten days, and then gradually reduced, so that by the end of four or six weeks the beginning dose is reached. The drug is then stopped and replaced by a full course of potassium iodide and tincture of iron. Of his 13 cases, 10 were so relieved as to be considered cured, while in 3 the treatment failed.

Surgical measures, such as dividing or resecting branches of the trigeminus, have been tried for many years. The drawback to these operations is, that in a large proportion the relief is but temporary, for the pain is apt, after varying intervals, to recur as bad as ever. I had a patient once who had three such operations on the inframaxillary nerve, but after a fourth recurrence he appeared to be cured by 30-grain doses of ammonium chloride taken in infusion of horseradish. He still remained free after six months, but was then lost sight of. There can be little doubt that the prolonged irritation of such nerves as the primary branches of the trigeminus is capable of establishing a permanent lesion in the Gasserian ganglion, so that nothing short of the destruction or extirpation (if that be possible) of this ganglion will afford a prospect of radical cure. Marchant and Herbert[2] have collected 95 recorded cases of this operation, 17 of which proved fatal; but, though relapses are reported even after this severe measure, the proportion of cures is much above that of other surgical procedures.

Cervico-occipital Neuralgia.—The pain in this form is oftenest in the great occipital nerve, with the characteristic tender points midway between the mastoid and the median line, where pressure can readily be made on the trunk of the nerve. It is sometimes accompanied with tinnitus and deafness, and it may radiate over the occiput to the parietal eminence or extend down the back of the neck to the vertebra prominens. It is bilateral much oftener than trigeminal neuralgia, and usually more of a continuously dull than paroxysmal character. It may be originally excited by cold draughts on the back of the neck, but I

[1] *Trans. Assoc. Amer. Phys.*, 1896, and *N. Y. Med. Record*, May 1, 1897.
[2] *Rev. de Chir.*, Apr., 1897.

have been struck with its frequent association in patients with severe or depressing mental strain.

Cervico-brachial or **brachial neuralgia** is located in the nerves arising from the four lower cervical and first dorsal segments, which make up the brachial plexus. The commonest seats of the pain are in the axilla and along the course of the ulnar nerve. As might be expected, the tender points over such extensive areas of distribution are very numerous, but as frequent sites as any are at the exit of the circumflex nerve, under the olceranon, and in front of the wrist. The movements of the upper extremity, either general or special, are likely to be so interfered with that this affection is very disabling, the acts of sewing or writing being particularly prone to start the pains. Traumatism, such as from falls or blows, is a more common cause than all others together.

Intercostal neuralgia is a very common affection, and closely resembles sciatica in its pathology. The tender points to pressure are found from the exit of the nerves beside the vertebræ, then along the lower edge of the rib, and then near the middle line in front. It is commonest on the left side, and often causes apprehension in the patients of heart disease. Under the scapulæ the pain is apt to be constant, dull, and wearing, worse at night and on changes of the weather. It is the form of neuralgia which is oftenest associated with rheumatism, though occasionally it results from traumatism or muscular wrenching. It is frequently mistaken for pleurisy, owing to the pain being aggravated by the movements of the ribs in respiration, but it is readily distinguishable from that affection by the fact that the pain on full respiration does not excite cough. Sometimes the pain is not accompanied by tenderness on pressure along the trunk of an intercostal nerve, but is localized at some one spot. Some writers therefore make of this a distinct form, to which they would restrict the term pleurodynia, with the theory that the seat of the pain is in the nerves of the pleura. I doubt this explanation, chiefly because, however the patient may aggravate the pain by inspiration, yet it never excites cough, and careful manipulation is generally sufficient to connect it with the peripheral distribution of a nerve in the thoracic wall.

True neuralgic affections of the spinal column are rare, except those already referred to as due to toxæmia or to radiated inflammatory pains or to hysteria. Pain referred to the spine, therefore, should be carefully examined for these causes before pronouncing it neuralgic. The chief exception to this statement is in a puzzling, and not infrequently a serious, condition resulting from concussion, as in the well-known "railway spine," where neuralgic pains may develop, often at a considerable interval after the accident. They may then become extremely obstinate in their continuance and in their effects, and there can be no doubt that the primary traumatism sets up in some of these cases slow, progressive changes in the spinal centres which may lead to very untoward results.

The TREATMENT of these different forms of neuritic neuralgias follows the general principles outlined in the treatment of trigeminal neuralgia, except that we can in them often resort more readily to various measures of counter-irritation. Thus the active irritation caused by the ointment of the biniodide of mercury applied to the mastoid processes

and to the back of the neck in cervico-occipital neuralgia is of much service, also along the track of the nerve in intercostal neuralgia or along the vertebræ in railway spine. Narrow blisters are likewise beneficial. The actual cautery, however, is, on the whole, the most effective, the simplest, and the best method for applying which is by heating the tip of a glass tube to redness in the flame of an alcohol lamp, and then making quick touches with it on dots of ink in line, half an inch apart, on the skin along the track to be cauterized. The hot-water douche is often beneficial in brachial neuralgias, but the principle of rest is here equally imperative, in bad cases, as it is in tic douloureux. It may be resorted to also in intercostal cases by firmly strapping the affected side. Local anodyne applications may be used at times with considerable benefit, such as—

℞.	Menthol,	ʒij ;
	Chloral,	ʒj ;
	Morphinæ hydrochloratis,	gr. vj ;
	Tincturæ aconiti,	ʒj ;
	Lanolini,	℥j.—M.
	Fiat unguentum.	

I have found this a good application also in coccygodynia when used after a hot-water douche.

MIGRAINE.

Synonyms.—Owing to its frequently affecting one side of the head the Græco-Latin term hemicrania has been applied to this affection, modified in several languages into the French migraine, German migrän, and English megrim. The French term, being closest to the original, is, on the whole, preferable.

Symptoms.—While its chief symptom is headache, its other accompaniments are so distinctive and significant that they should be regarded as equally entitled to consideration as respects both the etiology and pathology of the affection itself.

The first characteristic of migraine is its intermittency, resembling in this respect epilepsy. During the intervals the great majority of the patients are as free from headache as any other persons in good health. The attacks themselves often begin with a feeling of general malaise or of heaviness about the head, the countenance wearing a peculiarly dejected aspect. This is succeeded by a distinct pallor, the vasomotor spasm which this indicates being further shown by the smallness of the pulse and by a sense of coldness, particularly of the extremities. As the paroxysms of pain, however, come on, local flushings of the face, with redness of the ear, may succeed the paleness. Occasionally the eye on that side is retracted, and the pupil distinctly smaller than its fellow, or it may oscillate in size. Evidence that the spasm is limited to the arterioles may be noted by the temporal artery on the affected side remain-

ing thoughout the attack overfull and hard, and then returning to its natural state on its subsidence. Repeated attacks may result in a permanent enlargement of this artery, as has occurred with the left temporal in the case of the present writer, who for many years suffered severely from migraine. Thomas has shown that this may be due to a local arterio-sclerosis independent of a general affection of that kind elsewhere. Other trophic changes have also been noted. Thus, Anstie in his own case observed a distinct patch of grayness on the hair of his eyebrow over the supraorbital notch during severe attacks, which disappeared on their cessation. Permanent local grayness of the hair on the temple has been frequently observed, and Hutchinson draws attention to the supervention of xanthelasma at the inner canthus of the eyelid as more common in those who have suffered from migraine than in other persons.

In a large proportion of the cases—probably one half—the onset is marked by peculiar disturbances of vision, such as are characteristic of no other nervous affection. Their duration is temporary, from ten minutes to half an hour, but in some cases the symptoms are so pronounced, or may occur without any headache following, that their nature may be misinterpreted. I had a lady sent to me by an oculist for my opinion as to the cause of the attacks of what she described as total blindness, beginning with brilliant flashes of colored lights and ending with transient delirium, the optic disks during them being observed as quite normal. These attacks proved to be simply accompaniments of migraine, for, however peculiar the visual symptoms may be, the state of the retina rarely gives any indication of disturbance. In most patients there is only a blurring or indefiniteness of sight, particularly of near or small objects, such as the point of a pen. In others there is complete hemiopia, which may come on very suddenly—often, as the patients mistakenly imagine, first in one eye and then in the other. In others it uniformly begins as a dim spot, which may appear anywhere in the field of vision and then enlarge like a cloud as it does so, clearing in the centre and having on one side an irregular, zigzag, or angular periphery which may be brightly colored. These colored spectra may assume very varied shapes or else appear as moving bright spots on a dark ground. As these visual symptoms pass off the headache usually begins, but in some cases other sensory disturbances, equally transient, accompany them, such as sensations of numbness and tingling, usually in the arm of the affected side, which may simulate an epileptic aura passing up from the fingers, or there may be a feeling of paralytic numbness in the tongue, extending to the lips, cheeks, and throat, sometimes accompanied by a sensation of impending syncope. With some the attack begins with a feeling like a commencing coryza and stoppage in the upper nasal passages. Cerebral symptoms, such as transient delirium or aphasia, both sensory and motor, also occur, but usually only in those who were subject to severe attacks. In a very few cases choreic movements occur in the extremities, usually excited by some start. Photophobia is very common, with intolerance of noise, and occasionally tinnitus with vertigo.

The ocular symptoms are usually the earliest to occur, and in most patients precede by a definite interval the development of the headache. This begins quite habitually, in the majority, in a definite area, either in the temple, in the eye, on the forehead, or the occiput. With many

women, who have the attacks about the menses or during the menopause, the pain begins near the vertex. From its first local seat, however, it usually spreads till the whole side of the head is affected, and then the pain may extend down the neck. In a large proportion the pain involves the other side of the head also, or it may affect one side in one attack and the opposite in another. The pain, however severe, is purely neuralgic, and seems eased by pressure, so that many patients try to get relief by tightly tying a handkerchief about the head. The sense of mental and physical depression is extreme, and usually so much worse when sitting up or standing that the sufferers take to their beds and try to keep very quiet, dreading the waves of pain which the least movement seems to start. The headache may last from a few hours to a whole day and into the night, gradually developing a symptom which may finally replace the headache altogether, and which is a wretched nausea. At first it seems to be like an ordinary disturbance of the stomach, causing a total disinclination to take anything into it. This is followed by attempts to bring up flatus, then sickening, retching, and finally vomiting. Some are so unfortunate as not to be able to get beyond repeated retching, which in turn aggravates the headache. Free vomiting is often followed after a short interval by relief of the cephalalgia. In some the vomiting does not occur until some time after the headache has subsided, and then is very copious, showing in the matters ejected that all digestion had been arrested for hours in the stomach before the headache commenced. The urine passed during the attack is scanty and high colored, diuresis becoming very free on its subsidence. In many cases the complexion becomes quite sallow for one or two days afterward, and this fact, along with the quantities of bile brought up by repeated retching, affords good ground for these headaches being commonly termed "bilious."

ETIOLOGY.—Females suffer more frequently than males, in about the proportion of three to one. The disease begins in childhood as early as the fourth or fifth year, but becomes increasingly rare after fifty. Certainly more than three fourths of the cases begin before thirty years of age, and more than a half before twenty. On the other hand, wholly unlike epilepsy, it is a disease which patients definitely outgrow, for after forty it rapidly decreases in the number of its visitations, so that it becomes relatively uncommon after fifty even in those who have suffered from it in its worst forms. Heredity is very strongly marked in migraine, and in my experience more often in the maternal than in the paternal line of descent. Some writers, however, have exaggerated its association with other neuroses in its family history, as if it bore a relation to epilepsy or to insanity. As far as the numerical method goes, it can much more readily be shown to be associated with hereditary gout, but in neither case is the relationship direct, for the majority is altogether too large in which none of these affections can be traced in the family history. On the other hand, digestive disturbances are common in connection with all functional neuroses, and this fact will sufficiently account for migraine being also common in neurotic families.

PATHOLOGY.—Facts belonging to the etiology of migraine seem to have a distinct bearing also upon its pathology. Migraine requires an indoor life for its development, even among children. Men, and like-

wise women, who work in the fields, sailors, and miners, may have headaches, but not migraine. Among the poorer classes it is especially prevalent among needle-women, shop-girls, shoemakers, and tailors. Its chief victims, however, are among women who take but little open-air breathing or exercise, and who are by temperament susceptible to worry, and among men with sedentary or literary pursuits. Among the common exciting causes of the attacks are errors of diet, with many distinctly associated with indulgence in certain articles of food, also mental strain and fatigue, and with some exposure to a hot sun when the stomach is empty. As to the pathology of migraine, a view generally held is that it is a vasomotor neurosis, beginning with constricting spasm of the arteries of the affected area, followed by dilatation. This, however, is merely restating certain clinical features of the complaint. Liveing regarded it as the sensory equivalent of an epileptic discharge due to a gradual culmination in sensory centres of the morbid tendency till it explodes in a "nerve storm," as epilepsy explodes in motor centres. This analogy, however, does not hold good in those cases of epilepsy in which, instead of motor symptoms, there are only deranged states of the consciousness, but which bear no resemblance to the features of migraine notwithstanding the absence of spasms. The explosion theory, however, can be brought into close analogy with similarly developed nervous derangements occurring in toxic states of the blood. In puerperal eclampsia we have an example of a definite nervous strain, that of parturition, causing a poison, which had been accumulating in the circulation for weeks, to explode in convulsions. The same may be said of uræmic convulsions in general, that the poison which induces them often accumulates without any nervous symptoms denoting its presence until a certain culminating point is reached, when it may first manifest itself by severe neuralgic symptoms, notably pain in the head and disturbances of vision, and then eclampsia. Asthmatic attacks occurring in gouty patients afford another example of intermittent nervous disturbance dependent upon a more or less permanent blood poison. Even the peculiar ocular derangements of migraine are now illustrated by the remarkable toxic effects of the mescal button, *Anhelonium Lewinii*, described by Prentiss of Washington[1] and by S. Weir Mitchell,[2] in which colored spectra of the most wonderful variety are seen by persons under its influence. Instead, therefore, of ascribing migraine to nerve storms in a molecularly diseased nervous organization, we have a more probable explanation in ascribing it to a toxæmia, for it is in toxæmias that we meet with numerous instances of both cumulative and intermittent phenomena. Many facts, both clinical and therapeutic, point to absorption from the gastro-intestinal tract of some toxic substance as the specific cause of migraine. The immediate effect of stomach lavage in arresting the headache is now attested by numerous observers. C. A. Herter[3] found in seven cases in which the contents of the stomach were examined by him during the paroxysm evidence of complete arrest of the digestion, in one case nine hours after dinner. Navarre[4] reports a case of migraine which he had observed for eleven years, in which the attacks supervened exactly fifteen hours after every

[1] *Therapeutic Gazette*, Sept., 1895.
[2] *Brit. Med. Journ.*, Dec. 5, 1896.
[3] *Journ. Nerv. and Mental Dis.*, Feb., 1897.
[4] *Lyon médicale*, Mar., 1892.

error of regimen. Instances in which digestive derangements seemed to be directly connected with the genesis of the attacks might be cited indefinitely—a surmise which is further borne out by the beneficial effect upon the affection of every change from a sedentary to a more active outdoor life. Nor does the toxic theory of migraine militate against the clinical fact of its heredity, any more than the dependence of gout upon a blood poison militates against its heredity. In fact, there are no hereditary tendencies more pronounced than those connected with the functions of digestion, whether normal or abnormal. On the other hand, the predominantly unilateral character of migraine has been urged against its being due to a toxæmia. But other well-demonstrated toxæmias often occasion distinctly localized neuralgias, due probably to some predisposing cause of structural weakness in the part, and it may be affirmed of nerve poisons in general that they are characterized by selective affinities for certain nervous areas which with our present knowledge we cannot explain.

TREATMENT.—The treatment of migraine comprises both the management of the attack itself and its prevention. As above mentioned, evacuation of the contents of the stomach and washing that viscus out are among the most effective measures for relieving the headache, this being also in keeping with the common experience of these patients that free vomiting generally terminates the attack. Many drink large quantities of warm water to help both the vomiting and the headache together. Lavage is most effective when the stomach is full of undigested materials, but it is also beneficial if the stomach is empty. Among medicines my own experience is that ergot in full doses is superior to any other agent in arresting the headache, and in a number of severe cases of long standing it has seemed greatly to diminish the frequency of the attacks. A drachm of the fluid extract, with an equal quantity of elixir of cinchona, should be taken on the first symptoms of the attack, the patient then lying down and abstaining from all movement. A second dose may be taken in two hours if the symptoms are not relieved. Some patients cannot take the ergot by the mouth on account of nausea, when the same dose will be just as effective if taken dissolved in two ounces of water by the rectum. The coal-tar series of drugs, such as antipyrine, acetanilid, and phenacetine, have a just reputation in this complaint, particularly if taken early and the patients observe perfect quiet. In my experience the simultaneous administration of ammonia greatly promotes the efficiency of these remedies, a drachm of spts. ammon. arom. being taken in water just after 10 to 15 grains of antipyrine or of phenacetine, the dose of both being repeated in three hours if necessary. With some the further addition of $\frac{1}{40}$ grain of strychnine nitrate or 10 drops of the tincture of nux vomica is beneficial, particularly after the antipyrine or phenacetine seems to be losing its effect. This latter result is common to all the remedies which are recommended for the attacks themselves, and, moreover, individual experience varies greatly in the response to different drugs. Some derive no benefit from the antipyrine class, but are best relieved by morphine; others by full doses of the bromides; with others caffeine is effective in doses of 2–3 grains, or guarana, which contains a large percentage of caffeine, and which may be given in 30-grain doses of the powder, repeated every

hour. I have noticed that the cases of headache which are best relieved by caffeine are those which suffer least from nausea and vomiting in their attacks. The fluid extract of gelsemium in doses of from 5–7 drops relieves the headache in many, but not the nausea. A dose of 15–20 grains of ammonium bromide, taken with it, may much increase the efficiency of the gelsemium.

The most important therapeutic indication in migraine, however, is to prevent the attacks by correcting the conditions which occasion them. For this purpose no course is so effective as systematic open-air exercise of a pleasurable kind. Many find this best attained by the moderate use of the bicycle, which has the further advantage over walking of acting more directly upon the portal circulation. Mere muscular exercise, as in a gymnasium, is of little avail, and under all circumstances muscular fatigue is to be avoided, as it often is directly responsible for an attack. Attention to the digestive functions should begin by noting what kind of food seems to be most associated in time with the supervention of the attacks. Some patients find out for themselves that indulgence in certain articles of food or drink is pretty sure to be followed by headache. As a rule, it may be said that migrainous patients should eat but sparingly of red meats. This is especially the case in those who have a lithæmic diathesis, whether in the form of uric-acid gravel or gout. Eating heartily at night, and with men too free use of tobacco in the evening, are apt to make them wake in the morning with symptoms of the coming approach of the enemy. Lauder Brunton lays much stress upon correction by glasses of eye-strain in patients with hypermetropia or astigmatism as a cure in them of migraine, but it is doubtful if it be true migraine which is relieved by such means, though undoubtedly such ocular defects in migrainous patients would tend to aggravate, though not to cause, this affection.

For the medicinal treatment which would be both prophylactic and curative the guiding principle should be to improve gastric digestion. Stomach lavage, which is so beneficial in the attacks themselves, I have had occasion to note as distinctly injurious when resorted to in the intervals unless there is evidence of gastric catarrh—a condition which in migrainous patients is exceptional. The gastric dyspepsia in them is atonic rather than inflammatory, and the process of washing appears to depress the already enfeebled innervation of the stomach. The stimulant effect of a tumbler of hot water on rising in the morning, with a drachm of sodium phosphate and a grain of quinine or 5 grains of sodium salicylate, will often answer both to prevent headaches and to arrest their first beginnings. Mild acidulated preparations of phosphorus are beneficial, especially with women, a common prescription of mine being—

℞. Acidi phosphorici diluti, ℥jss;
Tincturæ nucis vomicæ, ℥ij;
Syrupi hypophosphitis, q. s. ad ℥vj.—M.
Sig. Two teaspoonfuls after meals.

Acidulated liquid preparations of pepsin are also serviceable, and in the majority lactic acid is preferable to hydrochloric acid, as in—

℞. Acidi lactici, ℥iv;
Essence pepsini, ad ℥vj.—M.
Sig. Two teaspoonfuls in water after meals.

Gowers recommends, as a prophylactic, nitroglycerin $\frac{1}{100}$ gr., not in tablet form, but in solution, to be taken after meals along with bismuth and pepsin, and claims that administered during the intervals, as bromides are taken in epilepsy, it lessens the severity and frequency of the attacks, and often after a time stops them altogether.

The prophylactic treatment in all cases should be systematically directed toward increasing the muscular tone of the stomach, and the manifest tendency in this complaint to gastric stasis suggests that the benefit claimed by some from abdominal massage is well founded. With reference to intestinal antiseptics, I have not found their use as certain as remedies more adapted for stomach derangements. An exception should be made, however, in the case of mild mercurial purgatives. Many of the severer cases are apt to have their attacks occur pretty regularly at weekly intervals, and I have repeatedly broken up this habit by a blue pill or by a grain and a half of calomel in six fractional doses, one every quarter of an hour, on the day previous to the expected return, the laxative action being ensured by a saline draught in the morning. Where the patients, however, are subject to intestinal indigestion, with an habitually brown-coated tongue, and especially if accompanied by gouty symptoms, the mercurial laxatives are certainly indicated, and may be very advantageously followed by a systematic course of 10 grains each of bismuth salicylate and sodium benzoate in capsules one hour after meals.

NEURASTHENIA.

By JAMES J. PUTNAM, M. D.

Definition and Nature.—Neurasthenia, in the strict sense, denotes that special sort of incompetence of the central nervous system in consequence of which fatigue occurs with undue readiness, while recuperation is abnormally delayed; but the term may also be legitimately used to cover the symptoms which arise indirectly in consequence of this primary incompetency, expressive of the efforts which the damaged nervous system makes to establish a new equilibrium.

It would be premature to assert that there is no difference between the state of a person suffering only from excessive physiological fatigue and that of a neurasthenic patient. Many of the phenomena and tendencies of these two states are, however, strikingly similar, and a familiarity with the laws of fatigue would be an excellent preparation for the study of neurasthenia. (See Symptoms, p. 563.)

The extent to which the nervous system is primarily involved probably varies greatly in different cases, but it must not be assumed, in a given instance, that the actual liability to exhaustion involves all the nerve centres whose functions seem disordered, for the neurasthenic condition is pre-eminently one where the effects of impaired inhibition and co-ordination, and of the formation of abnormal associations and nervous habits, make themselves widely felt. The results of this disturbance of co-ordination of the nervous centres are far-reaching, and even of fundamental significance. It is, for example, probable that fatigue-results occur with especial readiness when nerve functions which should reinforce each other fail to do so. As one illustration of this principle, the obvious importance of a failure in the working of the mechanism which should secure due response on the part of the vasomotor apparatus may be referred to. This often happens in neurasthenia, and as a consequence disorders of blood supply occur, and nervous centres not primarily at fault may be forced to work at a disadvantage, and may suffer under the strain to which they are subjected. In most cases it is probably the higher centres of the cortex cerebri which are mainly involved at first, as shown by the early failure of the powers of sustained intellectual effort; but the effects of this failure may make themselves widely felt throughout all the functions of the body. The disease is therefore a *psycho-neurosis*.

The question, What meaning is, in practice, to be attached to the term neurasthenia? may be approached in the interests of immediate clinical convenience or in those of scientific analysis. The former is the more practical need, but the attempt to meet it has led to a somewhat arbitrary classification of symptoms.

Unless we apply the touchstone of "exhaustibility" at each point, using the term neurasthenic as the equivalent of morbid liability to fatigue, and not letting it cover the other symptoms so often associated with this liability, we are forced to divide off neurasthenia by arbitrary lines from hysteria, melancholia, hypochondriasis, and the insanity of doubt and functional spinal affections, and, still more, from the large number of unclassified "mental incompetences"[1] that occupy the wide borderland between mental disease and health.

If we use the term in the sense indicated, it may be said that any patient, even if he have some other form of psycho-neurosis, may be, may have been, or may become neurasthenic, and require treatment as such. But if we use the term neurasthenia in a much larger sense than this, the result often is that we fail to grasp the trend and meaning of a patient's life-history regarded as a whole. Thus, if a neurasthenic patient should go insane, we might be tempted to conclude too readily that the insanity was based on the neurasthenia, though we ought not to draw this inference without carefully reviewing the tendencies which were present in infancy and youth before the neurasthenia become manifest. The term, in short, is a useful one in so far as it is used to denote the symptoms and results of undue exhaustibility, and it may, for convenience' sake, be allowed to cover certain symptoms of other origin frequently met with in connection with states of chronic exhaustibility, provided no better classification of them can for the moment be suggested.

Whether we consider it more convenient to regard neurasthenia as a *disease* or as a *state*, it is important to recognize that its symptoms may be present in very different degrees, and that the lighter forms are closely like health. No one's nervous strength is inexhaustible, and every neurasthenic patient has a certain share of endurance.[2]

The tendency to narrow the boundaries of neurasthenia, and, in general, to classify more carefully the related affections to which I have referred above, has made itself widely felt both in Europe and in America during the past few years, and the distinguishing marks pointed out by special students are attracting the attention of neurologists as helping out a promise of some fair substitute for the confused and irrational definitions of previous decades. The brilliant French school,[3] of which Charcot was for many years the most prominent figure, early strove to develop the doctrine of cerebral "degeneracy," and to claim as among the signs of this state the more pronounced forms of "morbid fears" which had by Beard been considered characteristic of neuras-

[1] *Psychische Minderwestigkeiten*, Koch.

[2] The shading from neurasthenia into health is well indicated in the following lines from Emerson's "Essay on Power:"

"With adults, as with children, one class enter cordially into the game and whirl with the whirling world; the others have cold hands and remain bystanders, or are only dragged in by the humor and vivacity of those who can carry a dead weight.

"For performance of great mark it needs extraordinary health. If Eric is in robust health, and has slept well, and is at the top of his condition, and thirty years old at his departure from Greenland, he will steer west and his ships will reach Newfoundland. But take out Eric and put in a stronger and bolder man—Biorn or Thorfin—and the ships will, with just as much ease, sail six hundred, one thousand, fifteen hundred miles farther, and reach Labrador and New England."

[3] Compare *La Neurasthenie*, 1891, Levillain.

thenia, while, at the same time, the attempt was made to show that neurasthenia was characterized by fairly definite "stigmata," of which the chronic pains in the head and elsewhere were the chief.

In 1891, Koch made a systematic attempt to classify all the neuropathic affections lying between health and mental disease according to their severity, their relation to innate tendencies and to specific causes, etc., and accorded to neurasthenia a relatively limited place. Strictly speaking, the neurasthenic condition does not correspond to any single one of Koch's clinical groups, but crops out as partly characterizing several of them.

In 1895, Freud[1] proposed the withdrawal of still another symptom-complex from neurasthenia—that, namely, which is characterized by the occurrence of mental states of hyperæsthesia and apprehensiveness, and a variety of bodily symptoms with which these states are often associated. Freud calls attention to the fact that Hecker had already pointed out the relation of many of these symptoms to each other, though without discussing the propriety of separating them from the neurasthenic complex. In like manner, the sexual neuroses are now studied and classified as a special form of neurasthenia, and the same might be said of the "traumatic" variety.

The conception of neurasthenia as the *neurosis of exhaustibility* has recently been re-stated and discussed by Knapp,[2] who points out, as the present article also attempts to do, that the mental obsessions and morbid fears and other abnormal mental states so often seen in neurasthenic cases do not form integral and necessary parts of the affection. Traces of these symptoms are seen in the fears and anxieties, partly well grounded, partly groundless, of "healthy" persons, and if they readily arise and grow especially rank on the soil of neurasthenia, the fact is not to be wondered at.

Neurasthenia may, indeed, as Müller and others have pointed out, lead to forms of true insanity, such as melancholia, "doubting insanity," hypochondriasis, or paranoia, but if the insanity actually occurs, it usually means that other special causes were active.

In accordance with this recognition of the propriety of confining the conception of neurasthenia to "morbid exhaustibility," with its immediate consequences, it is also recognized that some special category is needed for the "*nervous excitability*" which occurs, not as an outgrowth or corollary of fatigue, but as marking a departure from health in another direction.

This has been insisted on, among others, by Krafft-Ebing and by Möbius, who suggest the term "nervousness" (Nervosität) for this symptom-group. Eventually, some more rational and comprehensive classification for all these states, such as that of Koch, will probably be adopted, but for the present the terminology indicated must suffice. The essential point, after all, for the comprehension of these various but related psycho-neuroses is to recognize their close relationship to each other and to healthy states. If a series of triangles be imagined, all having the same base but divergent apices, one of them may be supposed

[1] "Angstneurose," *Neurol. Centralb.*, 1895, p. 50.

[2] *Boston Med. and Surg. Journ.*, 1896, p. 407. See also the able analyses of Krafft-Ebing, Von Hosslin (*Müller's Handbook*) Löwenfeld, etc.

to stand for health, while each of the others stands respectively for such affections as neurasthenia, hysteria, melancholia, and the mental states characterized by the presence of dominant ideas of anxiety, terror, false conception. These conditions are not mutually exclusive, but each represents a predominant tendency.

ETIOLOGY.—The influence which most strikingly predisposes to the occurrence of neurasthenia and its widespreading results is indisputably a *morbid inheritance*, though it may be admitted that the simpler and purer forms of the disease are those which are acquired as the result of overstrain in an ordinarily healthy person. All writers are agreed that this is true for the large majority of cases, though the patient is not always willing to admit that his case belongs in that category. The condition in the parent which gives rise to neurasthenia in the child may be some instability of the nervous system other than neurasthenia, or some acquired or inherited nutritional weakness.

There is little doubt that individuals may acquire peculiarities of nutrition which will show themselves as a neurasthenic tendency in their offspring. This seems to be conspicuously true where the acquired taint is due to syphilis or alcohol or to some serious impairment of the general nutrition, such as may come with a bacteriological infection. Phthisical parents are apt to have hysterical or neurasthenic children.

There is some reason to believe that the temporary condition of the *parents at conception* (*as alcoholic intoxication*), *and of the mother during gestation*, may affect the health of the child. Charrin and Nobecour[1] have contributed some interesting clinical observations showing that the children of mothers suffering from an *infectious disease* are liable to have a relatively feeble power of growth, and Charrin and Gley[1] have recently reinforced this view by their important experiments on the effects upon the offspring of rabbits (anomalous development) of inoculating the parents with specific toxins. It must, however, be admitted that the time is not yet ripe for asserting definite opinions as to just how acquired ill health of the parents acts upon the child—*i. e.* whether it is really a case of the transmission of specific cell changes or a case of unfavorable conditions for growth.

Another kind of hereditary cause of neurasthenia is an *imperfect provision for circulation of the blood or lymph*, as one form of which the small cardiac and arterial system, first noted by Virchow, may be mentioned. Beneke and Löwenfeld[2] have also claimed, on the basis of careful experiments, that the arterial system of the brain may be of small capacity relatively to that of the rest of the body. In like manner, it may be found that other inherited peculiarities of the circulatory organs, of the blood itself, and of other tissues and organs, which are generally spoken of as occurring symptomatically in neurasthenia, are really of causative significance.

In most cases the neurasthenic condition does not develop in a typical form until at or after the age of puberty, and patients are apt to refer their condition entirely to some special cause—an illness, an accident, a long strain, a severe grief, and the like. In so doing they usually

[1] *Arch. de Physiol.*, i., 1896.

[2] *Neurasthenia and Hysteria*, and *Arch. für Psych. u. N. Krankheiten*, Bd. viii., and references there given.

fail to recognize the inherited tendencies which were at work, and which, in most instances, the ordinary strains of life and physiological development would have made active. But the special cause does usually precipitate the result, and is also apt to determine, for a time at least, the form of the symptom-complex—a circumstance which indicates how large a part accidental, mainly psychical, associations within the nervous system may play in producing the symptoms of neurasthenia, though it be not so large as in case of hysteria. Thus, if a patient with developing neurasthenia suffers injury or disease of one or another part of the body, the back, the head, a joint, the pelvic organs, the eyes, etc., that part often remains sensitive and painful and the cause of prolonged disability. Such special injuries or diseases seem at times, especially in connection with railroad accidents or others of like nature, to turn a neurasthenic condition into one more like hysteria.

The questions are often asked, To what extent do the peculiarities of our *modern civilization* predispose to neurasthenia? and, Has it come to pass that the men of to-day are actually less competent to bear nervous strain than their remote ancestors were?

There are no sufficient data for satisfactory answers to these questions, and deductions from false premises are too often made to play the part of a reasoning from facts. At all events, it is only a matter of more or less, for the tendency to nervous strain must always have been present, since it is by changes in the nervous system, induced by competition, that man has advanced since the world began. We see the dangers amidst which we live, but cannot easily reproduce in imagination the exact conditions of the world without and within the brains of the men of centuries ago.

It is, on the whole, the opinion of the best writers that we do live in a nervous age, and that the civilization which has diffused the opportunities for education and communication has brought, also, temptations and cares to many who were ill prepared to meet them, and through them to their children. Yet there is doubtless too strong a tendency at the present time to recognize degeneracy and to despair of regeneration.[1]

These are problems that statistics have as yet failed to solve, and another of like difficulty is that of the relation of *race* to neurasthenia. Beard, whose name should be mentioned with respect by every writer on this subject, calls it the American disease. But if he had been a German, he would surely have called it the German disease, for there is no country that has contributed to the literature of neurasthenia so voluminously or spoken of it more as a national evil. It is probable that the psycho-neuroses in general are particularly common among the Hebrew and the Latin races, but with the former race, at least, the neuropathic tendencies usually pass beyond the limits of simple neurasthenia.

Developmental Epochs. — Neurasthenia generally shows itself first in the years immediately following puberty. This is probably due, in great measure, to the fact that this period is the epoch of new emotions, temptations, and responsibilities which the patient is often ill prepared to bear; but if one may judge from the frequency with which the symptoms break out in persons who have led an apparently healthy and protected life, the nervous system during these years must

[1] Compare W. W. Ireland: *Edin. Med. Journ.*, Sept., 1895.

be excessively vulnerable. "Never robust, but otherwise well until the last years of school," is a statement which one hears so frequently as to make it probable that the neurasthenic condition is simply a modification by natural evolution of certain neurotic instabilities of childhood, and owing its actual appearance to causes analogous to those which make this period of adolescence and early manhood prolific in outbreaks of epilepsy and hysteria.

Although neurasthenia usually develops in adolescence or early middle life, yet neither childhood nor the period of advanced years is wholly exempt. At the time of the menopause and during senile involution groups of symptoms are often met with which belong in this general category, though they may be different, in one or another respect, from those which occur at an earlier epoch, simple exhaustibility usually playing only one part in their characteristics.

It is oftentimes possible to recognize in childhood the tendencies which will come out as neurasthenia in adolescence or adult life. The symptoms may be of the same general character with those found in typical adult cases—*i. e.* the child may present no special characteristics but those of undue exhaustibility, or his nervous system may show a tendency to ill-working in other respects, and may thus place him at a disadvantage when brought into competition with those who work with more economy of force. It cannot be predicated of every nervous child that he will grow up neurasthenic, but in the doubt he should be given the benefit of a training calculated to develop all his capacity for hardihood, healthiness of body and mind, and self-control.

The influence of a *faulty education* of children both at home and at school is very important in this connection, but to save space its discussion will be taken up in the section on Treatment (p. 582).

Sex.—Neurasthenia is far more common among men than typical hysteria is, and some writers have attempted to show that it is more common among men than among women. This is probably incorrect, however, unless one excludes cases occurring in women where there is an admixture of hysterical symptoms. Possibly it might be said that in women as a class, as with the Hebrew and Latin races, a mobility and instability of temperament are often present, such as makes it less likely than in the case of men that the neuropathic tendency will arrest itself at the stage of neurasthenia.

Among the most important exciting causes of the neurasthenic condition are those which induce a *severe or prolonged strain*, especially those of an emotional sort. Strains of this kind are often due to cerebral perturbations which cannot work themselves out in adequate co-ordinated reactions of an outward kind, but excite inco-ordinated and mischievous disorderly inward reactions, of which serious disturbances of the cardiac and vascular innervation often form a part. It is by no means necessary for the result that the emotional strain should be consciously felt as such or in its full intensity, and, indeed, it appears sometimes to be a positive gain that it should be so felt, for then the emotion is more likely to vent itself in some outward expression which acts as a safety valve.[1] For example, the injurious effects of a railroad accident,

[1] See the interesting theories of Breuer and Freund with regard to the causes of hysteria: *Studien über Hysterie*, Wien.

of which the patient may in his ordinary conscious state remember nothing, are often prolonged and disastrous, and are greatly accentuated by all the influences which gather round a suit for damages.

As fright or humiliation may check digestion, disturb the action of the heart and the secretions of the liver, set up Graves's disease, or cause sudden death, so also it may accentuate or initiate the changes which underlie neurasthenia.[1]

It is a question for further investigation just how a single emotional outbreak acts in exciting neurasthenia, or whether the illness thus induced is identical with the forms which come on more insidiously. It is my opinion that this is probably the case, and that there is a closer analogy than has been heretofore admitted between the acute emotional neurasthenias and acute emotional hysterias, auto-suggestion, which is closely analogous to hypnotic suggestion, playing an important part in both cases.

Not only acute emotions, but prolonged strains, as those due to the anxiety or fatigue of nursing an invalid friend or relative or attending a life of extreme monotony, or the work of teachers or ambitious students or professional men, especially where grave responsibilities are undertaken on a basis of insufficient preparation, are important causes. In these cases the emotional factor is generally at work, though sometimes in a masked form, but it is also true that even purely intellectual and pleasurable work may be pushed beyond the danger line, in which case the "anæsthesia of fatigue" (Cowles) is sometimes the first sign of harm. The fact has not been sufficiently recognized that when, from lack of balance, the nervous system works under a strain—a condition often seen among the children of neuropathic parents—neurasthenia is very liable to develop, while, conversely, the neurasthenic tendency is a cause of neuropathic lack of balance.

To what extent may we assume neurasthenia to be an affection of *toxic* origin? It is easy to reason that as acute fatigue is due in part to the accumulation of toxic products—in support of which view various facts, such as the toxicity of the blood of fatigued animals, could be adduced—and as neurasthenia is a sort of chronic fatigue, and is accentuated and possibly induced by various poisons, such as lead and alcohol, so it is probable that auto-intoxication and the impairment of the self-nutrition of ganglion-cells play an important part in its production. But it is to be borne in mind that there is usually some peculiarity of the nerve centres which makes one person sensitive to these influences while another is not, and that of the nature of this peculiarity we are still ignorant.

Illness is an important cause of neurasthenia, but not all kinds among those of apparently equal severity are equally important. Most so are some of the infectious diseases, as diphtheria, the grippe, and malaria. Several influences here come into play, of which the chief are the poisoning from specific toxins, the effects of cardiac and vascular debility, and of disturbances of the general metabolism. Chronic diarrhœa and other disorders of digestion and metabolism which affect the quality of the blood may perhaps act in a similar manner to the

[1] See, among other works, Féré: *Pathologie des Emotions.*

infectious processes. The effect of the *habit of illness*, as leading to hypochondriacal tendencies through which an invalidism tends to perpetuate itself, as if the patient were unconsciously acting a rôle, also comes in as a complication.

But even in these cases hereditary influences are often at work also. Most persons recover so well from influenza, for example, after perhaps a slow convalescence, that one is tempted to ask, in case protracted neurasthenia results, whether the first illness did more than accentuate an existing tendency, just as the same sort of illness sometimes accentuates a tendency to locomotor ataxia and the like, causing the symptoms to develop with extraordinary rapidity. Chronic diarrhœa, again, though it may act as a cause of nervous debility, is also apt to occur as a sign of this state, the disturbance in innervation coming first.

A great many other poisons may act in a similar way with those just referred to, and the following list might be largely extended: 1 (as already noted), the substances generated by the chemical changes resulting from the action of the nervous system itself; 2, those from faulty metamorphosis of tissues; 3, alcohol, arsenic, lead, mercury, coffee, tea, tobacco.

As regards the second class of this series, it is undoubtedly true that disorders of general metabolism are important in relation to neurasthenia as causes, consequences, and complications. It is, however, also true that we know little that is positive, and that, although we speak freely of toxic diatheses, the doctrine as regards any special poison, such as uric acid, is by no means an assured one. The supporters of the lithæmic theory, such as Haig, believe that with certain persons—and all persons under certain conditions—there is an accumulation of uric acid in the tissues (due mainly to diminished excretion), which in its turn is brought about by an increased acidity of the blood on account of the presence of an excess of acid salts and other products of nitrogenous metabolism. The quantity of uric acid is also supposed to be increased by the consumption of "articles of food which contain it or other members of the xanthin group, as meat or fish or tea or coffee or guarana." In Haig's treatment the use of these articles is almost wholly abandoned in favor of a diet of milk, cheese, eggs, bread, vegetables, and fruit. The milk and eggs introduce nitrogen and even uric acid, it is true, but the salts formed by their decomposition do not lead to the making of acid products to the same degree as do those of meat, and they tend rather to increase the urea than the uric acid. On the other hand, fish is about as objectionable as meat.

One of the prominent effects of the accumulation of uric acid, according to Haig, is a high arterial tension, due to contraction of the arterioles, and it is through improvement in this respect that the good effects of treatment are sometimes first to be seen.

There are difficulties in the way of making a diagnosis of uric-acid diathesis, and equal difficulties in drawing further conclusions from purely clinical observations.[1] The simple presence of free uric acid in

[1] A recent and careful research by Frederic L. Hills (*Boston Med. and Surg. Journ.*, March 19 and 26, 1896) has shown the necessity of caution in this matter. See, also, a valuable paper by R. T. Edes, published in the *Trans. of the Assoc. of Am. Phys.* for 1897.

the urine often means only a precipitation, not an excessive formation, and it can only be by inference that we conclude as to storage in the tissues. In fact, the whole theory has been elaborated on a slender basis of facts, and, had it not been for the labor involved in an experimental disproof of the doctrine, it probably would not have stood so long. As regards the argument from the success of a special diet and treatment, it is well known that any treatment which is fully believed in by the physician is apt to help the neurasthenic patient by restoring hope and confidence; and, in fact, some neurasthenic patients do particularly well on a meat diet. Still, there is no question that the urine of certain classes of neurasthenic patients is apt to be of high specific gravity and to contain free uric acid; and good observers[1] believe that the lithæmic doctrine represents principles that are of great importance, even if we cannot now exactly formulate them.[2] It is probable that these disorders of metabolism are rarely the sole cause of neurasthenia but rather an accentuating influence, and probable also that the various special substances which have been incriminated hitherto are not the cause of the symptoms with which they are found but only the signs and by-products of a disorder of nutrition which is as yet unknown to us.

The case of *syphilis* requires special consideration. Kowalewsky[3] has asserted that it may cause neurasthenia by its own toxic influence; through the moral and emotional disturbance to which the infection gives rise; by necessitating a use of remedies which are often given to an excessive degree; by engendering changes in the parent which are inherited as neurasthenia by the offspring. Löwenfeld thinks that Kowalewsky goes too far in attributing so much causal action to the direct toxic influence of syphilis, but in the main his observations are certainly correct. The fear lest infection has occurred also acts, like other mental strains, to produce serious results of this sort.

Neurasthenic symptoms are often associated with those of *anæmia*, but it is noteworthy that in cases of typical neurasthenia, even among feeble and pale-looking persons, the number of red globules to the cubic millimetre and the relative amount of hæmoglobin are apt to be surprisingly near the normal. It may be that the whole quantity of the blood is diminished in such cases, but in general it would seem to be the case that it is not so much anæmia as toxæmia that is productive of neurasthenic conditions.

It has frequently been observed[4] that neurasthenic patients have a *high tension*, even though feeble pulse. More recently the attempt has been made to show that a typical arterio-sclerosis sometimes underlies this state, and Löwenfeld practically endorses this view. The proba-

[1] Löwenfeld, *loc. cit.*, and *Objective Zeichen der Neurasthenie;* Lehr: *Die Nervöse Herzschwäche*, 1891, p. 49; Schmidt: *Neurologisches Centralb.*, July 15, 1894.

[2] *Transactions of Association of American Physicians*, 1895; *Med. News*, May and October, 1894.

[3] *Centralbl. für Nervenheilkunde u. Psychiatrie*, March, 1893.

[4] See Webber: *The Pulse-tension in Neurasthenia;* Edes: *The New England Invalid;* and other writers on the subject. Federn of Vienna (*Blutdruck u. Darmatonie*) lays great stress on the causative influence of this high arterial tension, and believes that it is due, in many cases, to excitation starting from a local atony of the bowel. The argument which he adduces with regard to the latter point does not seem convincing, but his studies on tension are decidedly interesting. Compare, under Symptoms (p. 569), the recent observations of Oliver and others.

bility is that any disturbance of the circulation sufficient to impair the nutrition of the nervous centres may lead to this form of incompetency in persons predisposed thereto, but it has not been my experience that neurasthenic symptoms are especially common among persons showing what might be called typical arterio-sclerosis, or that typical neurasthenic patients are apt to suffer prematurely from the usual results (as retinal and cerebral hemorrhages, nephritis, etc.) of arterial disease. Von Basch, whose authority stands high in these matters, does not think that tortuousness of the arteries, so often seen in neurasthenics, is a sufficient evidence of sclerosis.

It is certain that neurasthenia may coexist with an excellent state of general nutrition, and equally so that a state of poor general nutrition, even if chronic, does not necessarily entail neurasthenia. There are persons, for example, who are too thin, and who suffer easily from the cold, and often have an abnormally low temperature, and whose muscular system is relatively poor, yet who may be wiry, and good sleepers, and for whom the designation neurasthenic would be a misnomer, though they may be "nervous" or "neuropathic" in another sense. On the other hand, the neurasthenic temperament is often associated with one of several types of bodily disorder, which have in part been graphically described by Clifford Allbutt.[1] Such persons (usually men) are apt to be slender, with sensitive, intellectual faces, often sallow skins, thin hair inclined to turn prematurely gray, large pupils, prominent subcutaneous veins, and of delicate digestion. They are hard and good workers, but rarely carry large enterprises to a successful close. Edes[2] has given good descriptions of two types seen in women—one dark and spare, the nervous symptoms being those of relative excitability; the other, characterized by lighter complexion and larger frame, and associated with more phlegmatic temperaments and less capability of improvement.

It is difficult to say here, as between some of these disorders or peculiarities of nutrition and the nervous weakness, which is cause and which is effect, or whether it is an affair of cause and effect at all.

Among the conditions which are comparatively rarely mentioned in connection with neurasthenia, and yet which may occasionally stand in an important causal relation to it, is the so-called *enteroptosis*, or prolapse of the abdominal organs, to which attention was called by Glénard in 1885 as being a frequent cause of nervous states. Of late years the prominent writers on disorders of digestion have investigated the matter further, and very recently the subject has been reviewed by Schwerdt and Gotha[3] and in a valuable monogragh by Stiller.[4] The view taken by this observer is that enteroptosis [or splanchnoptosis] is not so much a cause as a sign of a general disorder of nutrition or development, of which neurasthenia is one symptom; and he finds a further confirmation of this view in the fact, which he has observed, that in these same cases there is often a "movable tenth rib." The type of neurasthenics in which these conditions are found is the slender type with dyspeptic symptoms.

[1] *Nervous Dyspepsia.* See also Koch: *Die Psychopatischen Minderwertigkeiten.*
[2] Edes: *The New England Invalid.* [3] *Deut. med. Wochen.*, Jan. 23, 1896.
[4] "Ueb. Enteroptose in Lichte eines neuen Stigma-neurasthenicum," *Arch. f. Verdauungs Krankheiten*, 1896.

The signs and symptoms differ somewhat according to which organ is principally affected, but in almost all cases there are great relaxation of the abdominal walls, a collection of gases in the bowel, and diminution of intra-abdominal pressure. When the stomach is involved the signs are those of atony, with the addition that the line of the lesser curvature as well as of the greater is lowered. In a similar manner, the intestine, especially the transverse colon, may be affected, also the kidney, the liver, the spleen, the uterus, the rectum. The principal causes are hereditary tendency and unsuitable hygienic conditions during childhood and youth, leading to muscular fatigue, muscular feebleness, and an atonic condition of the nervous system. Various chronic diseases tending in the same direction are effective during adult life, and the use of corsets and lack of care after childbirth are also important causes. The details of the subject would repay more attention than can be accorded to them here, but so far as the relation of the disease to neurasthenia is concerned, which Glénard considers to be a very close one, the history of the affection gives one proof the more that persons with neurasthenic tendencies must learn, in early life, to live hardily and sensibly, and to shun the luxurious habits which stronger persons might indulge in without harm.

Frequent *pregnancies*, especially if associated with puerperal affections or uterine disorders, and *prolonged lactation* act occasionally as powerful factors in inducing neurasthenia. The history in many cases is one of ill health dating back to the birth of a first child, at which time some complication occurred. It must not, however, be forgotten that, on the whole, marriage and pregnancy bring health rather than sickness to women.

Pelvic disease in women and *disorders of the genital tract in men* are often credited with causing neurasthenia. But the conditions here are usually complex, and the special cause acts, in the way above indicated, rather to crystallize and give a particular form to the neurasthenic tendency than actually to excite it. In the case of sexual neurasthenia especially over-excitement and fatigue of a fundamentally important and emotional function come into play, and disturbances of local, spinal, and cerebral functions combine to produce grave results. The subject is too large to be done justice to here.[1]

One other cause of neurasthenia may be mentioned which has been little discussed elsewhere—namely, *blindness and deafness* occurring in adult life. The depression which these causes induce often, indeed, transcends the bounds of simple neurasthenia, but that element is nevertheless present to a marked degree. In one blind patient recently under my care the "helmet sensation" has been present so long and in so severe a form as strongly to suggest some more serious trouble, which, however, did not exist.

A protracted and harassing discomfort, like that from chronic *eczema* or *pruritus*, may help in the development of neurasthenic symptoms.

PATHOLOGY AND PATHOLOGICAL ANATOMY.—What is the nature

[1] See *Nervosität und Neurasthenische Zustände*, by Krafft-Ebing; *Handbuch der Neurasthenie*, Müller; *Hysterie u. Neurasthenie*, Löwenfeld; and the recent monographs of Fürbringer, Schrenck-Notzing, Eulenburg, in which this important subject is fully discussed.

of the primary defect which thus invites fatigue and delays recuperation? Binswanger,[1] the latest systematic writer on this subject, gives a careful discussion of the recent views, and concludes that *in the hereditary cases* the nerve cells probably have a diminished power of forming stable chemical syntheses and a diminished capacity for storage of latent force. It may be assumed, in a general way, that the chemical and physical structure and the mode of action of the neurasthenic nervous centres are slightly, but only slightly, different from those of the healthy individual. Arndt long ago claimed to have noted a tendency in the ganglion cells of neurasthenics to remain in a relatively embryonic stage of development, but since, in spite of the recent activity of histologists in the study of nerve cells, no authoritative corroboration of this observation has appeared, we must regard it as suggestive rather than demonstrative. If a progressive nutritive disturbance was at work, we ought to find traces of it in the form of neuron degeneration, but nothing of this sort has been observed: neurasthenia does not appear to lead to destructive and sclerotic changes.

Assuming, however, that one element in neurasthenia is a real impairment of the fundamental power of self-nutrition on the part of a greater or less portion of the central nervous system—and this assumption we must make, though the importance of the subnutrition in itself is usually overrated—the problem how far self-nutrition is a specialized function of the nerve cell becomes one of great interest. This problem cannot at present be regarded as solved in either sense, but it should be approached with an open mind.

Strictly speaking, there is at the present moment nothing known in pathological anatomy of the fundamental nerve lesions in neurasthenia, and it has seemed best to set down under Etiology (p. 556) and Symptoms (p. 577) the few observations which indicate the character of nutritional disorders with which the nervous disturbances seem to be more or less clearly related.

There is, however, no reason to deny that the day may come when we shall have a good conception of the changes at stake. A beginning has already been made through the admirable researches of Hodge, Nissl, and others into the pathological anatomy of acute fatigue and the early nerve-cell changes in disease, which seems to show that demonstrable alterations take place in the nucleus and protoplasm of the cells, while the nerve processes seem to be exempt. The anatomical secrets of the psychoses are also being diligently probed. Before we can find the changes characteristic of neurasthenia we must learn where to look for them, and for this purpose we must make closer physiological and psychical studies of the mode of origin of symptoms.

Attention may be called, in this connection, to two recent speculations on the dynamics and chemistry of nerve force by recent English writers, Gowers[2] and Broadbent,[3] as suggestive of one direction in which departures from the normal processes are to be sought.

There is much to be said for the conception that toxic substances are produced during acute fatigue, and that to them the symptoms are partly

[1] *Die Pathol. u. Therap. der Neurasthenie*, June, 1896.
[2] *The Dynamics of Life*, London, 1895; also, *London Lancet*, 1895.
[3] *Brain*, 1895.

due. It is, again, not to be forgotten that, although we cannot now describe the fundamental changes in neurasthenia, there are many secondary changes with which we are somewhat familiar, and which have an importance not only as results, but, finally and in their turn, causes of the neurasthenic condition. Such are the catarrhs of the stomach and naso-pharynx, etc., the vasomotor changes leading perhaps to arteriosclerosis, the cardiac disorders predisposing to impaired nutrition of the heart muscle.

Symptoms.—It is well to consider, in studying the complex symptomatology of neurasthenia, that we have to do partly with exhaustion-symptoms properly speaking; partly with their remote and immediate consequences, both as they occur with nervous systems relatively free from native weakness and as they accentuate pre-existing tendencies. Finally, there are symptoms which are not to be ranged under either of these headings, yet which, on clinical grounds, can be classed better under neurasthenia than elsewhere, in the present state of our knowledge.

The uncomplicated fatigue-form of neurasthenia is represented by —1, the simple exhaustibility of convalescence, as from infectious illness or prolonged strain; 2, the lifelong feebleness of certain persons, often of neuropathic or tubercular inheritance, who have been saved through the possession of firm dispositions and well-trained wills, or by peculiarly favorable surroundings, from forming mental and nervous habits of a morbid sort to any considerable degree; 3, the prolonged exhaustion sometimes following overwork or mental strain.

The prime symptom of this state is of course that the patients are unable to count upon themselves for any considerable effort either of the attention or as involved in the exercise of the various functions. Or if, under the "anæsthesia of fatigue" or by special exertion, the effort is carried through, a more or less prolonged state of exhaustion follows, attended by impairment of the normal balance of the nervous functions, leading to mental irritability, insomnia, disorders of the digestion and of the sexual functions, the heart's action, and the vasomotor responses. The reactions of fatigue, some of which will be referred to at greater length farther on, are likely to occur under these circumstances. These are important, because they may give indications which the patient's consciousness does not reveal.

Thus, investigations among school-children have shown that the power of estimating the pressure of two points upon the skin[1] often grows less after some hours of study at school, while at the same time they show a muscular restlessness which is sometimes of rhythmical character.

Marked defects of sensibility or motion are, however, not characteristic of fatigue or of simple neurasthenia.

It has been thought that the progressive concentric narrowing of the visual field during a test observation, as first described by Förster and afterward studied by Wilbrandt, Sänger, and others, was of neurasthenic origin, but the recent studies of Sänger make it appear probable that this interesting sign, which is of especial significance in the nervous states consecutive to trauma, is analogous to the permanent anæsthesias of hysteria.

[1] Griesbach.

It is, as has been said, only in carefully protected cases of neurasthenia that the symptoms show themselves exclusively on and after exertion, as in the fatigues of health.

As a rule, fixed habits soon begin to form, and the symptoms crystallize around one or another special function, as the use of the eyes, the digestion, etc., while at the same time morbid peculiarities show themselves, even during the intervals of fatigue-bringing efforts, all tending to betray a neuropathic tendency which is, indeed, often hereditary.

A large proportion of the symptoms of an advanced case of neurasthenia are, in fact, of secondary origin. Even in the mildest cases the patients are apt to show an excess of sensitiveness and mobility, an extreme refinement of taste, fastidiousness, or over-conscientiousness, if nothing more.

It is extremely important that the manner in which these secondary symptoms arise should be understood, and, in addition to what is said here, the reader is referred to the sections on Definition and Nature and on Mental Functions (pp. 549–563).

It is highly probable, though we have no right to assert the fact dogmatically, that the greater part of the symptoms of neurasthenia are of cerebral—that is, of psychical—origin. For the purposes of this inquiry the nervous system may be considered as made up of superimposed layers of reflex mechanisms, the same physiological events being represented over and over again, but in constantly increasing complexity of association. The cerebral cortex is the final "projection plane" where perhaps all the phenomena of which the body is capable are represented, but represented in combinations which are unstable in proportion to their complexity. When the functions of the nervous system become disarranged by a disorder of general "incompetence," such as neurasthenia, it is usually the cerebral functions that first feel the disturbance. Of course some one of the lower centres also may chance to represent a weak spot, and a focus of special disorder may thus be found. But this is probably comparatively rare, because the functions of the lower centres are relatively fixed and stable; and even when it does occur, if the case is one of neurasthenia, the brain is almost sure to pick up and recast the local affection. On the other hand, the cerebral disorders may of course manifest themselves outwardly in the form of local disturbances, which occur in endless variety, and which would formerly have been considered as of spinal or peripheral origin or as indicative of disease of a special organ. For the researches of later years have extended the domain of the cerebral functions. "Spinal irritation" has now no standing as an anatomical diagnosis, and even "myelasthenia," though it probably exists, is of much less frequent occurrence than used to be supposed. It cannot be admitted that symptoms which are of physical origin should all be stamped "hysterical;" the distinction between hysteria and neurasthenia is in this respect one rather of degree than of kind.

It is very noticeable that after the characteristic symptoms of a given case have become well established a very slight provocation may induce an elaborate group of phenomena entirely out of proportion to the cause, and for which the patient will often, with almost ludicrous per-

sistence, seek some special explanation. Thus one person, if he gets over-tired or annoyed, can count upon having special depressive ideas which will last a certain length of time; another will have a special form of indigestion or loose movements of the bowels; another a pain in a certain part of the body; another an attack of sweating or palpitation or trembling; and so on. Each neurasthenic has his own demon of this sort.

There is another general tendency which is characteristic even of mild cases of neurasthenia—namely, that the symptoms tend to recur with a more or less regular periodicity. The patient is apt to seek a cause for each new outbreak, and indeed exciting causes are generally present, but they would have been ineffectual if the time had not come for the pendulum to swing back. This tendency is of course seen to a much greater degree in the constitutional degenerative psychoses, but it may occur in a marked form in neurasthenia,[1] and in slighter degrees is very common.

The more important of the special symptoms may now be dwelt upon somewhat more at length, and it will be best to begin with the mental peculiarities, because they generally give the color to the case, and it is with them that the cure must begin. If the physician clearly understands their mode of growth, he can often help the patient to extricate himself from the web of dominant fears and morbid habits which so strongly tend to weave themselves around the nucleus of exhaustibility, and to get rid of some of his habit-pains and local weaknesses.

Mental Functions.—There are a good many cases, as has been said—and this makes the justification for the term neurasthenia—where the readiness to become fatigued constitutes almost the whole trouble. With sufficient rest and under a sufficiently protected life such patients feel well and cheerful and make useful and often highly intelligent members of society. In a great majority of cases, however, more or less elaborate intellectual and emotional disorders soon come in to complicate the situation. These consist mainly in quasi-logical outgrowths, based partly on the nervous exhaustion as such, partly on inherited nervous tendencies of various sorts. The mode in which these symptoms originate is sufficiently clear. The brain of the robust, healthy individual may be regarded as a machine for producing active results. Even the pleasures and the whole emotional life of such an individual tend to this end. The wider his experiences, the more numerous are the associative processes which converge to bring about a prompt and appropriate reaction to a given stimulus; and each set of cerebral processes, when their function is fulfilled, promptly yield their place to those which meet the needs of the next moment.[2] Increased difficulty only stimulates to more perfect co-ordination, and greater fatigue prepares the way for sounder repose. The self-consciousness of the healthy man is subordinated to the pursuit of outward interests. In the case of the neurasthenic all these tendencies are more or less changed. The associated processes of the brain fail to reinforce each

[1] See Oddo: "Circulare Neurasthenie," *Fortschritte der Med.*, May 1, 1895.

[2] Janet quotes Napoleon as having said that his thoughts were arranged, as it were, in drawers. Having done with one set, he could shut it up and take out another, or, if he wished, he could close all the drawers and then let himself fall asleep.

other, or do so haltingly and ineffectively, while inhibiting emotional states, or their equivalent in the subconscious mental life, become more and more firmly established as causes of new failure. With the experience of ineffectiveness comes the dread of failure, a consciousness of inability to command one's best powers under a stress of need, an exaggerated sensitiveness, self-consciousness, morbid reserve, timidity, self-depreciation, in all their endless forms; and these symptoms are liable to be exaggerated because of the patient's inability to get the sleep which should bring recuperation.

When it is considered that, in addition to this, the neurasthenic patient has usually more real reason to be anxious about his present and his future than the healthy man has, it is not remarkable that the morbid ideas which come to every one's mind at special moments should take a strong hold.

In the severer or more advanced or improperly treated cases we see a new set of symptoms, which are partly the logical outcome of those already mentioned, but which rarely occur in exaggerated forms unless in persons of strongly neuropathic tendency. Instead of simple timidity, we find "morbid fears," such as forbid the patient to be alone or on high places or in a crowd, a church, the theatre, or too far from his own home, or to travel on a train, or to cross open spaces, or such as give him a haunting dread of insanity or bodily disease or infection. The number of such fears is infinite. Eventually, simple depression may grow to a persistent tone of melancholy, anxiety, or apprehension (Angstneurose), to which violent attacks of vague fear associated with cardiac symptoms are sometimes added. An exaggerated conscientiousness may change to elaborate states analogous to the disease which the French have classified as *folie du doute* and *délire du toucher;* instead of morbid self-consciousness, we may find suspiciousness, suggesting the systematized sense of persecution. The diagnosis under such circumstances can be made only by estimating the essential tendency of the case taken as a whole.

It is certain that slight forms of these dominant ideas and impulses or imperative conceptions may occur under the influence of acute or chronic fatigue, and pass away with rest. Traces of such symptoms may indeed be observed in what must be called health, and neurasthenic tendencies, together with the mental and physical habits that they engender, contribute to their fuller development. Nevertheless, some of the best of modern alienists think it preferable to consider the persistent imperative conception, properly speaking, as marking a condition quite distinct from that of neurasthenia. The case is really somewhat like that of the relation of neurasthenia to hypochondriasis. The question is to be decided by the prominence of the part played by the hypochondriacal delusion—*i. e.* whether it is of primary or secondary significance.[1]

It is also a common experience that, just as morbid and depressive

[1] Those who wish to pursue this important inquiry further are referred to Cowles's "Neurasthenia and its Mental Symptoms," *Boston Med. and Surg. Journ.*, 1891; Krafft-Ebing: *Nervosität und Neurasthenische Zustände;* Thomsen: *Arch. für Psych. u. Nervenkrankheiten*, 1895, vol. xxvii. p. 319; Kraepelin: *Psychiatrie;* Diller: *Med. News*, 1896, vol. lxviii. p. 38; and the modern text-books of Psychology.

thoughts easily get possession of the fatigued or neurasthenic brain, and return again and again to make good their hold, so these patients sometimes fall a prey to the tendency to morbid inward repetition of words or phrases[1] or to an irresistible "counting" mania.

There is a momentary relief in giving way to these teasing temptations, just as it is a relief to scratch an itching spot, though we may anticipate bad results therefrom, or as rhythmical or restless motions give a temporary relief in states of fatigue or embarrassment. The recognition of this sort of tendency is believed to account for the fable of the Wandering Jew.

The tendency to exhaustion on the part of neurasthenics leads to another set of psychical phenomena which have to do with attention and memory. Such patients usually fall readily into abstraction and day-dreaming as a relief from the effort of thought, or they find themselves obliged to read a paragraph many times over before they can grasp the meaning. The effort of intellectual attention during fatigue-states may lead to drowsiness, or, on the other hand, perhaps after a renewed attempt at concentration, to excessive wakefulness (fatigue-anæsthsia, Cowles). Such persons are poor memorizers, and suffer easily from inability to recall names at will. Yet, with all this, the logical processes may be remarkably clear and sound.

The same tendency which checks the prompt and efficient action of the higher cerebral functions interferes also with those of the lower grade. Many disorders of this class could be mentioned, but among them a tendency to mistakes in writing may be mentioned, which lead to elisions and omissions of various sorts. Arising first as fatigue or inattention symptoms, such tendencies soon become habitual.

The *emotional tendency* of neurasthenics is productive of further mischief. Their peculiarities make no exception to the general rule that when an impulse fails to find promptly its due outward expression, it goes to reinforce the vague emotional states and to intensify their often pernicious effects in altering the conditions of blood pressure, vascular tension, digestive activity, and the like. The typical neurasthenic is, however, not especially liable to violent emotional outbreaks, such as characterize the hysterical patient, but rather to feelings of the depressive and hypochondriacal order.

Another symptom, which is at first only an exaggerated form of self-consciousness, is the tendency for the patient, in obedience to a sort of social instinct (Royce), to stand apart from himself and see or hear and criticise himself in the performance of acts of various sorts. Such patients have a painful sense that they are not spontaneous, but acting a rôle, and this gives a morbid quality to their relations to other persons which might, as an eventual outcome, lead to the conviction that those about them had changed. In a similar way arises the sense of having lost one's natural affection.

It is important to remember, especially in the interests of treatment, that the most important primary outcome as regards the mental life is, in most cases, the paralyzing sense of ineffectiveness and anticipation of failure which are so apt to quench fine impulses almost before they are born.

[1] See "Ueber Perseveration," *Wiener klinische Wochenschr.*, 1896, p. 905.

These symptoms might be universally present were it not that, on the one hand, a good proportion of people in general, including perhaps a smaller proportion of neurasthenics, are content to drift passively, and so to save themselves from being depressed by their lack of success; and that, on the other hand, intelligent neurasthenics often take advantage of their logical capabilities and their acute sensibilities to make a strong fight for the preservation of their will and courage, and strive to lose themselves in their true and best interests, with the result of reducing their ineffectiveness to the limits fixed by the amount of their original and actual lack of endurance.

It is this end which patient and physician alike should strive to reach, and it is the possibility of attaining it which justifies a careful study of each patient's mental peculiarities with a view to his mental education. In the end, if the education is to be successful, it must be carried to the point that the necessity of the struggle is not always present as a drag-weight in the patient's mind, but has given place to an instinctive acceptance of necessary limitations, and a habit of husbanding and utilizing force on a basis of cheerfulness and wide interests.

It is also important to remember that the hypochondriacal and depressive thoughts and fears which so afflict neurasthenics, and make them dread insanity or reproach themselves with weak-mindedness, are, as has been said, unlike the imperative conception of the insane person, in being secondary and relatively superficial, and so also relatively easy to throw off, for a time at least. They are thoughts and fears or imaginings such as pass through the minds of healthy persons in idle moments, but are soon dismissed. It is only because of his general state of anxiety that the neurasthenic thinks that in his case they have some more definite significance. If such a person can once persuade himself that he has the same right with a healthy person to think of bridges and towers as places from which people sometimes jump, or that razors are sometimes used for illegitimate purposes, while recognizing that the danger does not exist for him, he has made a solid gain in control.

It is perhaps an instinctive desire to find relief from painful and distressing thoughts and emotions that leads neurasthenic patients to foster a habit of keeping the mind blank, and this increases a tendency, already present, to limitation of the field of consciousness.

Those who have followed the psychological literature of the past decade know that this limitation of the field of consciousness, whether primary (Janet) or induced by anæsthesia (Sollier), is a characteristic mark of hysteria. The limitation in the case of neurasthenia is less marked and less prominent than this, and more within the patient's knowledge, but nevertheless does exist.

Disorders of Sleep.—These often usher in the chronic neurasthenic condition, and remain one of its most trying symptoms, always threatening to lead the way to worse things, which, however, strangely enough, often fail to appear.

Not every chronic insomniac, it should be remembered, is a neurasthenic, for the disorder may show itself as an isolated symptom in a person perhaps almost always of neuropathic parentage, but not necessarily showing typical symptoms of neurasthenia.

The varieties of insomnia and dyssomnia are manifold, and their immediate causes are various. In so far as they bring special indications for treatment they will be referred to under that head (see p. 593), and only a few peculiarities of the symptoms need be mentioned here. One of the most striking of these is the unrefreshing quality of the sleep when it does come, especially that of a nap taken in the daytime. The early morning hours are a trying time to neurasthenics, as to many melancholic patients, though sometimes the cold bath brings prompt relief. This inability to get refreshment is sometimes due to the fact that the patient is so far in arrears for sleep, but it sometimes occurs when he sleeps regularly and even with unusual heaviness. I have in mind a typical case of chronic neurasthenia in a man of early middle life and presenting the appearance of blooming health. In his case, as sometimes happens, a feeling of drowsiness is almost always present, making reading or writing almost impossible and suggesting strongly the action of a narcotic. Haig has suggested that symptoms of this class may, in fact, be due to uric acid, and points out that the amount of this substance in the blood is probably greatest in the morning when the blood is alkaline. But demonstrations of this hypothesis—important as the subject is—are for the future, and it is certain that changes of diet, such as Haig suggests, often fail to give relief. (See under Etiology, p. 556.)

Féré notes that the reaction-time, even with healthy persons, does not reach its greatest rapidity till the stimulating influence of light and heat has operated for some time, and nocturnal paralysis and morning tire are explained by him as exaggerated phenomena of this sort.[1]

The frequent occurrence of troubled dreams, that recall the persistent vexing thoughts which keep neurasthenic patients awake after going to bed, testifies to the partial character of the sleep when it does occur. It is to be noted that the loss of sleep, of which some patients complain so much, may leave them, nevertheless, with nutrition and functions unimpaired. In short, it is often light sleep rather than short sleep from which they suffer.

A really sleepless night often has a similar effect to a prostrating emotion in interfering with the tone of the arterial system and with the action of the regulatory apparatus of the heart. The pulse-rate is raised for a day or more, the heart may beat irregularly, flushing and chilliness occur more readily, and sometimes there is an abundant flow of limpid urine, as after nervous attacks of other sorts. Sometimes, however—and here the cause of the attack has a determining influence—the effect of the loss of sleep is less felt the day immediately following than at a later time, probably because of what Cowles has called the "anæsthesia of fatigue," an important and frequent symptom.

Attacks of insomnia, like many other symptoms of neurasthenia, are apt to occur with a certain degree of periodicity, which, however, is liable to be broken in a favorable or unfavorable sense by influences of many sorts.

Vasomotor disorders,[2] to one form of which attention has just been

[1] Cited by Bergström: *Am. Journ. Psychology*, Jan., 1894, No. 2, p. 264.

[2] Important studies as to the vasomotor and cardiac disorders in neurasthenia are given by Löwenfeld in his *Objective Zeichen der Neurasthenie*, München, 1892. The work of Lehr (*Die nervöse Herzschwäche*) there referred to is especially noteworthy.

called, are of fundamental significance in neurasthenia, and when the habit of their occurrence has become established it goes far toward intensifying various symptoms and opposing the restoration of health. The intimate relation of vasomotor changes throughout the body to cerebral activity, especially of the emotional sort, is familiar to every one, and the simpler and commoner vascular disorders of neurasthenia present phenomena of these sorts in an exaggerated form. Flushing of the face and head and ears occurs with undue readiness, and may be almost habitual or may alternate with pallor. The congestion of the conjunctiva may lead to low grades of inflammation, and the catarrh of the naso-pharynx which is so common is perhaps due in part to this cause. Every species of local irregularity of the circulation is observed, varying with the case and special conditions. The extremities are usually cold, but sometimes hot and flushed, and unilateral disturbances of either sort may occur.

It would be impossible to utilize here the immense number of interesting details as regards the vasomotor condition which have been collected by the many students of neurasthenia, and only the principal types and tendencies can be noted. Such are—(1) an entire freedom from such disorders. This is observable in light cases and where robust conditions of general nutrition prevail, but is, on the whole, rather rare; (2) exaggerations of the various peculiarities of circulation met with among healthy persons (flushed or pale skin, excessive emotional reactions, red ears, etc.); (3) the type characteristic of generally impaired asthenic nutrition; (4) the types characterized by special abnormalities of reaction (cramp-like contractions of vessels after cold baths, persistence of local vascular dilatations, as in dermography, "dead fingers," unilateral disorders of circulation); (5) marked and more or less permanent dilatation of even large arteries, associated with distressing pulsation. Dana[1] has described interesting cases of this sort, in one of which the internal carotid of one side was tied for relief of pulsation in the head, and with good effect.

It should be noted that, as throughout the history of neurasthenia, these special symptoms bear no fixed relation to the other phenomena of the case. As regards the pulse, it has already been said (Etiology) that high-tension pulses are often met with, but this is not invariably true, and the tension may even be habitually low. It is common enough to see neurasthenics, generally men of spare habit, with thin faces and tortuous temporals, whose pulses seem to bear out Löwenfeld's opinion that conditions of premature senility are present. Von Basch[2] does not agree that this form of tortuousness, even when associated with high tension, necessarily means arterio-sclerosis, and it is my own opinion that when this is present the case was usually, from the outset, something more than one of neurasthenia. Finally, it should be remembered that patients often complain of pulsation when nothing abnormal can be detected by the finger, so that local or central nervous hyperexcitability must be assumed to be present. The tinnitus of neurasthenics is partly referable to this cause, though it is more than probable that actual vascular dilatation plays an important part. These abnormal vascular

[1] "Pulsating Neurasthenia," *Journ. of Am. Med. Assoc.*, Jan., 1895.
[2] *Arteriorhigosis.*

conditions and reactions probably characterize the circulation of the viscera and account for some of their disorders.

Anjel[1] published in 1894 some experiments well worthy of repetition which seemed to show that the general tendency to irritable weakness affects the vasomotor centres in such a way that the stimulus of cerebral activity causes a convulsive response, while, on the other hand, the conditions required for persistent mental action are not assured.

Using the plethysmograph of Mosso, by which the changes in the circulation of the arm can be accurately measured, Anjel found that, whereas in health mental activity, whether emotional or intellectual, increases the flow of blood to the brain at the expense of that of the rest of the body, this readjustment fails, under the ordinary conditions of the experiment, to occur during neurasthenic states. Further observations showed that this failure was due to the fact that the nervous arrangements regulating the action of the vasomotor nerves in relation to cerebral operations are so excitable and mobile in severe and acute neurasthenic states that the vasomotor centres do not have their normal tone and power or response. The experiment is, so to speak, over before the observation can begin. If special means are taken to increase the tone of the vasomotor system, results more like the normal are obtained. These experiments not only give an excellent picture of the typical neurasthenic reaction in general, but they indicate a reasonable explanation of some of the most typical symptoms. They point, at the same time, to the importance, as regards treatment, of influences addressed to increasing the tonicity of the vasomotor centres, and also of the avoidance of the emotional stimuli which are so prone to excite these morbid vascular responses.

Anjel also found that these morbid reactions were most marked in the morning, and especially before eating, and suggests that the fatigue and lassitude of the early hours of the day are partly referable to this impaired circulation of the brain. It must, of course, be admitted, as regards these suggestive observations, that the neurasthenic conditions to which Anjel refers are relatively acute and severe. Nevertheless, acute fatigue occurs so readily with some neurasthenics that it may almost be regarded as habitual. The value of these observations is also reinforced by the result of those of George Oliver,[2] who found that the effects of posture on the size of the peripheral arteries is different in asthenic states from what it is in health. Ordinarily, the calibre of the radial arteries diminishes under the influence of recumbency and increases when the patient stands. In asthenic states the radial artery is small in the upright position, and generally increases when the patient lies down, probably, Oliver thinks, because of the contraction of the arterioles. These vascular peculiarities are perhaps not so characteristic of neurasthenia in itself as of conditions which frequently occur in neurasthenic patients; and perhaps the same may be said of the important observations showing that the pulse-tension of neurasthenics is frequently high (Webber, Edes, Löwenfeld, and others).

[1] *Arch. für Psychiatrie, etc.*, vol. xv.

[2] *Pulse-gauging*, London, 1895. The same writer has also brought forward in a recent series of addresses (*Lancet*, 1896) facts of much importance with relation to the vasomotor responses of the abdominal vessels and their failure in asthenic states.

The vascular relaxation which occurs so readily in neurasthenia sometimes leads to œdema, and, probably, at least contributes to the occurrence of nervous diarrhœa. Any one who is interested to utilize further the studies which have been made with regard to fatigue will find much that is of interest which cannot be referred to here.[1]

To one who does so the necessity for caution in drawing conclusions will become apparent. It appears, for example, that under certain conditions the brain may work actively for a time independently of the ordinary stimulus of the circulation; also, that the Mosso reaction does not invariably occur in health, since pleasurable emotions may cause an increase, instead of a decrease, in the peripheral circulation (Féré).

Cardiac disturbances are likewise common, and obey the general rule of neurasthenic disorders; that is, impairments of the higher inhibitory functions show themselves first, and then come disturbances of the automatic apparatus, until finally "habit" disorders may become established which may dominate the whole symptom-complex of the case.

Palpitation is the most common symptom, then irregularity, sometimes with præcordial or sternal distress or tenderness, while occasionally, but rarely, abnormal slowness of the heart beat is seen.

These affections occur under the form of abnormal responses to special excitations or become habitual for a longer or shorter period, so that, for example, the pulse may be constantly rapid or intermittent. Complex attacks, in which acute and intense general distress and nervousness, with asthmatic or sighing respiration or dyspnœa and faintness are prominent, sometimes occur, or præcordial sensations of painful character and disturbed action of the heart may form the central features. In one case where I was able to have some of these attacks carefully watched no constant change could be noted in the radial pulse, though sometimes real palpitation was present. In another case these attacks came on at times so suddenly as to suggest epilepsy.

"*False angina pectoris*," strongly suggesting structural disorders of the heart and making exertion impossible, may occur occasionally or recur persistently during prolonged states of neurasthenic debility of high grade.

Finally, the cardiac, like the vascular, disorders are thought by patients to be present oftener than they really exist, because of hyperæsthetic conditions of the central or peripheral nervous system.

I have often noticed that the familiar systolic whiff, due presumably to the displacement of air in the lungs, is heard much oftener with young, nervous patients with thin chest-wall than with more robust persons, and I have learned that the same observation has been made elsewhere.

An extended statement of the cardiac disorders and their outcome may be dispensed with here, because these affections are now best studied and described, at least in their severer forms, by those who devote themselves to diseases of the heart.

It may be said, however, that structural changes of serious amount are a rare outcome of the disorders of innervation, though the latter no doubt act now and then as contributive causes.

Disorders of Digestion.—On the grounds just cited we may also

[1] See Bergström, *loc cit.*, and Féré, *Path. des Emotions.*

spare ourselves a lengthy discussion of the neurasthenic *disorders of the digestive tract*. Their very frequency and importance has made them objects of study for every physician, and in the monographs upon digestion their relations upon other digestive disturbances are fully considered. It is now a matter of common knowledge that with neurasthenic patients distress during the digestive process is not necessarily attended by a corresponding ineffectiveness of the function, as indicated by observations with the stomach-tube; while, on the other hand, it cannot be said that the presence of neurasthenia excludes the possibility of the occurrence of any or all of the actual changes, such as hyperacidity or insufficiency of the secretions, or a lack of motor reactions, or atony, dilatation, and the different forms of gastric catarrh.

The same is true with regard to digestive functions of the intestines, and it must not be forgotten that in the case of both the functions of the brain, even the higher functions, play an immensely important part.

Not only the stomach and the intestine, but, though to a less degree, the functions of all the other organs which are concerned in the digestive processes, may become impaired. In some cases the digestive disorders are initiated by profound impairment of appetite or of the power of retaining the food in the stomach, which are of distinctly psychical origin.

Bad cases of nervous indigestion, even where the actual nutritional disorders of the gastric and intestinal tracts are not of serious degree, may be among the most dangerous and difficult cases with which the physician has to deal, so extreme and far-reaching are the prostration of the nervous forces and of the general nutrition.

The form of the neurasthenic complex which is associated with protracted indigestion, especially if that be in the form of intestinal catarrh, is somewhat peculiar, in consequence of the intimate relationship which exists between the digestive function and emotional state, and also, perhaps, because of the susceptibility of the neurasthenic nervous system to the toxic substances set free in the intestine. Profound hopelessness is not uncommon in such cases.

The mistake in practical diagnosis in cases of nervous dyspepsia is likely to be that of taking too narrow a view of the relation of the symptoms to actual digestive incompetence on the one hand, and to psychical conditions on the other.

It must not be assumed that because the diagnosis of "nervous dyspepsia" appears well grounded the patient should be able to digest anything and everything with equal ease, even though the test-breakfast is taken care of in due course; nor, on the other hand, should it be overlooked that great skill, experience, and patience are often needed for the due recognition and management of the psychical factors in a given case. In the former condition even stomach-washing (perhaps mainly on account of the douching of the walls) is sometimes unexpectedly useful, while absolute isolation in bed, and even hypnotic suggestion as a last resort, may be required for the latter.

The psychical influence is especially marked in exciting such symptoms as sudden watery diarrhœa, which may occur in attacks, as a result of auto-suggestion, under trifling emotional excitement, as an exaggerated

form of a symptom met with in health. A patient now under my care, an intelligent professional man, cannot put himself under any conditions where a loose movement would be embarrassing without being in great risk of such an attack—another instance of "association neurosis" analogous to habit pains. In similar manner absurd but obstinate idiosyncrasies of appetite and digestion arise. Thus, one of my patients finds the slightest morsel of cheese to act "as a poison," and most neurasthenics think themselves unable to bear milk. A possible cause for this latter peculiarity is that, having taken milk in earlier days so often when they were suffering from indigestion and nervous symptoms, they have mistaken juxtaposition for cause, and then have allowed auto-suggestion and errors of observation to do the rest.

For the significance of *enteroptosis* in neurasthenia see under Etiology, p. 558.

Sexual disorders play an immense part in the etiology and symptomatology of neurasthenia, and of late a number of important monographs have been published, mainly in Germany, which deal with this relationship.

We do not, in neurasthenia, find the great perversions of the sexual instinct that are met with in states of deeper psychical degeneration, but instead we do see every conceivable disturbance of the different parts of the sexual functions, mixed in varying proportions, but traceable to weakness and irritability of the sexual centres in the spinal cord and the brain; and not only of them, but also of the functional centres with which they stand related, the vasomotor and cardiac, the respiratory, the urinary, the intestinal. Furthermore, the emotional relations of the sexual function are so widespread that the mental disorders of sexual neurasthenics are especially severe and numerous, varying from hypochondriacal depression to serious imperative conceptions.

Most of the sexual symptoms are so well known that only reference to the tendencies which are at work will be needed. The simpler forms are exaggerated pictures of normal peculiarities. Excessive reactions of the general nervous functions mark the onset of puberty. Nocturnal emissions, with or without a basis of masturbation, may occur with abnormal frequency and lead to the more important forms of spermatorrhœa and prostatic irritation. Sexual desire is sometimes deficient, but usually easily excited, while sexual capacity is poor and often early to disappear in middle life, and the sexual act is frequently unduly depressing in its effects. Sexual irritability may disturb sleep, and thus adds one centre of disturbance the more to the many that exist, at least where persistent irritations of the genital tract are present, from which circles of emotional disorders spread.

The sexual functions play such an immense part in the emotional life of many individuals, and their exercise is so often the cause of considerable temporary exhaustion, that it is not surprising to find these disorders recognized as potent causes of many psycho-neuroses. Within the past few years several large and important monographs have been written upon this subject, especially by German physicians, but all that can be attempted here is to indicate the lines upon which progress is being made.

The sexual instincts, though not consciously recognized as such, are

the basis of much of the emotional instability of early puberty and middle life, not to speak of the fact that they are the principal basis of marriage, and responsible, directly or indirectly, for the vast influences for good and evil which that state introduces or which cluster around the unconsummated desires and hopes of new manhood and womanhood.

It is important, and yet difficult, in attempting to define the true influence of sexual disorders as causes of neurasthenia, to separate the effects of the sexual functions, regarded as purely physiological processes, from those of the emotional states associated with them.

This is eminently true of *masturbation*, but the best modern opinion is in favor of the view that if it was possible to separate the act itself from the sense of wrong-doing and humiliation and unfavorable engrossment of thought, it would not be more, perhaps less, injurious than sexual intercourse of the same amount—a matter to be decided for each special case. The same is true of *seminal emissions*, but in both cases it is certainly true that after a time a highly morbid irritability of lower or higher nervous centres may be set up, which is both a sign and cause of exhaustion.

Similarly, throughout the period of "engagement" preparatory to marriage, and throughout married life, every degree and kind of sexual excitement which is notably in excess of that which is necessary for the maintenance and moderate exercise of the function is, in proportion to its amount, injurious to the robustness of the nervous system in all its parts, though at the same time this injury need not be of great amount, and is materially accentuated by morbid self-reproaches leading to no result. It may be permissible to anticipate the section on Treatment to the extent of saying that the patient or physician who finds himself confronted by sexual disorder, calling for treatment by self-control, will be bound to fail if he neglects to estimate the strength of the instinct with which he has to deal, and to equip himself correspondingly for the attempt.

It is important to note that the momentum of an unnaturally strong sexual instinct, and the difficulty of controlling it, become all the greater from the fact that after a time it leads to functional disorders of the heart and vasomotor system, occurring in occasional outbreaks, and seriously disturbs sleep and other functions, though these affections may be, at the same time, independent of conscious excitement.

Forced or voluntary *abstinence from sexual intercourse* occasionally, though rarely in normal individuals, accentuates neurasthenia, and *impotence* leads indirectly to the same result.

The *sexual perversions* need not be studied here, as the psychopathic disorders with which they are associated are usually far more serious than simple neurasthenia. (See Sexual Psychoses.) It is only necessary to say that they should not be considered as manifestations of a fatalistic and implacable destiny, but as habits growing easily on a morbid soil, but often preventable and curable, and of—so to speak—accidental origin.

Head-pressure and Headache; Backache; Ovarian Pains; Muscular Pains; Joint Pains; "Habit" Pains; Paræsthesias.—Sensory symptoms of these different classes are eminently characteristic of

neurasthenia. The tormenting sense of pressure referred to the back of the head, the vertex, the temples, the orbits, is almost invariably present, either constantly or during times of fatigue and strain. The cause is unknown, and nothing is gained by assuming congestion or anæmia. The pressure is often associated with tenderness of muscles and with creaking vertebral articulations, or may be exchanged for sensations of burning or, more rarely, of cold. Sometimes the entire head feels as if compressed in an iron helmet (Charcot), while at the same time streams of pain invade the back of the neck, the eyes, the ears, the throat. A patient under my care has suffered intensely in this way for months without intermission except at night (for these sensations rarely disturb sleep), although but few other special, localized signs of neurasthenia are present.

The distressing backache, of which "spinal irritation" is the severest form, the coccygodynia, the ovarian pains, need but to be mentioned.

The least familiar point with regard to them is that, like all the other symptoms of neurasthenia, either one of these may take on a preponderating importance, so that unless a close examination is made it may seem to stand alone and to indicate some local disease. These painful affections, and others like them, shade into the "*habit*" *pains*, a name which is especially fitting for the localized pains, not otherwise classified, that affect one or another part of the body where some local disturbance—a sprain, an inflammation—has been present, but has passed away. They are of psychic origin, and so might be called hysterical, but they occur in typical cases of neurasthenia.

In like manner, the back, the coccyx, the limbs, chest, the abdomen may be the seat of tenacious habit pains due sometimes to a local cause long since banished. It has already been noted that the intense sense of fatigue which sometimes comes on so suddenly and on such slight provocation may be a habit pain of this order.

Joint pains and muscle pains may belong in a somewhat different category. Inflammations of low grade about the joints ("arthritism"), especially the finger-joints, belong to the nutritive disorders of neurasthenic origin, and so also do pseudo-rheumatic affections of the muscles or muscular nerves, which seem to underlie the very common muscular tenderness and soreness which is increased by bad weather and fatigue.

In certain cases, however, of which I have seen two marked examples, the slightest muscular exertion may bring on severe muscular aching, not of inflammatory origin, which is very slow to subside. The patient to whose case I mainly refer has been almost a prisoner for years on this account. Such cases lead to the *akinesia algera* (motion restricted by pain), so called by Mœbius, who first described it.

The hands and feet of neurasthenics become *paresthetic*, or "go to sleep," on very slight pressure over the nerve trunks, and even without any very definite pressure. At times the paræsthesia may occupy the whole half of the body or an arm and leg, a hand and foot. One would be inclined in such cases to make the diagnosis of hysteria, but it may at least be said that the other symptoms often fail to bear out this view.

Affections Related to Sight and Hearing.—*Asthenopia*, often on a basis of slight error of refraction or weakness of the ocular muscles,

is sometimes so prominent a disorder that the case appears to turn upon this symptom alone, though careful inquiry elicits many others, among which photophobia is likely to be one. It is generally, to a greater or less degree, a habit symptom, and partially relievable by treatment of which systematic training and other forms of mental influence are prominent parts.

The asthenopia is probably sometimes muscular, sometimes of retinal origin, or both sorts of weakness may be combined. Wilbrandt has recently called attention to an important test, which consists in determining the field of vision by means of phosphorescent disks in a dark room. It is found that even with healthy persons who have just come in out of the light the field of vision is smaller when tested by the faint phosphorescent light than it had been in the daylight, but that it returns in the course of ten or fifteen minutes to normal limits. With neurasthenics and hysterical patients the restoration may take place eventually to an equal degree, but a much longer time, even several hours, may be required for this result.

Tinnitus and noises in the head, often associated with vertigo, and sometimes with more or less nervous deafness, may be classified in much the same category with asthenopia. The subject is an important one, and has lately been skilfully treated in a brief article by Cozzolino,[1] of which I give a brief summary because of the importance and obscurity of the problems which come in question. Cozzolino distinguishes between an idiopathic oto-neurasthenia, an affection presumably of the nervous portion of the auditory apparatus, and a secondary form in which the nervous disturbance is associated with disease of the middle ear, leading to changes in labyrinthine pressure. In the idiopathic form the symptoms are often, not always, bilateral; in the secondary, or, more properly speaking, the "associated," form, unilateral—*i. e.* they attach themselves mainly to the diseased or most diseased ear.

There is often hyperacusis (corresponding to photophobia) in the essential form, and vertigo varying in severity from a slight to an intense degree and occurring sometimes in violent paroxysms (Ménière). This form is but slightly amenable to treatment, while the secondary form is more apt to be associated with general neurasthenic symptoms (cerebro-cardiac form), and, like them, to be in some degree susceptible of cure. The slighter attacks of vertigo in these cases are not unlike those of pure neurasthenia, and, in fact, even when the middle-ear disease exists, it is to the neurasthenic tendency that the vertigo is mainly due. In general the bad forms of vertigo are signs of a degenerate brain.

The "secondary" form of oto-neurasthenia is apt to be associated with the "helmet sensation," and is a cause of mental depression.

Vertigo is a troublesome symptom and presents itself in a variety of forms. It may closely simulate the reflex vertigo of aural disease, or may be based upon slight degrees of this affection, or may be associated with psychical states like embarrassment or inability to trust one's self on high places. Again, it may be brought on by slight disorders of digestion, disturbances of the action of the heart, eye-strain of different sorts, or by forced position of the head or sudden movements of the body.

[1] *Ann. des Mals. de l'Oreille et du Larynx*, 1894, p. 911.

It will be seen that most of these forms merge into those which may occur under special conditions in health, and, indeed, a healthy person may make the observation, if he stands upon a high place, that he can bring on or dissipate, at will, a tendency to be dizzy or not, by lending himself to one or another set of thoughts.

The vertigo may, in exceptional cases, be so severe as to cause the patient to stagger or even fall, but generally it consists in an annoying sense of giddiness, the worst feature of which is it absorbs the patient's attention and increases a tendency to hypochondriasis.

The *volitional motor functions* are not exempt from signs of weakness which may strike at the whole nervous system, now here, now there, with special force. Here also a great variety of forms occur in which sometimes the sense of weakness—*i. e.* the more psychical form—predominates, while in other cases the trouble seems to lie less in the sensations of the patient than in the inefficiency of the spinal or peripheral motor processes. The motor weakness may go so far, as in a case formerly under my care, that a walk of a dozen rods or the use of the arms in driving may give rise to a painful prostration; and these cases perhaps form a connecting link with those described by Mœbius under the name of akinesia algera, or muscular inefficiency induced by pain. Cases also occur, of which I have seen two striking instances, where an extraordinary exhaustion on muscular effort occurs, not necessarily associated either with pain or with the usual symptoms of chronic neurasthenia to any high degree. It is somewhat doubtful in what category these belong. The weakness of the eye muscles has already been alluded to.

The *secretions* are liable to be disturbed, so that, for example, the skin may be abnormally dry or abnormally moist or the secretion of saliva diminished or excessive. The *sweating* is sometimes so severe as to be seriously annoying, and this symptom may occur paroxysmally, not only both in the neurasthenia of the menopause, but also with young and strong male patients.

The urinary symptoms have been clearly described by Dana[1] of New York, who thinks that the examination of the urine can be made use of to great advantage in the classification of neurasthenias. In the forms characterized purely by nervous symptoms of the degenerative type there may be no marked "vice of nutrition" or change in the urinary secretion. In all other forms, he thinks, such changes are present, showing themselves as lithæmia, glycosuria, phosphaturia, oliguria, polyuria. Of course the disorders in quantity, if they occur spasmodically, are apt to be dependent largely upon vasomotor disorders, and may occur in nervous conditions of any sort where these are present, especially after special outbreaks or after special fatigues or loss of sleep. In a good many chronic cases, however, Dana finds (agreeing, in the main, with Haswell)—first, that in certain types of neurasthenia the amount of solids excreted by the urine is regularly small as compared with the amount of food taken; second, that in other types the amount of solids is large and consists in part of products of incomplete metamorphosis; third, that in others, again, there is polyuria. In cases of the first type he thinks there may be occasionally a real renal inadequacy, and

[1] *The Medical Record*, Jan. 16, 1886; *The Dietetic Gazette*, Oct., 1888.

this being perhaps the first indication of a cirrhosis of the kidney. The patients of this type are generally old or feeble or have degenerated vessels. The patients of the second type are "cerebral" and "gastric," "irritative" or "diathetic" cases.

These types certainly exist, but our comprehension of them is vague, and we need, especially, completer analyses of all the excreta as an aid in their analysis. Löwenfeld[1] has analyzed the newer data as to the urinary secretion with great care. He refers the polyuria and the oxaluria, in the main, to an affection of the thirst function (polydipsia), the phosphaturia to a neurotic disorder of tissue-metamorphosis, and thinks that the (vaguely understood) disturbance of metabolism to which we give the name of uric-acid diathesis is a partial, though only partial, cause of neurasthenic conditions.

General Nutrition.—It is usually said that neurasthenics may present a picture of blooming health; and, indeed, this is true, though the statement applies much more to the psychopathic than to the strictly neurasthenic cases. As a rule, however, characteristic disorders of nutrition can be detected. The so-called *stigmata of degeneration*—such as are, to a certain extent, characteristic of the more serious constitutional nervous diseases—cannot be considered as characteristic of the neurasthenic condition pure and simple. This, at least, may be said of those manifold deformities or deep-seated disorders of nutrition which point to a high degree of bodily degeneration.

The fact has already been alluded to that neurasthenic patients are apt to be of one or another type of bodily habitus, and it is unnecessary to repeat the descriptions here. The pupils are usually large, even in the absence of anæmia, and it is important to note, in view of the fact that this sign is one of ill omen in other respects, that differences between the two pupils are occasionally observed, though they are transitory. The teeth frequently decay early, the nutrition of the hair is likely to be poor, the voice may be thin and lacking in firmness, the hands and feet moist and cold, there may be a movable tenth rib [Stiller], and poorly developed genitals.

General Course.—As a rule, the onset is insidious, and even if the disorder originated in illness or accident, a certain period generally elapses before the characteristic symptom-complex is established, though a skilful eye would commonly have been able long before to detect signs of mental instability or weakness. The first symptoms to attract attention are usually the failure of some particular function, a sexual weakness, distressing sensations about the head, an impairment of digestion, weakness of the eye, poor sleep, sense of fatigue, depressing thoughts; and gradually these disorders tend to become accentuated by auto-suggestion and absorb more and more the current of the mental life.

Sometimes, and especially during the later years of puberty, a somewhat severe general collapse may come on after these symptoms have been present for a longer or shorter time. Girls at school and young men at college often suffer in this way, and the more so, of course, if the influence of some special cause comes in. Under appropriate treatment substantial recovery may then take place, but it happens oftener

[1] *Die Objectiven Zeichen der Neurasthenie*, München, 1892.

that the disease becomes established in a chronic form, though the type varies greatly as regards seriousness. In the lighter cases the patient has simply to take an extra amount of care, and after a time even this may become so habitual that unless he is subjected to some unusual strain his friends will cease to regard him as anything more than "somewhat nervous." In the severer cases the patient may be a continual sufferer and in continual danger of becoming hypochondriacal, and may be either wholly incapacitated for work or compelled to work at the cost of great fatigue and occasional collapse. Or one function may suffer disproportionately, so that the patient may think and say that if it were not for his eyes, his stomach, or his bowels, etc., he would be well enough; but, in fact, these disabilities, and the disorders of the digestion above all, may make him a serious invalid.

The power of attention, the memory, the will, are apt to become involved early, and in equal step with the impairment of the capacity for effective thought and work; the emotional reactions come to play an unduly important part. Weakness is apt to lead to failure, timidity, and morbid self-consciousness, and then definite morbid ideas and impulses may become established.

Certain neurasthenic patients are liable to suffer from time to time, usually after prolonged strains which are not necessarily very severe, from sudden and serious *acute collapses* of indeterminate duration. It is often hard to say why such attacks, so out of proportion to the cause, should occur, but it is probable that a disturbance of the higher cerebral centres—such, for example, as follows acute and overwhelming moral shock or fright—is chiefly involved.

In such attacks the efficiency of the various functions of the nervous system may sink to a low point; the temperature may fall to below normal; mental effort is out of the question; and in the worst forms, which, to be sure, are rare, even death may occur, though this is very unusual.

Diagnosis.—We need to speak here only of the affections with which neurasthenia might be confounded, and several of these have already been sufficiently alluded to. The most serious danger is that of confounding neurasthenia, on account of such symptoms as mental depression, temporary aphasia, persistent headache, with some more serious localized brain trouble, or, on account of morbid and impulsive thoughts and acts or hypochondriacal delusions, with progressive forms of insanity. As regards the former class, only a careful study of the cause, with conscientious search to determine the presence or absence of localizing signs, can, in some cases, justify conclusions, and it must be recognized that the neurasthenia may be symptomatic, and perhaps almost the only symptom, of such a disease as tumor of the brain. As regards the latter class, the relation of the delusions and the depression to the general state is important. In hypochondriasis of serious moment the delusions are the chief thing, whereas in neurasthenia, distressing as they are, they are relatively superficial and secondary, and analogous to those seen in healthy persons. The same thing is, in general, true with regard to melancholia, though for all cases of this class we have to remember that we may be dealing with a real first stage of a severer trouble.

There is sometimes considerable difficulty in distinguishing between

neurasthenia and an early stage of *general paresis*, the more so that in the former malady a transitory difference between the pupils, disturbances in speech and handwriting, and exaggerated knee jerks may be present. Here the diagnosis depends upon a study of the case as a whole and the behavior of the patient at different periods, as well as on a strict analysis of the individual symptoms.

The most common mistake which is made in the diagnosis is that of confounding neurasthenia with the affections which come nearest to it—namely, simple "*nervousness*" and *hysteria*. To a certain extent this difficulty is insurmountable, since these affections shade into each other, or, to say the least, overlap each other, in such a way that it is impossible to draw a sharp line between them. It is, however, important to recognize in any given case the different tendencies that are at work. Every one who is familiar with the subject must admit that there are large numbers of patients whose nervous systems show reactions of irritability in many forms, and yet who are neither neurasthenic nor hysterical.

For clinical purposes it is sufficiently exact to classify these patients as of "nervous temperament," not even applying the term neurasthenic to them, unless, in addition to their unnatural mobility of reaction and their tendency to nervous outbreak and eccentricities, they show actual signs of exhaustibility. On the other hand, it is best to reserve the term "hysterical" for those patients who show at all times, or as a rule, characteristic disorders of special or general sensibility, or motility, or disturbances of the visceral functions of analogous sorts. Attention has already been called to the fact that the sense of exhaustion sometimes comes and goes with such rapidity as to suggest that it belongs with symptoms of "auto-suggestive" origin characteristic of hysteroid states. Similarly, patients who seem to be neurasthenic often fail to improve under treatment appropriate to that condition, but get well promptly under "mind cures" of one or another sort.

Prognosis.—In the majority of cases of neurasthenia we have to deal with a chronic constitutional weakness which cannot be expected to pass entirely away, though under favorable conditions active symptoms may be generally in abeyance and the patient may lead a useful and pleasant life.

In cases where the symptoms come on in adult life after an illness or some special strain complete recovery may be expected, provided the native neuropathic tendency is not too strong. An estimate of the degree of this tendency must in every case enter into the prognosis, and it is therefore desirable to have some method of classifying cases in this respect. Koch's analysis of the whole great group of affections (not otherwise distinctly tabulated) that occupy the borderland between health and pronounced mental disease, to which he gives the name of *psychical incompetencies* (Minderwertigkeiten), are painstaking and valuable. He classifies the psychopathic conditions, according to their severity, under the headings of psychopathic tendency (Disposition), psychopathic taint (Belastung), and psychopathic degeneration. Strictly speaking, it must be remembered that when we estimate the future of a neurasthenic patient with the signs of one or another of these states, it is his prospects as an individual that we consider, not that of his

neurasthenia regarded as a separate disease. This distinction is not purely one of academic interest, for it involves that of the relation of neurasthenia to the psychoses.

How much danger is there that a given patient with neurasthenia will go insane, a possibility so often dreaded to the very verge of anticipation?

The answer depends a good deal on how one looks at the matter. Cowles has pointed out, and with justice, that many forms of insanity pass through a preliminary stage of neurasthenia, and that much good may be done for them by helping to throw down the structure of morbid ideas and impulses which has arisen on that basis. But may we not expect through close observance of diagnostic marks to recognize the destiny of the majority of these cases at an early period? Some advance at least is likely to take place in this direction, and meantime it is certain that a person may be highly neurasthenic—timid, anxious, and depressed to the verge of despair—for many years, and yet go no farther.

The outlook for improvement under favorable conditions and proper treatment is fairly good for any purely neurasthenic patient who has still enough intelligence, elasticity, courage, and interests to really care so much about getting better as to be willing to make serious sacrifices therefor.

The lighter cases often do surprisingly well, and I have been gratified to learn through correspondence with a number of patients that the improvement may maintain itself satisfactorily even after the cessation of treatment.

On the other hand, it cannot be overlooked that the prolonged strain under which many neurasthenics must live, and the serious disorders of digestion and metabolism from which they sometimes suffer, may lay the foundation of constitutional diseases, or, even without the intervention of these, many pave the way for a fatal exhaustion. Serious cases of this sort may follow railroad disasters or other causes of severe shock.

TREATMENT.—We have concluded that neurasthenia is due partly to structural or chemical peculiarities of the cells composing the central nervous system, partly to "abnormal" co-ordinations—*i. e.* co-ordinations expressing themselves in reactions unfavorable to the healthy physiological and social life of the individual. Both of these sorts of influences are of such a kind that they impair the endurance of the nervous system on the one hand, and lead to the establishment of fixed morbid habits of thought and action on the other hand. To these are often added morbid habits affecting the organic, nutritive life, stereotyped by years of repetition. In accordance with this view, the object of the physician should be, on the one hand, to restore the affected nerve elements to a normal state, either by providing a nutritive material peculiarly suited to their needs or by supplying more favorable external conditions for their nutrition; on the other, to secure a more healthy action of the nervous system as a whole by influences which may be classed as educational, in the widest sense of the word.

I shall therefore discuss first the modes of treatment which might be called in some sense "specific" or "causal;" then those which have to do

with the hygienic, the social, and the psychical life of the individual; next, certain special treatments, as baths, electricity, etc.; and, finally, the treatment of certain special conditions and symptoms.

Prophylaxis.—Before taking up these problems it will be worth while to speak of the *prophylaxis* of neurasthenia, for this involves questions which demand the best thought of the public-spirited physician. There is no doubt whatever that the children of neuropathic parents are very liable to become neurasthenic, if nothing worse, and this fact ought to be realized, even though it may be legitimate, in a given case, to conclude that the danger is not so great as to forbid marriage, especially in view of counterbalancing advantages. A moderate amount of neurasthenia is by no means incompatible with happiness and usefulness, and, although itself a heavy burden, it may be associated with qualities which are eminently valuable. On the other hand, from the same soil which gives rise to neurasthenia grave neuroses and psychoses are likely to spring, which the neurasthenia may help to develop.

The mother should, in any event, take great care to lead a quiet and healthful life during gestation; and it must not be forgotten that there is some evidence for the view that an unfavorable condition of the parents, perhaps especially the father, at the time of conception may make itself felt on the offspring.

As regards the infant, it is certain that judicious training in self-control may begin at the cradle, and even parents of nervous temperament can often be led to adopt sensible measures for dealing with their infants. A suitable nurse should be chosen, and the child trained to be independent of complex and stimulating amusements, and to depend upon its own resources for getting sleep and entertainment. It is generally considered to be better that a highly nervous mother should not nurse her own child at the breast, provided, of course, that equally good nourishment is to be obtained in other ways. As the child grows older two principles of education become more and more important. One is that the nervous system exists for action, for performance, and that emotion and impulse fail of their usefulness and are actually harmful if they do not lead to this end. The child should be trained to *act*—to bear responsibilities adapted to his powers, and systematically to overcome obstacles. The kindergarten, if well managed, is a valuable adjunct in these respects to the training of the nursery and the fireside.

It is not meant that the emotions and the imagination should be left to lie fallow, for they are the mainspring of action; but if the bond which should unite them to the motor results is not firmly and quickly established, the child may be left with habits of indecision and a sense of helplessness instead of strength. On the same principle, it is of little use to "preach" to a child or to make it feel regret for shortcomings, "on theory," as it were. There are two paramount influences in moral education—that of the individual's own acts, and that which comes from contact with those around, and which might be called the influence of "imitation" in a general sense. The second principle is that good habits should be made to take the place of repeated voluntary effort just so far as the substitution can be made without sacrifice of the result. The child with neurasthenic tendencies is none too likely to err in being over-systematic and business-like, and will gain from acquiring

fixed habits in these respects. Of course it is to be understood that the establishment of a habit is not to be allowed to check the further growth of conscious effort, but as a starting-point for advance.

As the child grows older the problem of school-training comes to the front. There seems to be little danger in this country that children under twelve or fourteen will suffer, under present conditions, from mental over-pressure in school, but for older children this danger sometimes exists. It is a great strain for a child to keep its attention closely fixed for more than a few moments at one time, even when interest in the subject is the motive impulse, and restlessness is often its natural mode of seeking a needed relief. On the other hand, neurasthenic children, above all others, need thorough training and preparation at each stage for that which is to come next, in order that they may not be forced, either during school life or professional life, to compete at a disadvantage with those who are better prepared.

Individualization in teaching is especially important for them, but this does not mean that they may not gain in robustness by taking common lot with other children in certain directions. The point is that the child should be developed, not forced into a mould.

It is getting to be generally recognized that quality of mental achievement counts for more than quantity of acquirement; character for more than quickness; wisdom than knowledge. For inculcating these powers the personal characteristics of the teacher are of higher importance than his learning. Again, the child cannot acquire nor the teacher inspire effective enthusiasms for high ideals with a fatigued brain, and the questions become important, To what extent is fatigue observable among school-children? How is it to be recognized, and how avoided? The literature on this topic, both abroad and at home, has rapidly grown during the past few years,[1] and the Germans especially are becoming aware that their enormous thirst for knowledge is driving many weaker pupils to the wall. There is a growing acknowledgment of the need of medical inspection of schools and school-children, and the researches of Warner and others have already shown how large a proportion of the school-children bring defective nervous systems, which the school-life is likely to make worse unless it sets out to counteract them.

Close analysis[2] has shown that the sensibility of the skin as shown by measurements with the æsthesiometer, estimation of the power of working with numbers, and other tests of like character are reliable means in skilled hands for determining the advent of fatigue. More available for the ordinary teacher are the movements of a child. "Fidgets" usually mean fatigue, and they bring a double indication: first, that something must be done to prevent the fatigue itself; second, that the child who thus shows itself ill provided with endurance is doubly in need of learning to make systematic effort. Of course, withal, the possibility that the restlessness means only indifference and ill-breeding is not to be forgotten.

Extremely important influences in the life of the growing child are

[1] See *Outlines of School Hygiene*, W. H. Burnham; *Pedagogical Seminary*, vol. ii. No. 1, with references; Dr. Warner's report of the examination of fourteen London schools; and various papers by Eulenburg and others in the German periodicals; also, *Energetik und Hygiene des Nerven Systems, etc.*, Griesbach.

[2] Griesbach: *Loc. cit.*

a faulty *moral* training and contact with a temperament like that which the child himself is in danger of showing at a later period.

The psychological researches of the past few years have demonstrated to what an amazing degree our mental tendencies for good or evil are dependent upon conscious and unconscious imitation and "suggestion," whether by words or acts of parents, teachers, comrades, and the community. Cheerfulness is a duty. Pessimistic parents and friends help to make of nervous children neurasthenic men and women.

In our attempts to guide children on the right path it is to be remembered not only that acts speak louder than words, but that ill-judged words which are meant to convey a suggestion of a favorable order, whether in the form of encouragement or rebuke, often act in reality by exciting what the hypnotizers call a "counter-suggestion." Let the child once detect a false note in a suggestion "not to be afraid" and the like, or be led to take a contrary position in regard to any moral act, and the chances are that the strong tendency to self-imitation will confirm him in his perversity. This points out an important difficulty in moral education, but it is a difficulty which must be met, and when the parent's or teacher's course is not clear to himself, it is best not to run risks by attempting too much, and, above all, to be plain and honest in dealing, especially with nervous children.

It is self-control, ability to make a choice and stand by it, readiness to postpone a present pleasure for a distant result of greater value, independence in work and entertainment, hardiness in endurance, that need to be cultivated, while every influence is to be avoided that might tend to morbid self-reproach and self-distrust. These are the shadows and the germs of more serious dangers.[1]

Nervous children are especially prone to *masturbation*, and especially likely to suffer from its ill effects, of which the chief are a sense of humiliation and dread on the one hand, and on the other irritable weakness of the primary sexual nerve arrangements, together with hyperæmia and catarrh from vascular and general muscular relaxation of the generative organs. All of these effects prepare the way in their turn for the development of serious neurasthenic symptoms. The best means which the parent or teacher has at his command for warding off this danger is by early and friendly explanations, and, fortunately, this is often effectual. It should not be left to schoolmates to introduce the child to the knowledge of his sexual life.

The advent of *menstruation* and *puberty*, the increase of *social pleasures* and *excitements*, the engrossments and strains of *college life*, the entrance into *business*, all bring their serious dangers and problems demanding perpetual thought and care.

With delicate young women of nervous tendencies the risk of paying too much attention to slight complaints of fatigue, etc. is only exceeded by the risk of paying too little attention to them. It is my experience that on the whole college-life is useful to young women, though it unquestionably introduces special dangers. It should not be forgotten, in estimating the importance of these, that even under the most favorable circumstances, where exercise and outdoor life can be freely had, and where, to all appearance, no specially morbid inheritance seems

[1] See editorial, *Med. News*, Dec. 28, 1896.

present, the health may break down during these important years. Both parents and physicians must learn, so far as that is possible, to distinguish between the child or young person who is best treated by having the details of his daily life mapped out by some one else, and the one who is best treated by being taught to exert his own intelligence and fortitude.

The adult neurasthenic patient may present himself for treatment on account of *acute, chronic, or localized symptoms*, and not only the characteristics of the disease, but those of the patient, have to be considered as a guide to the course to be pursued. To a great extent, however, the principles of treatment are the same throughout, and the most important are the following:

First of all, the physician should have a clear conception of what could conceivably be accomplished provided the patient was willing and able to make the necessary sacrifices, and provided the best modes of treatment were at his command. We are still too much under the traditions of incurability with regard to neurasthenia, such as prevailed with regard to phthisis half a century ago. To-day the phthisical patient will be at endless pains and expense to regain his health, because it has been found that in this way great benefit can be obtained. And so the neurasthenic patient may often be assured that he can get relatively strong and self-reliant if he is willing to make sufficient sacrifice of luxuries and comforts and pleasure-giving susceptibilities of temperament, and if he does not demand that he shall be rid of all his troubles, but only the worst. He must have high hygienic and ethical ideals, and must live by them. If he does not, he will fail. And a by-result of the discovery that improvement is possible under the best conditions is that with the downfall of the fatalistic tradition in respect to prognosis physicians may be stimulated to renewed efforts to make the most of the methods of treatment which are available to them.

An awakening of this sort is already going on, and a sign of it is the multiplication and improvement of sanitoriums and hydrotherapeutic establishments for nervous invalids. I heartily sympathize with Krafft-Ebbing in his call for more and better institutions of this sort, not only for the rich, but also for the poor, and desire to point out that even where it is impossible to have thorougly equipped hospitals much good may be done by well-managed hydrotherapeutic establishments where ambulatory patients may receive treatment.

Whatever special treatment be adopted, the observing physician, if used to psychological analysis, will admit that the factor of *mental influence* makes itself felt through them all, and that, too, no matter how much both doctor and patient seek to eliminate it.

Neither the consciousness of the physician nor that of the patient controls or grasps the whole of his mental operations, and an interchange of intelligence is continually going on between the two of which neither may be aware. It is important for the physician to realize this, and, so far as he can, to increase the value of his advice by adopting the attitude of confidence, courage, and determination; and the patient is also wise who throws himself absolutely upon this physician, without stopping to examine and criticise the treatment.

It is the patient, not the disease, that is to be cured, and it is for the

physician to cure him by his personality as well as by his prescriptions. It is often the misfortune of the neurasthenic that he has "tried electricity" and this or that other treatment, and thinks he has probed their usefulness and found it lacking. It is well for him if he can cultivate a "good forgetfulness" in this respect whenever he goes to a new physician. The draught which the latter offers, though it looks familiar, may turn out to be an enchanted elixir. The Old Testament story of Naaman, who went to Elisha to be cured of his leprosy, is full of suggestiveness in this connection, and the physician is quite right who quietly insists that his patient must bathe in the particular Jordan of his selection.

Is the neurasthenic condition, as such, susceptible to anything which could be called *specific treatment?* In so far as special causes exist which are capable of removal, this question may be answered in the affirmative. Lead, syphilis, alcohol, and a host of other poisons, both exogenous and endogenous, may act as exciting causes of neurasthenia, and some of them may be expelled or neutralized by appropriate treatment. On the same grounds the casual conditions in each case, whether they be physical or moral, demand close investigation.

In so far as the ultimate changes in the nerve cell are concerned, however, whether those structural peculiarities which prevent it from nourishing itself or those through which the fatigue-making poison is produced, we have no "*specific*" *remedies* at our command. The most obvious nutritive indications are to help out the tissues by an abundant supply of *healthy blood*, to provide means for carrying off waste products, and to reduce destructive expenditure. Sometimes, no doubt, structural deficiencies in the blood and lymph systems make it impossible to meet the two former requirements beyond a certain degree, but it is always desirable to keep the vessel walls elastic and free from degenerative changes, and habituated to respond promptly to normal stimulation, by appropriate contractions and dilatations. The best training for this end is a systematic hydrotherapy, which may need to be persisted in for indefinite periods, but it must be done on a basis of rest and good hygienic conditions.

Much was hoped for from "spermin," a principle contained in various tissues or fluids of the body, especially the secretions of the testicle and the thyroid, which is believed by its discoverer (Poehl), on the basis of original investigation, to increase the oxidizing power of the tissues. Löwenfeld[1] and also Dercum[2] claim good results from the clinical use of the remedy. This should encourage further investigation, but as yet the observations have not been corroborated by any considerable number of physicians. Quite recently, indeed, even the significance of the experimental evidence adduced by Poehl has been seriously called in question by Spitzer.[3]

The use of *nuclein* is also claimed to be of service. It would be premature to express an opinion on this point at the present moment, but the few observations that I have made do not substantiate the view. It is, however, obvious that only carefully selected cases of neurasthenia

[1] *Neurasthenie u. Hysterie.*
[2] *Nervous Diseases by American Authors*, Philadelphia, 1896; art. "Neurasthenia."
[3] *Berliner klin. Wochenschrift*, 1896, p. 360.

ought to be used for this test—those, namely, where the effects of the primary nutritive failure are not too much overlaid with symptoms due to secondary morbid mental action.

The use of *emulsions of nerve substance* as special nerve foods, and of *testiculine*, unless in the form of spermin, is now abandoned by almost every one. We should not, however, forget the lessons which the episode of the "organic-extract" movement has taught us, one of which is that more cases are curable or relievable—though it may be only by some form of suggestive treatment—than would have been readily believed.

It is indeed premature to say even now that these preparations exert absolutely no therapeutic action in a chemical way, in view of the favorable opinion expressed by Löwenfeld and Althaus and a few others. My own observations, however, have led me to the opposite opinion. The large use of *fatty foods* which Beard so warmly recommended as half specific is not now generally looked upon as more than a valuable means for restoring the normal balance of nutrition in special cases. *Special diets* are occasionally called for to a limited extent in special diatheses (see under Etiology, p. 558), and of course to meet gastro-intestinal indications, but in general a varied diet, with perhaps a relative substitution of vegetable for animal albumin (except milk), as less likely to give rise to toxic waste products (Krafft-Ebing), is the best.

Are there any *special tonics* for unduly fatiguable nerve cells, either such as supply oxygen or such as increase the reparative power of the tissue? None of the former kind, so far as we know, but possibly some of the latter. Pure oxygen cannot be forced in through the lungs to beyond the normal amount; potassium permanganate oxidizes the contents of the stomach, but is itself decomposed there and does not supply oxygen to the blood.

On the other hand, we cannot say that some of the agents which stimulate certain parts of the central nervous system, like strychnine, for example, or which increase tissue metamorphosis, like arsenic, may not directly hasten the repair. Patients often improve under their use, though this is owing to indirect causes in some measure.

It is important to remember that the treatment suitable for neurasthenia may vary with the cause, even where the latter is not of a chemical or social nature. A few of the less obvious indications may be noted here: *Trauma* is of importance as among the acquired causes, in that the neurasthenic symptoms are especially apt to take on a hypochondriacal or hysterical cast, and are best treated, after the first signs of shock are over, by vigorous moral influence and in general rather by moderate exercise and activity than by rest, whereas rest is generally necessary for a long time in the neurasthenia which follows fatigues. In case of neurasthenia associated with *pelvic disease* in women, of the *sexual organs* in men, and with *chronic digestive disorders*, in all of which the hypochondriacal element is also apt to be strongly marked, special and local treatments are often needed, to be sure, but usually they have to be subordinated to the general treatment, and sometimes they must be entirely ignored.

Hygienic, Social, and Psychical Treatment.—The most obvious indication in states of chronic fatigue is that the patient should proportion

his work to his strength, and if he can get on, even with occasional attacks of exhaustion, by dint of extreme moderation in work, in the use of stimulants, and in his sexual life, he is fortunate, and should be taught not to account himself an invalid.

Generally, however, the effects of simple weakness and fatigue are overlaid and accentuated by the subtle effects of psychical deception and auto-suggestion. The patient lives in a world of pains and fears which is, to some extent, of his own making. And as the keynote of the psychical symptoms is discouragement and dread, so the keynote of the treatment should be the substitution of sentiments of encouragement, hopefulness, confidence, and determination.

The physician must use every means by word and act to induce the patient to catch from him this sort of sentiment, and may in suitable cases use one or another of the suggestion treatments, even hypnosis if possible, to increase the carrying power of his influence.

Above all things, it is important that the patient should not get into the habit of letting his power of active work degenerate from lack of use. He needs more than others to study the education of the will and the cultivation of his active interests. He should learn to realize that if he is to be an invalid, he should be a cheerful one, and that, in fact, he will be much less of an invalid if he systematically studies how to strip off the superadded tangle of morbid thoughts which annoy him. The encouragement and the good habits which come from a series of resolute acts, inspired by better sentiments, together with the influence of a confident and sympathetic physician, are of service in this task.

Pure explanation and exhortation are useful as an entering wedge, but are not, I think, as effective as might be supposed in helping the patient to strengthen his resolution. There is danger that each time the perplexity occurs the elaborate and fatiguing process of reasoning which the explanation has suggested will have to be gone over again, and that the sentiments of hopefulness and confidence will evaporate in the process. It is the patient's acts that really count, and the physician's confidence in the result.

As regards *hypnotic suggestion*, neurasthenic patients are proverbially hard to affect strongly in this way—though the experience of the best observers shows that it by no means is impossible [1]—but it is not difficult to get the patient into a passive, uncritical state which is favorable to the reception of effective feelings of encouragement, especially if he is kept lying down for a long time, half an hour or an hour, during the treatment.

Modes of treatment which are soothing and refreshing, as static electricity in the form of the prolonged spray is to many persons, and various hydriatic applications to others, also act partly in this way.

I have repeatedly seen morbid habits of pain and disability grow less or pass away under such measures. They may be combined with suitable explanations and encouragements, but verbal suggestions are often unnecessary, and even best avoided for fear of arousing "counter-suggestions" of an unfavorable sort.

It is a fortunate circumstance for the neurasthenic patient if his

[1] See Schrenck-Notzing: *Ein Beiträg zur Psych. u. Sugg. in der Beiträg Behandl. d. Neuras.*, Berlin, 1894.

family and friends take the proper view of his condition, and treat him not as an invalid, much less as one whose disease is a disgrace, but rather as a person with certain limitations as regards exertion, and yet to be called upon to do his work in the world, even if it involves from time to time considerable fatigue. An important chapter could be written on the effects of the moral tone of society, of the social circle, or of the family on the nervous health of its members.

The practical question often presents itself whether neurasthenics do better in the city or the country, or whether the difference in favor of the latter is so great that it is worth while to make considerable sacrifice to secure it. The problem is largely an individual one, for city life may be very exciting or fairly quiet according to circumstances, and, on the other hand, the monotony and loneliness of a prolonged isolation, away from familiar scenes and excitements, may have a distinctly bad effect on those who are unaccustomed to it.

My own belief is that the essential thing is to secure such a life as will give a reasonable amount of real absorbing interest without too great a tax on the strength. It is undoubtedly extremely important to be rid of many of the excitements of the city and of the rain of jarring sounds falling constantly on the ear, as well as to breathe an air free from impurities. But these are, after all, of secondary importance, and although it is true that their effects in inducing fatigue are often greater than is realized at the moment, yet, on the other hand, familiarity with them unquestionaly robs them of a great part of their power for harm.

As regards *occupation*, the first requisite is that the patient should feel that he is doing something for which his interest and talents fit him, and which he regards as important; but oftentimes he must content himself with a subordinate position which does not involve much responsibility.

Exercise and Rest, and Bed Treatment.—It would be impossible to do justice to this important branch of the subject without discriminating as among the various classes of cases to a greater extent than our space permits. Acute attacks or exacerbations demand absolute rest, while, on the other hand, the capacity to take physical exercise freely is a great boon for many patients, and the systematic pursuit of suitable gymnastics affords a training of high value. I fully agree with Löwenfeld in his statement that neurasthenic backache is often better treated by engrossing and pleasurable exercise than by rest.

A practical danger is that feeble patients may be allowed and tempted to exhaust themselves in conscientious efforts to improve the health by exercise, and a good practical recommendation for all who can follow it is that the day's work should be broken by an hour's rest at noon before the midday meal. I could cite a large number of appreciative recognitions for this advice which I have given for many years, almost as a matter of routine, enjoining, at the same time, as an essential factor, that the rest should be followed by a cool or cold bath, generally taken in the form of an affusion while the patient stands or sits in warm water.

Where it is impossible to give up the noontime to resting, the night's rest may begin early and end late, or a rest followed by a bath may be taken before the evening meal.

Where serious failure of the nutrition and disgestion is present, or where an acute prostration of the nervous strength occurs, a complete "*rest-cure*" may be indicated. It is unnecessary to dilate at length, to a community familiar with the masterly expositions of Weir Mitchell, upon the value of this treatment or the principles under which it is to be carried out. The *Practical Dietetics* by W. Gilman Thompson contains the details of several modifications of Mitchell's method, which the reader is warmly recommended to consult. The essential points to be borne in mind are that the injurious effects of the rest should be counteracted by massage and passive or active exercise in bed, and its good effects reinforced by such influences as would be likely to secure a favorable mental attitude on the patient's part. The day should be well filled with healthful occupations, so to speak, carried out with extreme regularity and confidence by a good nurse, so that the patient may feel himself swept along in the direction of health. Skim milk, gruel, buttermilk, koumyss, and eventually other sorts of food, may be given at short intervals. With these, and with massage, baths, electrical applications, breathing exercises, and simple movements for the hands and feet, it is not difficult to fill out the day, and as the patient gains in strength more privileges may be allowed, one by one, which stimulate and encourage and give new confidence.

The following prescriptions of daily routine as regards food are copied from Thompson's book.[1] The physician may make modifications if he chooses. but should not forget that the secret of success will often lie in the adoption of some sharply-defined plan, to which he must confidently adhere.

Weir Mitchell, who was the pioneer of this method of treatment, prescribes an exclusive milk diet less rigidly than formerly. After five or six days of such treatment a chop or a poached egg may be added at noon. The next day bread and butter or bread and milk are given besides for supper, and then an egg or a little meat at breakfast, until the patient is taking three good meals of plain food daily, but in addition at least two quarts of milk. The exclusive milk diet is believed to prepare the digestive system for the assimilation of other foods.

Playfair's Diet.

"Playfair's diet for neurasthenia is a good example of a milk diet, soon combined with other foods, as follows:

"First Day.—Twenty-two ounces of milk in divided doses.

"Second Day.—Fifty ounces of milk in divided doses.

"Third Day.—Fifty ounces of milk in divided doses. Massage, half an hour.

"Fourth Day.—Fifty ounces of milk in divided doses; egg, bread and butter; dialyzed iron, forty minims in two doses. Massage, one hour and a half.

"Sixth Day.—Fifty ounces of milk in divided doses; mutton chop. Massage, one hour and fifty minutes.

"Eighth Day.—Fifty ounces of milk in divided doses; mutton chop; porridge and a gill of cream; maltine twice daily. Massage, three

[1] *Practical Dietetics*, New York, 1898.

hours; electricity, half an hour, continued to the end of the treatment. The solid food is now gradually increased until such a diet is reached as the following for the

"Tenth Day.—6 A. M., raw meat soup, ten ounces; 7 A. M., a cup of black coffee; 8 A. M., a plate of oatmeal porridge, a gill of cream, a boiled egg, three slices of bread and butter, and cocoa; 11 A. M., milk, ten ounces; 2 P. M., rump steak, one half pound of potatoes, cauliflower, a savory omelet, milk, ten ounces; 4 P. M., milk, ten ounces, three slices of bread and butter; 6 P. M., a cup of gravy soup; 8 P. M., a fried sole, roast mutton (three large slices), French beans, potatoes, stewed fruit, and cream-milk, ten ounces; 11 P. M., raw meat soup, ten ounces.

"Fifteenth Day.—Three full meals daily of fish, meat, vegetables, cream, and fruit; two quarts of milk and two glasses of burgundy.

"Twenty-second Day.—Amount of food lessened."

Climate.—The effects of climate have been much studied, but in a given case the considerations here involved are apt to be overbalanced by those of social and medical significance. The consideration of chief importance is that exposure to rapid changes and to hot winds should be avoided so far as practicable.

In general, frequent changes of scene and air are useful, but it is often a mistake to advise long journeys if they interfere with carrying on a regular pursuit at home. Opportunities in the way of holidays and half-holidays should be sought and taken advantage of to the fullest extent.

Hydrotherapeutics.—The systematic external use of water is of the very highest importance in the treatment of neurasthenic conditions, and the conveniences of a well-arranged establishment, where douches can be had of varying force and temperature, are to be secured when possible. It would be out of the question to give here a satisfactory treatise on hydrotherapeutics, and I shall therefore attempt only to indicate the most important principles at stake.

The results which we gain are mainly through excitation of the nerves of the skin, actual withdrawal of heat counting for very little. If this excitation is mild and applied over a large surface at once, as when a warm bath is taken at the temperature of the body, ideas of restfulness are suggested and feelings of irritability relieved. If, on the other hand, cold is used, a powerful reflex stimulation is exerted on the bloodvessels of the skin, the muscles, and the deeper lying organs, including the brain, in consequence of which widespread vascular contractions, followed by dilatations, take place, and at the same time the arteries and nerves are trained, as it were, to respond with greater promptness and efficiency another time, the whole process forming what has been fittingly called by Baruch gymnastics of the bloodvessels. Besides the vascular effects of a cold bath, the whole muscular system is stimulated and heat production is increased and the respiration is deepened. In order that these satisfactory results should occur, it is necessary, in the first place, that a fairly good capacity for normal vasomotor response should be present, or, in popular language, that the patient should have a good power of reaction. If, as in many cases of neurasthenia, this power is defective, the reaction will either

fail to occur or, occurring, will not persist, so that after an hour or two a sense of chilliness will come on which may lead to a catarrhal inflammation of some of the mucous membrane.

The essential means for securing a good reaction are—first, that the skin should be warm at the time the bath is taken; next, that the degree of cold and the force with which the cold application is made should be proportioned to the patient's power of response; third, that by friction during and after the bath the reactionary glow should be excited as rapidly as possible, and that it should be maintained by suitable exercises or massage. In practice, the first of these conditions is secured by steam or hot-air boxes, by warm baths of short duration, or by wrapping the patient in blankets with or without a wet sheet next to the skin. With vigorous persons the preliminary warmth may be obtained by exercise, but here the danger comes in that the patient will have his reaction interfered with by fatigue. As regards the second indication, it is necessary that patients who are not accustomed to the use of cold water should be gradually habituated to it. For this purpose the body may first be wrapped in blankets, and one part after another uncovered, bathed, dried, and re-covered. The patient may next sit or stand, after a blanket-pack in warm water, while cooler water is dashed liberally over the head and shoulders, the temperature of the cooler water being reduced one or two degrees each day. By this means even very delicate persons may be enabled to stand cold douches or sprays at a low temperature and delivered under high pressure. The high pressure soon becomes of great advantage, since the mechanical impact of the water helps to secure the reaction, but not every patient can stand a powerful stream at first. Where no means but the simplest are at hand the cold affusions may be made more and more stimulating by reducing the temperature of the water, even icing it if desirable, and by prolonging the application. Further means for securing a reaction are furnished by the wet pack, the patient being first enveloped in a cold wet sheet, then rapidly in blankets which are drawn tight and snugly tucked in. The pack is generally removed at the end of half an hour or an hour, and the patient rapidly bathed in cool or cold water or given a cold bath or an application of the "dripping sheet."[1] For this purpose, while the patient (previously well warmed) stands or sits upon a stool, an assistant envelops him from behind with a coarse linen sheet previously immersed in cool or cold water. The assistant supplements the stimulant action of the cold water by rapidly stroking the back and limbs with his hand outside the sheet, while the patient rubs himself in front. After a moment or two the wet sheet is removed and a dry sheet substituted. It is said that the good effects of massage are obtained more fully and rapidly if preceded by some such application as this.

By *properly graduated* treatment of this sort the tonicity of the arterial system is undoubtedly raised, the metabolism increased,[2] the power of digestion improved, the thoughts turned into new channels, and a sense of power and well-being imparted. It has been claimed, and may be conceived of as true, that even the nutrition of the vascular

[1] See Simon Baruch: *The Therapeutic Uses of Water*, and other publications.
[2] See Mary Putnam Jacobi: *The Cold Pack and Massage in the Treatment of Anæmia.*

walls may be maintained or improved in this way—an effect of great importance for the nutrition of the nervous system.

Electricity.—There is no doubt that electricity in its various forms can be made of material service as an adjuvant in the treatment of neurasthenia. It is not probable that it exercises a direct influence over the altered nerve cells, but it affords a convenient means for carrying out a systematic course of treatment—a mode of action which might, in general terms, be call "suggestive," though the influence which this name covers is by no means a simple one. There is some reason for thinking that the general nutrition and metamorphosis of tissue is favorably affected, but this is not beyond question.

The methods of using electricity which give the best results are—by the static form, both the breeze and the sparks being available; by galvanization of the head and back; by general faradization of the skin and muscles; and by electric baths. Levillain[1] believes, with Vigouroux, that static electricity comes near to being a specific for neurasthenia. From the clinical standpoint I am almost ready to share this opinion, though I believe we are not in a position to deny that the good results are largely of a psychical origin, the prolonged and soothing application tending to throw the patient into a quiet and hopeful mental state.

Many observers think very highly of galvanization of the head and back and of faradization of the muscles of the skin. The latest and one of the best writers on this subject is Remak, and on the whole he supports this view. At the same time, this writer points out the great discrepancies which prevail as regards the most suitable strength of current, at least where galvanism is concerned; and one who is familiar with the peculiarities of neurasthenic patients must admit that the apportionment of results as between actual chemical or nervous action and "suggestion" is difficult to make.

In the treatment of certain local neuroses the wire brush and the sparks from the static apparatus are of marked service. Goldschieder in his recent review of methods for the treatment of pain in its different forms discusses the rationale of these measures, and thinks that the inducing of a new set of sensations is an important factor. I have long held this view, and believe that the new sensations are of greater value in proportion to their unfamiliarity. I think it is for this reason that vibrations, especially those which are communicated by a vibrator powerful enough to act deeply, are of so much value. It must, however, be remembered that when we say new sensations or unfamiliar sensations we use that term, not in an exclusive sense, but as standing for a whole series of modifications in the distribution of central nerve energy, whether conscious or unconscious.

Löwenfeld praises electricity in the treatment of nervous disorders of the heart, using large electrodes and an ascending galvanic current from the cardiac region to the neck (vagus), with gradual increase and diminution of the current.

All the writers on sexual neuroses of every sort advise the different forms of electricity, applied locally and generally in strong currents. There is no positive agreement as to the form.

[1] *La Neurasthenie*, Paris, 1891.

Treatment by Drugs.—This is, perhaps, the least important part of the treatment of neurasthenia, and yet there are, of course, many special indications which are well met in this way.

I can only speak of the more important of these. *Strychnine*, in increasing and eventually large doses, has seemed to me of great value. I have used it in the form of nux vomica, carried from ten to twenty, and even to thirty, drops three times a day. Occasionally it causes diarrhœa or unpleasant signs of stimulation, but in the vast majority of cases patients feel better under its use. *Iron* is useful in large doses, when anæmia exists, but it is a striking fact that in many cases of neurasthenia blood tests fail to indicate marked anæmia even when the patient looks pale. *Arsenic* is said to improve the vasomotor tone, but there is little real ground for this belief, and where it is of service, as it undoubtedly sometimes is, the result is probably due to its action on metabolism. A recent observer, commenting on the signs of slight arterial sclerosis, such as are sometimes seen in neurasthenia, asserts that he has found the remedies that lessen blood tension to be of marked service. I have no experience bearing on this point, except that bromide salts are certainly valuable, and that the drinking of a large quantity of water is also useful. Other grounds can, however, be assumed for these results. *Stimulants* and *narcotics* are, in general, to be avoided, and the habitual use of hypnotics commonly entails more distress than the drugs relieve. They do, however, undoubtedly bridge over sometimes a very important crisis, and are in that way very useful. The *bromides* especially, given from time to time for a very few days, induce quiet and sleep, and sometimes initiate a new and better phase of the disease.

Krafft-Ebing recommends *piscidia* as a good substitute for the bromides.

Treatment of Special Conditions and Symptoms.—The *insomnia* of neurasthenics, as it often leads to the development of the disease, so also often forms one of its most distressing symptoms, though, on the other hand, the blooming appearance which those who complain of it often present may justify the conclusion that here too fixed ideas play an important part, and that the patient really sleeps better than he has believed.

When true insomnia exists the question at once arises, Shall it be treated with hypnotics? If this would entail the necessity of a continual resort to such remedies, they are best avoided altogether. If this be resolutely done, the patient will be the more ready to adopt adequate measures for correcting the underlying state, and may find that even if he loses a few nights' rest now and then, he will get more refreshment from the natural sleep which he does secure than that which hypnotics furnish.

The inability to sleep is generally induced either by over-fatigue (leading to the "fatigue-anæsthesia" described by Cowles), or to worrying thoughts, or to morbid habits, based in part on disordered expectancy, or to distressing sensations of sexual or other origin. The former tendency is best counteracted by a rest in the daytime, as described above, and by passing the evening in quiet indolence or with appropriate reading. The sort of book which is the best is generally one which deals with details of an interesting but not over-stimulating sort. Treatises

on natural science or unstimulating stories fill the need for many persons. Baths in the day and baths at bedtime, and even in the night, such as warm baths followed by cold affusions, or the cold dripping sheet, are extremely valuable hypnotics, partly because they pave the way for a clearer and healthier action of the brain, partly because they increase the tonic condition of the bloodvessels and help digestion. A bowl of hot Indian-meal or other simple gruel (sometimes best without milk) is an excellent night-cap. Bromide salts in full doses, taken several times a day for several days in succession, will often break up an attack of insomnia and thus help, also, the patient's mental state. The stronger hypnotics may of course be required for emergencies, but the detailed consideration of their respective merits may be omitted. The decision whether or not to use them must often be determined by observing whether the insomnia is a temporary phase of the malady or has become a chronic and somewhat independent affection, in which case it might often be called a neurosis of habit or association. Narcotics are much less admissible in the latter case than in the former. In either case the use of electrical treatment—not especially at bedtime—is often of great service. Prolonged static applications are perhaps the best, but, whichever form is used, a better result may be expected if the immediate effect is soothing, and it is, in fact, due to the induction of a subconscious expectation of a favorable sort.

Disorders of Digestion.—It is well recognized that neurasthenic patients may suffer distress during digestion even when careful tests show that the food is well disposed of and the digestive idiosyncrasies of purely mental origin abound. In view of these facts it is always important, and sometimes all-sufficient, to place the patient under favorable conditions as regards avoidance of fatigue and mental worry. The Mitchell rest treatment shows its value in no class of cases more than here, and its principles may be followed, in a modified form, for cases not requiring the full treatment. It is important in case of doubt, or sometimes for relieving the patient's mind, to use the test meal and stomach-tube to eliminate real catarrhal gastritis, and in that connection it should not be forgotten that the occasional use of the stomach-tube, at least in the form of a douche, is often of service as a therapeutic measure in these cases. Where catarrh is absent the motility or power of normal reaction of the stomach may be at fault and may call for special treatment, as by external or internal electrization, massage, the use of pepsin, alkalies, or taka diastase, etc., in accordance with principles laid down elsewhere. But in the great majority of cases far more will be gained by an insistence on proper and thorough hygienic, tonic, and mental treatment, though serious cases tax the ingenuity and determination of the physician to the utmost.

Intestinal indigestion is equally hard to cure, but the principles of treatment are the same—first general, next local, and both thoroughly applied. The danger is that the physician will fail to recognize how large a part is played by morbid nervous habit and morbid hypochondriacal or subconscious ideas. The presence of starchy indigestion must be met by cathartics, diet, and special digestants, such as the taka diastase.

Cardiac and vasomotor disorders are important, but obstinate, affec-

tions. Where continuous cardiac irritability is present, complete rest for a time, with bromides and iodides or arsenic, and galvanization of the cardiac region (Löwenfeld), are indicated at first, but suitable Swedish or other exercises, if carefully chosen, are important later. So also the tonicity of the vasomotor system can perhaps often be improved by persistent hydrotherapy. But the pulsatile forms of vasomotor disorders are intractable, and so also are the paroxysmal forms which are often associated with sweating. A vigorous general treatment is the best.

Asthenopia may call for careful fitting of glasses and even muscle section, but is often very amenable, within certain limits, to careful training, as by Dyer's method, according to which the patient reads with extreme regularity—at first one or two minutes three times daily, then every day one minute more up to ten minutes, after which more rapid increase is made until several hours' reading is done daily. Static electricity is of material service in such cases, probably by imparting confidence and encouragement.

Headache, in so far as special treatment is needed and is not pointed out by other indications, is sometimes relieved by electricity and by massage. In bad forms it is extremely obstinate.

For the details of the treatment of *sexual neurasthenia* the reader is referred to the special treatises. The following suggestions are, however, of prime importance :

The patient should be instructed constantly in sound views, and where self-restraint is needed its difficulties and possibilities should be clearly pointed out, with a view to the stimulation of the will and the counteraction of morbid self-reproach. It should be recognized that sexual intercourse is not the main object of marriage.

On the other hand, an unnatural struggle for extreme abstinence is not good for the neurasthenic patient, and the physician can often bring material aid to the patient in arriving at a wise conclusion as to detail.

Excessive *seminal emissions*, which so often unduly terrify adolescent and even adult patients, can often be checked, so far as that is necessary, by a thoroughly healthy mode of living ; the use of atropine or hyoscyamine and gelsemium through the day and at bedtime ; hot sitz baths ; and by measures addressed to overcoming hyperæmia of the prostate and distention of the seminal vesicles. This can be done by cold urethral sounds or double-current catheters, and often with especial success by the use, for half an hour or more daily, of the "psychrophor,"[1] a double-current rectal tube, with ice water.

Digital compression of the seminal vesicles, and applications of silver nitrate in the prostatic urethra, and also electricity, are of excellent service in the hands of experts.

[1] Made by Tiemann of New York.

OCCUPATION NEUROSES; AFFECTIONS OF THE NEURO-MUSCULAR APPARATUS DUE TO SPECIAL OCCUPATIONS.

By CHARLES. K. MILLS, M. D.

Synonyms and Definition.—A large number of affections of the neuro-muscular apparatus result from the monotonous and prolonged use of certain groups of muscles in the pursuit of special occupations. These are described by systematic writers under various names, such as *occupation neuroses*, *fatigue neuroses*, and *occupation spasms*. Other terms have been suggested to embrace these diseases, but none have become firmly rooted in professional literature. Morris Lewis[1] invented the term *copodyscinesia*, derived from certain Greek words the interpretation of which is "faulty or difficult motion due to constant repetition of the same act." *Anapeiratic paralysis*, *professional impotence*, *professional hyperkinesia*, and *professional dyskinesia* are other more or less general terms which have been suggested for these affections. No single descriptive definition can fully cover the different disorders which belong under the head of these occupation neuroses; but they may be defined in a general way as affections of the neuro-muscular apparatus, usually presenting themselves as forms of local spasm, tremor, or paralysis, or some combination of these symptoms, often with the addition of pain and other sensory symptoms. They arise from the excessive and long-continued use of groups of muscles which are, as a rule, concerned in the highly specialized movements necessitated by particular occupations.

An objection to the use of the general term "occupation neuroses," or "affections of the nervous system due to occupation," is found in the fact that other entirely distinct forms of nervous disease are sometimes the direct result of particular occupations. Diseases of the nervous system may, for example, originate from the effects of certain toxic agents, such as lead, mercury, aniline dyes, nitrobenzine, and carbon monoxide, with which workers come in almost constant contact in the pursuit of certain occupations. Exposure to the injurious effects of tobacco, electricity, heat, compressed air, or any other agency capable of inflicting injury upon the nervous system may likewise give rise to nervous diseases.[2] Our present concern is simply with those affections which are generally classed by English and American writers as the

[1] Lewis, Morris J.: "The Neural Disorders of Writers and Artisans," *A System of Practical Medicine by American Authors*, 1886, vol. v. p. 504.

[2] One of the most valuable contributions to the subject of "the diseases of occupation" is the exhaustive article on this subject in the *Twentieth Century Practice of Medicine*, by James Hendrie Lloyd of Philadelphia.

"fatigue neuroses." The term fatigue neuroses was first suggested by George Vivian Poore.[1]

Occupations most Frequently causing Fatigue Neuroses.—The list of occupations the pursuit of which has caused neuro-muscular affections that can properly be classed under the general head of fatigue neuroses is now very large, and is being constantly added to by the reports of single cases or groups of cases in the medical journals. This is, of course, what would be expected, as any occupation involving prolonged and monotonous exertion may sooner or later give rise to such diseases, especially if this exertion is of such a character as to involve innumerable repetitions of the same acts.

The following is a list of the classes of persons following occupations which have caused fatigue disorders, either in recorded cases or in the experience of the writer: Scriveners, telegraphers, stenographers, typewriters, pianists, violinists, drummers, cornet-players, seamstresses, operators on sewing machines, artists, dancers, turners, shoemakers, shoefitters, tailors, blacksmiths, carpenters, watchmakers, goldbeaters or other users of hammers, weavers, milkers, money-counters, compositors or typesetters, grinders, cutlers, stonecutters, sawyers, masons, brickmakers, bricklayers, fruit-packers, cigarette-makers, letter-sorters, dairymaids, knitters, makers of artificial flowers, actors, professional voice-users, auctioneers, car-drivers, motormen, and preparers of photographic plates.

General Remarks on the Nature of the Fatigue Neuroses.—It may serve a good purpose to briefly discuss the relations of fatigue to the production of any or all of the fatigue neuroses. What is the explanation of their development? How do they arise? How do they progress? In general terms, they are the result of interference with that rhythmic nutrition which Sir James Paget[2] has declared to be one of the great laws of nature—a law the violation of which entails pain, disease, and, it may be, death. It is not work, physical or mental, which leads to these dire results, but overwork in the strictest sense of the word. Work within normal limits leads to normal fatigue, which in turn calls for rest in order that recuperation may occur; overwork leads to abnormal fatigue, and at its worst destroys even the inclination to that repose which helps to carry out this great law of rhythmic nutrition.

The diseases which are at first functional, peripheral, and neuro-muscular, and simply the temporary effects of interference with this law of rhythmic nutrition, in time become affections of the spinal and still later of the cerebral centres.

"There is a certain limit," says Lewis, "to which exercise of a given group of muscles may be carried without producing fatigue and local congestion, or perhaps even inflammatory results: this varies greatly in different individuals, but if it is continually and uninterruptedly overstepped, and insufficient time is given for rest and recuperation, the cen-

[1] Poore, George Vivian: *A Text-book of Electricity in Medicine and Surgery*, London, 1876; "An Analysis of Seventy-five Cases of 'Writer's Cramp' and Impaired Writing Power," *Medico-Chirurgical Transactions* London, 1878, 2d series, vol. xliii. p. 111.

[2] Paget, Sir James: The Croonian Lecture "On the Cause of the Rhythmic Motion of the Heart," *Proceedings of the Royal Society of London*, 1857, vol. viii. p. 473.

tres in the spinal cord which regulate the action of the various muscles implicated become over-stimulated, and the result is an undue amount of nervous energy induced by the peripheral excitation, or there is a distortion of the central impulses in passing through these centres; a perturbation of the co-ordinating power ensues, and inco-ordination is the result. Under rest and appropriate treatment these symptoms may pass away, but if the part is continually used it is highly probable that nutritive changes will be produced in that part of the spinal cord from which the nerves supplying the overtaxed muscles proceed." The weight of recent opinion is undoubtedly in favor of the view that the seat of the greatest pathological change in these cases is in the central nervous system; doubtless in some of them it is in the gray matter of the brain cortex.

The researches of Hodge[1] on the condition of the nerve cells of animals both before and after the induction of excessive fatigue furnish the closest approach to a scientific explanation of the pathological substratum of the fatigue neuroses. The spinal ganglia and brain cells of various animals, as the honey-bee, sparrow, swallow, dog, cat, and frog, were studied microscopically both before and after fatigue. The spinal ganglion of a cat which had been submitted to prolonged electrical excitation was compared with the corresponding ganglion of the opposite side which had not been subjected to this exhaustive procedure. Both the ganglia and the brain cells of bees and birds were studied before and after long flights. The examinations were made with scientific precautions. In all the cases studied changes were found in the nerve cells examined. Among the most important of these changes were marked decrease in the size of the nucleus, changes in its outline, and loss of its usual open reticulated appearance. The protoplasm of the cells became shrunken, with marked vacuolations. The cells of the cerebrum and cerebellum shrank considerably, the pericellular lymph spaces enlarging. Experiments such as these afford a reasonable explanation of some of the changes in spinal and cerebral centres which are brought about as the result of prolonged and excessive work in special occupations.

One of the effects of local as well as of general fatigue is the production of a toxic substance which, circulating in the blood, affects the nerve and nerve centres, and thus indirectly causes the series of phenomena presented in the fatigue neuroses. Mosso[2] found that the blood of a fatigued animal when injected into an animal at rest produced in it the characteristic symptoms of fatigue. Muscles, nerves, and nerve centres—the entire neuro-muscular apparatus—probably suffer not only from loss of substance and change in structure, but also from the effects of the toxic agents circulating in the blood. These "fatigue substances," as they are sometimes called, may therefore also play an important rôle in the production of the symptoms which are present in the occupation neuroses now under consideration.

Symptoms: General Symptomatology.—Owing to the diversity of occupations which give rise to fatigue neuroses it is almost impossible

[1] Hodge, C. F.: "A Microscopical Study of Changes due to Functional Activity in Nerve Cells," *Journal of Morphology*, 1892, vol. vii. p. 95.

[2] Mosso, A.: "Les lois de la fatigue studiées dans les muscles de l'homme," *Travaux de Lab. de Physiol. de l'Université de Turin*, 1889.

to give a general symptomatology for the neural and neuro-muscular affections under consideration, but certain symptoms are more or less common to almost all forms of copodyscinesia. Generally, the most prominent is probably given to certain motor disturbances, and especially to local spasm, tremor, and paralysis or paresis. Inco-ordination is undoubtedly present in many cases, and perhaps too little stress is laid on this symptom by most writers on this subject. In the discussion of the symptomatology of the fatigue disorders the term *cramp* is usually spoken of as synonymous with local spasm. The cramp, however, of which the patients so frequently complain is a sensory as well as a motor disorder, a combination of pain or paræsthesia with spasm or contracture. Local spasm when present usually appears first in the muscles or muscular groups which are most directly concerned with the necessary movements in the occupations which are followed, and is generally most marked in them. It does not, however, continue to be restricted to these muscles. If in the fatigue diseases of writers, telegraphers, or stenographers the spasm first and chiefly affects the muscles of the fingers, hands, and forearm, it may soon extend from these to those of the arm and shoulder, and even, in an irregular manner, to the musculature of the entire half of the body on the affected side. In some cases associated spasmodic movements of the muscles of the other side occur. The extensors may be affected in one case, in another the flexors, while in still others both the extensors and the flexors may be involved. In most of the affections the upper extremities, and especially the right upper extremity, will be the seat of the disorder, but the lower extremity may be involved in occupations in which the work is chiefly done by the foot and leg, like turning, walking, and operating sewing machines. Loss of power occurs in a considerable percentage of cases; this is usually partial, the patient being paretic. The parts affected are easily exhausted by a comparatively small amount of exertion. At first only a sense of weakness may be complained of after the limb or part has been used for a more or less considerable period. As the disorder progresses the sense of weakness may be constant, and the patient may be unable to use the affected parts for more than a few minutes or in some cases not at all. Diminution of power is commonly associated with the tendency to spasm or cramp, but special cases may be purely paretic, and in rare instances may be altogether of a spasmodic type.

While tremor is not nearly so common a symptom as local paresis and local spasm, in some instances it is a marked symptom, and may even in rare cases be the most important manifestation. Paresis and tremor are a more frequent combination than spasm and tremor, but spasm, tremor, and paresis may be all combined in the same case.

Inco-ordination may be apparent rather than real; that is, the inability of the patient to harmoniously perform the movements necessary in his occupation may be the result of spasm and paresis, or of tremors, spasm, and paresis, rather than of a true ataxia. A genuine ataxia of movement may, however, be present.

Pain may be present, or the patient may simply suffer from a sense of discomfort associated with various paræsthesias, such as numbness, heaviness, tingling, or burning.

A true subacute or chronic neuritis is not rare, the patients in these

cases suffering not only from subjective pain and discomfort, but also from tenderness of the nerve trunks and endings. In some occupations—as in brickmaking, for instance—the neuritis present is due in whole or in part to exposure and to actual traumatisms inflicted upon the nerves. Even a mild form of arthritis is occasionally observed. Vasomotor phenomena, such as redness, pallor, swelling, and œdema, occur, particularly in those cases in which genuine neuritis accompanies the other conditions resulting from over-use, but such vasomotor changes are not always dependent upon neural inflammation.

Hypertrophy on the one hand and atrophy on the other have been noted in different forms of fatigue neuroses, and special trophic changes may be observed, as ridging, brittleness, or discolorations of the nails.

Attention might be directed here to a sensory symptom which Bernhardt has especially studied—a painful condition affecting the region of the external epicondyle of the humerus and occasionally the head of the radius. The patients complain of pain beginning in this location, sometimes extending down the forearm to the wrist, or even to the fingers. The pain is increased by various motions, such as closing the hand, extending it, grasping tools, or even holding a pen, and so interferes with various occupations. At rest it is slight or absent. Of 30 cases carefully studied, 27 were men and 3 women, and with one exception all the patients were above the age of thirty. Various occupations were represented, and in almost all cases over-exertion, especially of the affected arm, was alleged by the patients. In connection with the history of over-exertion Bernhardt[1] calls attention to the numerous important muscles attached to the points especially involved, the excessive use of which might easily cause irritation of the periosteum. In 2 cases the internal epicondyle was sensitive.

The symptoms of general neurasthenia are quite common in patients who suffer from the local phenomena which have just been described. The most important of these symptoms are—general mental and physical feebleness, diminution in the powers of attention, and evidence of irritable weakness, as shown by undue emotionality and excitement.

Occasionally the stigmata of true hysteria are present in those suffering from the fatigue neuroses. One of the important points in diagnosis, as will later be considered, is to distinguish hysterical manifestations from those which are dependent purely and simply upon fatigue; but the fatigue neurosis itself may bring to the surface grave hysterical manifestations, especially in those who are neurotic. Hysteria and a fatigue neurosis may, in other words, be coincident in the same case, or the former may be induced by the latter.

The electrical changes present even in advanced cases are, as a rule, of a quantitative rather than a qualitative character. Diminished readiness of response both to the galvanic and faradic currents and increased irritability of both nerve and muscle are the most frequent electrical phenomena.

Some details of the symptomatology of a few of the separate disorders produced by occupations, as writer's cramp and telegrapher's

[1] Bernhardt, M.: "Ueber eine bekannte Form der beschäftigungs neuralgie," *Neurologisches Centralblatt*, Leipzig, Jan., 1896, No. 1, p. 13.

disease, and a few illustrations of some unusual forms of fatigue neuroses, will now be given.

Writer's Spasm.—The synonyms of the fatigue disorder from which clerks, scriveners, and others whose occupations require almost constant writing suffer, are numerous, but the most important are *writer's paralysis, dysgraphia, graphospasm, mogigraphia, chorea scriptorum,* and *paralysis notariorum.* Although not strictly descriptive of all the cases, the term *writer's cramp* has been chosen as the one best known, and also the one which is explanatory of the most frequent and important manifestations of the disorder. It is an affection showing itself usually by local cramp or spasm, paresis, pain, and paræsthesia, and is most frequently observed among authors, clerks, bookkeepers, and others who live by the use of the pen.

Writer's cramp in different cases may develop in somewhat different fashions. Occasionally the trouble in writing appears suddenly; more frequently it comes on somewhat insidiously. A cramp or spasm, usually slight at first, affects the muscles chiefly employed in writing, and the writer may at first experience only a slight awkwardness.

Poore and others have carefully studied the action of the muscles concerned in writing and the order and degree of their implication. The muscles of the hand employed in writing are first and chiefly affected, and later those of the forearm in greater or less degree. As the case becomes more and more serious the number of the muscles involved increases, and sometimes the entire arm becomes affected, and even other parts may be implicated.

The patient varies the method of holding his pen in order to overcome his difficulty. The affected extremity may be the seat of tremor or of ataxic and choreic movements, and some loss of power can in time usually be determined in the muscles affected. When real paralysis is manifestly present either grave hysteria or some organic cause should be looked for; nevertheless, the limb in serious cases of true writer's cramp may be profoundly disabled. Some pain in the parts affected is experienced in nearly all cases, or, if not pain, some feeling of discomfort in the hand and arm. The patient may complain of intolerable aching. A severe neuralgic pain may be fixed in the thumb and fingers or in the wrist and forearm. The fingers and hands may become flushed, with sensations of throbbing. Muscular atrophy has been reported by some careful observers, but it is certainly rare.

Evidences of a slight or more severe neuritis, such as hyperæsthesia along nerve trunks, glossy skin, nail changes, or alterations in the hair, may be present; but although neuritis may of course be associated, it is doubtful whether this should be regarded as an essential element of a fatigue neurosis.

Writer's cramp is not of so frequent occurrence as formerly, because of the very general substitution of dictation for writing, especially by authors and by professional and business men. If the case is one of true writer's cramp, the disorder is one essentially due to over-use and fatigue. Its causes are both predisposing and exciting, the former playing a part almost as important as the latter. Many persons in good health go on for years writing many hours in the day without the production of any spasmodic, tremulous, or paralytic affection; others of

different temperaments and of less perfect general health will develop the disorder in a very short time. General neurasthenia lays the foundation for local neurasthenia and a permanent impairment of nutrition of the parts affected. The manner of using the pen and the kind of pen have something to do with the production of the fatigue disorder. A good stub pen is assumed to be less likely to cause trouble than the ordinary pen, and the affection is less frequent in those who write with free arm movements.

Telegrapher's Disease.—Next in importance to writer's cramp is a similar disease which afflicts telegraphers. This was first scientifically studied by Onimus[1] in 1875, but many writers since have given it their attention. Our limits will not permit us to go into a study of the telegrapher's work nor to analyze the fine and rapid adjustments necessary in telegraphing. The reader is referred to articles on the subject written by Robinson,[2] Fulton,[3] Lewis, and Lloyd.[4]

The symptoms of this affection are so largely spasmodic that it probably deserves the restrictive name of an occupation spasm. The operator finds that he makes, or tends to make, errors in the number of dots and dashes which correspond to given letters; not infrequently his first difficulty will be with one special series. The cramp or spasm is generally of the extensor variety, causing the fingers to be jerked away from the instrument, or even the whole hand may be spasmodically retracted. Pain in the affected parts may or may not be present; usually but little pain is complained of unless the operator persists in spite of the fatigue and difficulty in his work. Sometimes neuralgic pains develop here and there in the arm, with at times numbness and tingling, but true anæsthesia is not present. The fingers and hands may be the seat of a paresis or pseudo-paresis, but this is usually not marked. Atrophy and electrical changes are not present. If the operator persists in spite of this trouble, the difficulty becomes greater and greater, until mistakes may be so serious as to prevent a continuance of work. Efforts, at first successful, are made to substitute the other hand, but in most cases after a time this also gives way. The operator may resort to various expedients and new methods in order to lessen or overcome his difficulty.

Compositor's Spasm.—A few cases of spasmodic fatigue neuroses have been reported as occurring among typesetters. One case of this kind came to my service at the Philadelphia Polyclinic in 1894, and has been recorded by my chief of clinic, J. W. McConnell.[5] The most important facts from the report of this case will be cited, as it is rather an unusual type of what appears to have been a nervous affection due to occupation.

[1] Onimus, M.: "Le mal télégraphique ou crampe télégraphique," *Comptes rendus des Séances et Mémoires de la Biologie*, Paris, 1880, p. 92.

[2] Robinson, Edmund: "Cases of Telegraphist's Cramp," *Brit. Med. Journ.*, Nov. 4, 1882, vol. ii. p. 880.

[3] Fulton, Thomas Wemyss: "Telegraphist's Cramp," *Edin. Clin. and Path. Journ.*, Feb. 2, 1884, vol. i., No. 17, p. 369.

[4] Lloyd, James Hendrie: "The Diseases of Occupations," *Twentieth Century Practice*, ed. by Thos. L. Stedman, New York, 1895, vol. iii. p. 311.

[5] McConnell, J. W.: "Case of Pronator Spasm in a Compositor," *Philadelphia Polyclinic*, Apr. 28, 1894, vol. iii., No. 17, p. 161.

The patient was a man twenty-nine years old, a compositor, with a good family history. He admitted the use of alcohol and tobacco in moderation, and he had had venereal disease some years previous to coming under observation. He believed that the trouble with his arm began six or eight years previously, although when he was learning his trade he noticed that he held his composing-stick in a manner somewhat different from others. Instead of allowing his hand to remain quiet, he kept it moving toward him, as though to facilitate dropping in type.

In 1885, before the present trouble became marked, he would, after a day's work, become conscious of some pain in and around the left shoulder-joint, but, as other compositors suffered the same discomfort, nothing was thought of this. In 1887 he noticed for the first time that while he was working his left hand, in which he held the composing-stick, would, as he describes it, "turn over," and many times spill a stickful of nearly completed work. No such motion took place when the arm was at rest. For some time previous to this the stick slipped in his hand, as though he had not sufficient grasping power. The peculiar turning motion became so persistent that the patient was forced to give up working at his trade and accept employment for nine months on a strawberry ranch, during which time the arm improved. Picking berries did not cause any increase in the spasm; on the contrary, while thus employed he was free from any physical disability. When he returned to his trade the arm condition again became annoying, but he found that by supporting the hand on the type-case the trouble was lessened in degree and at times was absent.

He worked at his trade for two years, during which period he was treated generally and locally, but in 1890 he was again forced to quit his trade. At that time he was "very nervous, often so much so that he was unable to see his copy;" he suffered with insomnia and vague pains. The left hand was very sore from resting on the case, and the palm at the hypothenar eminence was thinner than in the right. When he attempted to use the fingers of the left hand for fine movements the muscles of this hand and forearm became cramped and painful. Since 1890 the patient has had other employment.

His condition on coming to the Polyclinic was as follows: He is a well-developed man who is naturally left-handed. He suffers no pain, but when he attempts to use his left hand for such purposes as his trade requires the ulnar group of muscles swells and becomes quite hard, the hand is pronated and flexed on the wrist, and he is unable to keep it in a supine position except by forcing it into this position with his right hand. Attempts to bring the hand back to the supine position without aid cause contraction of the muscles of the upper arm and some of the trunkal muscles. The muscles involved in the spasm are hypertrophied and easily stimulated to contraction. Testing with the galvanic current shows no serial change in the contractions, but the response is so prompt and strong as to be almost myotonic or tetanic. Muscular power in both arms seems about equal. The measurements of the left arm exceed those of the right by half an inch at every point.

Brick-sorter's Spasm.—Recently another somewhat uncommon case of occupation disease appeared at my Polyclinic service, and has also been

recorded by McConnell.[1] The patient, a man fifty-two years old, had been employed for twenty-one years as a brick-sorter. He had never been a user of tobacco or alcohol, and his family and personal history were excellent. In 1894, after an idleness of some weeks, he resumed his occupation, and his first day's work caused great discomfort in the interosseous tissues of the right hand, which discomfort later became pain, and involved the whole hand, wrist, and forearm of that side as well as the left hand and arm. Labor increased the pain, and enforced rest for five months was the result. Under like circumstances he suffered from apparently the same condition in 1895, the trouble persisting for a few weeks. In July, 1896, a third very mild attack followed a spell of cholera morbus, involved both hands alike, and disappeared in two weeks' time. In December, 1896, two weeks of idleness were followed by the new employment of brick-setting. A few hours of this work sufficed to cause pain and disablement of the right hand. The right wrist, forearm, and arm, and the left hand and wrist were soon involved. A sensation as of a tight band about the right biceps developed and persisted until the next day. The muscles about the right shoulder were somewhat paretic, and the whole arm felt as if bruised. Some swelling was noticed at first, but soon disappeared.

There is no pain on manipulating the hands, fingers, or arms; no swelling, no paralysis nor paresis, no changes in sensibility to pain, touch, temperature, or position, and no ataxia are present. Reflexes are normal and the electrical reactions are unchanged. Attempts to perform such movements as are made in his occupation cause pain and muscular spasm in the hands, wrists, and forearms, so that he quickly releases the object with which he is experimenting.

The case is an uncommon one, rare in that both arms were affected simultaneously and in the number of attacks with recovery.

A Fatigue Neurosis of the Ocular Muscles in an Actor.—The following case, apparently of a fatigue disorder due to prolonged over-use of certain ocular muscles was seen by me in consultation. The man was a singer and actor in a popular troupe. In order better to counterfeit the character which it was his duty to represent on the boards, he was in the habit, nightly, for many minutes at a time, of frightfully contorting his face and eyes, accomplishing a strikingly effective but grotesque artistic feat. Both eyes were made to strongly converge, the forehead at the same time was corrugated, and the muscles of the cheek were drawn into special positions. One day he suddenly saw double. His double vision persisted and became more annoying, and it was soon discovered that he had paresis of the external straight muscle of one eye, with some accompanying pain. That the affection was really due to exhaustion of nerve centres appeared to be demonstrated by the fact that he could, by great effort, for a moment or two cause the affected eye to rotate outward beyond the median line, which ordinarily he could not do.

Weaver's Spasm.—A handloom weaver, forty-eight years of age, came to me complaining that for two years his left arm and hand seemed to be getting weaker and weaker, and for two months before coming under observation he had not been able to work because of "cramping" of his

[1] McConnell, J. W.: "An Uncommon Case of Occupation Neurosis," *Philadelphia Polyclinic*, Mar. 20, 1897, p. 126.

hand and fingers. These were almost constantly in a state of tremulous or spasmodic motion. The movements somewhat resembled those used in performing on a piano. Against his will the fingers of the left hand would flex, and he could not extend them except by forcing them with the other hand or by a persistent and painful effort. He suffered considerable pain, chiefly in the wrist. In his regular occupation of weaving he threw the shuttle many thousand times a day, and he believed that his work had something to do with bringing on the spasm, although he had never heard of any other handloom weaver being similarly affected. He denied venereal taint.

Fatigue Neuroses of the Larynx.—A few authors, as Fränkel,[1] Wyllie[2] and Lloyd,[3] have given some attention to the discussion of certain disorders of the larynx which are comparable to writer's disease and other similar neural affections of those engaged in the trades, arts, and professions. They are due to over-use of the voice, and are often associated with a general neurasthenic state. In their most typical forms they are seen in opera-singers, clergymen, public lecturers, professors, and others who for prolonged periods make use of their voices. The fatigue neuroses of the larynx are particularly likely to develop when the individuals are subject to unusual emotional strain or when work is carried on under circumstances of difficulty or embarrassment. Central changes are probably not so frequent in the fatigue neuroses of professional voice-users as in most of the other nervous diseases of occupation. The clerk, stenographer, typewriter, telegrapher, and pianist continue the strain upon the energizing and co-ordinating mechanism for a longer given period than do those who are compelled to make use of their voices in a professional way. Some increase in the laryngeal neuroses is to be noted as a result of the now almost universal practice of dictation in the preparation of material for public use by lecturers, clergymen, political speakers, and others. The symptoms in the laryngeal neuroses vary according to the particular variety of the disorder; they may be spasmodic, paretic, or paralytic. The nervous symptoms are usually associated with the evidences of chronic inflammation of the larynx or pharynx. The laryngeal fatigue neuroses are to be distinguished from various forms of organic disease of the vocal apparatus, such as inflammation, tumors, and palsies due to organic lesions, peripheral or central. A distinction must also be made between the pure fatigue disorders and hysterical affections of the voice; the two are, indeed, often associated. Hysterical aphonia is not infrequent in public speakers and singers.

Miscellaneous Forms of Nervous Disease due to Occupation.—It is not necessary to consider in detail all the so-called varieties of fatigue neuroses recorded in journals, textbooks and treatises. While a particular affection may give special features, some of the symptoms or syndromes considered under general symptomatology and in connection with the disorders just described will be present in all of the cases. A few words should, however, be said about some of the more important

[1] Fränkel, B.: "Ueber die Beschäftigungschwache der Stimme Mogiphonie," *Deut. med. Wochensch.*, Feb. 17, 1887, No. 7, p. 121.

[2] Wyllie, John: *The Disorders of Speech*, Edinburgh [reprinted from *Edin. Med. Journ.*, Oct. 1891–May, 1894.]

[3] Lloyd, James Hendrie: *Loc. cit.*

of the forms of fatigue neuroses not treated of in the preceding pages.

Performers on musical instruments, and especially pianists and violinists, as victims of these affections probably come next in order of frequency to writers and telegraphers. The disorder in these cases is usually of the spasmodic variety. Violinists may be affected either in the hand which manipulates the strings of the instrument or in the one which holds the bow, but most often in the former. Recently I have seen a violinist whose chief complaint was of pain in the wrist and great weakness in the hand and forearm.

Engravers are sometimes attacked with a spastic affection of the fingers, especially of the little fingers, and they occasionally suffer from anæsthesias and paræsthesias of the hands.

Blacksmiths, goldbeaters, and others who constantly make use of the hammer suffer from what is sometimes designated as hammer palsy and smith's cramp. The affection in some instances is so severe as to cause the hammer to be jerked from the hand, or a form of paresis or paræsthesia occurs which is more or less constant, efforts at using the hammer inducing the spasmodic manifestations. Tailors and seamstresses sometimes suffer seriously from attacks of cramp or spasm in the fingers and forearm. Flexion of the fingers may be suddenly induced. The patients may have a constant feeling of weariness and aching in the hands and arms. I have seen several cases of fatigue neurosis in artists, who have been compelled to discontinue using the brush. Cases of fatigue disease, almost identical in the main features of their symptomatology with writer's and telegrapher's disease, have been recorded as occurring among dentists from using the mallet or drill, among shoemakers in connection with their use of the hammer and awl, among ballet-dancers in the lower extremities, and among all the others enumerated when speaking of the occupations most frequently causing fatigue neurosis. Interesting papers have been written on the affections of the nervous system and muscular apparatus which occur among the Sheffield grinders and cutlers.

Burr[1] refers to a case which he saw in Morris J. Lewis's clinic, of a car-driver whose work required that he should press the brake-handle with the palm of the right hand continuously. Some months previously he noticed slight trouble in grasping the brake with the fingers, extension of the arm occurring with every attempt. Finally, the spasm and pain became so great that he was unfit for work. There was also occasionally clonic flexor spasm.

The most that can be affirmed with regard to the sewing machine is that its excessive use by women who are in poor health and illy fed may give rise to myalgia affecting the loins and limbs, and also to some general nervous symptoms. Women become accustomed to the use of the machine, as both men and women do to other forms of work involving a certain amount of nervous and muscular effort. Occasionally cramps occur in the legs. In some women of very delicate nervous organization the jar of the machine and its noises may have an influence in producing temporary nervous disturbances. A case has been reported

[1] Burr, C. W.: "Local Spasms, Occupation Spasms," *A Text-book on Nervous Diseases by American Authors*, ed. by F. X. Dercum, Philadelphia, 1895, p. 270.

of a drunkard's daughter who suffered from sciatica followed by amyotrophic paralysis in the popliteal region after excessive use of the sewing machine (Charcot[1]).

Cases have been recorded of players on the cornet and other wind instruments who have gradually become unable to sound the proper notes.

Miner's nystagmus is thought to be due to the irregular action of the ocular muscles as the eyes follow the flickering light of candles and lamps. In a curious case reported by J. A. Smith[2] the miner's sight completely left him when at work, but returned when he ceased working. When he assumed his working position (on the left side with the leg doubled under him), even in broad daylight, there was noticeable a slight oscillation of the eyes (Riggs[3]).

Diagnosis.—The diagnosis of writer's cramp, telegrapher's cramp, and of the various local fatigue disorders must be made on the same general lines. Although a knowledge of an occupation and of the method of pursuing it which might lead to any of these disorders would be of importance, still, it is necessary to guard against error in making diagnoses founded upon apparent etiology. I have known cases of paralysis agitans, disseminated sclerosis, muscular atrophy, syringomyelia, monoplegia of organic origin, gouty paræsthesias, alcoholic or other toxic tremors, and hysterical paralyses to be attributed to special occupations, such occupations at the most having acted only as exciting or precipitating causes. A case diagnosticated as engraver's neurosis, for instance, and treated as such for some time, proved to be paralysis agitans, although the patient was a comparatively young man. It will only be necessary to sound this note of warning with reference to patients suffering from organic disease who present symptoms simulating any of the manifestations of the fatigue neuroses. Hysteria is to be diagnosticated by some of its classical stigmata. Localized neuritis from traumatism or other cause must be carefully differentiated. One of the most important diagnostic points is one that is almost self-evident—namely, that the spasm, inco-ordination, or other disorder affects only or chiefly those muscles or groups of muscles which are concerned in the production of the movements peculiar to the occupation. The symptoms cease or are in abeyance when the parts are at rest.

Prognosis.—Mild cases of recent origin under favorable conditions may rapidly recover. If in the initial period of the affection, when the symptoms first become threatening, the patients are willing to give up their work and resort to proper methods of treatment, much can be accomplished. If the spasmodic symptoms have existed for a considerable time and are severe in type, the prognosis must be guarded, it then being more than probable that changes in the central nervous system have occurred, and that these are of a degenerative character.

[1] Charcot, J. M.: "Un cas de sciatique avec paralysie amyotrophique dans le domaine du poplité, determinée par l'usage exaggéré de la machine à coudre," *Le Progrès medical*, Apr. 4, 1891, tome xiii., 2d series, p. 273.

[2] Smith, J. A.: "Miner's Nystagmus," *Brit. Med. Journ.*, Aug. 29, 1891, vol. i., p. 476.

[3] Riggs, C. Eugene: "Nervous Disorders and Paralyses from Excessive Use of the Parts Affected—Vertigo, Tremor, and Lead-poisoning," *System of Practical Therapeutics*, ed. by H. A. Hare, Philadelphia, 1892, vol. iii. p. 419.

Even these cases are much improved by change of occupation and long-continued rest, but the disorder returns if the patients resume their former occupations. If the right hand is first attacked, as is often the case, and if the left hand is substituted, in most cases eventually both extremities will be implicated.

TREATMENT.—In all forms of fatigue neurosis prophylaxis is of the utmost importance. Those engaged in occupations predisposing to such disorders should live regular and systematic lives, should avoid dissipation, and should maintain their general health at as high a level as possible. Much can be done in the way of prevention by studying the methods least likely to give rise to unusual fatigue. The best methods of sitting, holding the pen, of forming letters, of using instruments, etc. should be those involving the least strain and awkwardness—methods which will allow the freest movement of the parts in use. The pencil or penholder in the case of writer's and stenographer's cramp may be held at different times in different ways, and thus avoid the extreme over-use of certain muscles.

With reference to the effect of writing on health and on the development of such affections as writer's cramp, much discussion has taken place as to the best methods of holding the paper and of forming letters. Neither the large angular writing so much affected by some of the fashionable schools, nor the old-fashioned inclined Spencerian and other methods of writing, are to be commended in these respects. In a newer and preferable system the up-and-down strokes are straight or vertical, and the writer naturally takes the least constrained and least harmful position.

Various appliances to assist the crippled scrivener have from time to time been invented, as Von Nussbaum's bracelet and the apparatus of Matthieu, of Charrière, and of Velpeau. Nearly all are constructed with one or two objects in view—either to give a larger surface for the hand and fingers to grasp, so that arm instead of finger movements may be used, or to bring muscles other than those affected into play; or both of these objects may be aimed at in the appliance. The penholder is sometimes passed through a wooden ball, an oblong piece of weighted cork, or even through a small potato, in order to give a larger grasping surface. Different forms of telegrapher's keys have been invented to decrease the monotony and fatigue of manipulation.

In laryngeal fatigue neuroses rest of the organs affected should always be insisted upon, and this for as long a time as possible. When accompanying inflammatory and catarrhal conditions are present, mild astringent gargles, like those of rhus and of solution of potassium bromide, will be found useful. In some cases massage of the throat and the application of a mild faradic current seem to have more than a merely psychical effect. Vocal gymnastics may be carefully used after a period of rest, and these may be combined with mild galvanic treatment, on the same principle that rhythmical exercise and galvanism are used in the treatment of neural disorders occurring in the limbs. Tonics and nutrients are of use in neurasthenic patients. If hysteria is present, and even when it is not clearly made out, advantage should be taken of suggestion.

Among the most important therapeutic measures for any or all of

the forms of fatigue neurosis are rest, general and local electricity—faradic, galvanic, and static—massage, Swedish movements, gymnastics, and special forms of internal, hypodermic, and local medication. Surgical procedures, such as myotomy, tenotomy, and nerve-stretching, also have been resorted to, but with little success.

J. Wolff[1] of Frankfort-on-the-Main originated a method of systematic gymnastics and massage which appears to have been very successful, especially in the treatment of writer's cramp. Wolff was a teacher of penmanship, and his method is a combination of active and passive movements of the muscles of the arm and forearm with massage and percussion. He practises massage daily for about twenty minutes, beginning at the periphery, and combined with this percussion of the muscles and peculiar lessons in pen-prehension and writing. As stated by himself, he employed "massage and gymnastic exercises, both of which, in addition to the material and mechanical influence which they exercise on the various nerves and muscles, have the far more important and essential object of acting on the psychically affected centres—that is, upon the morbidly affected will of the patient—and thereby moderate the action, or rather, by drawing the attention from the affected point, influence some new action of the mind."

Electricity frequently proves of value in the treatment of the fatigue neuroses, and galvanism should be thoroughly tried in conjunction with internal medication. Weak currents and long séances are the best. Temporary relief of the spasm is almost invariably produced by the use of galvanism. Faradization may be used, but it is usually contraindicated, especially in the spasmodic forms of the disease, when it may be harmful by over-stimulating. According to Riggs,[2] the tonic effects of general franklinization in debilitated and neurasthenic patients are decided. The actual cautery applied superficially over the affected muscles or over the spinal centres acts sometimes with surprising promptness in the relief of the pain and spasm.

The hypodermic use of atropine is not usually attended with much success, although sometimes it acts beneficially. When fatigue is marked the hypodermic use of strychnine salts may accomplish something. According to Lloyd, bromides and all sedative medicines are absolutely contraindicated for writer's cramp. As in most if not all cases evidences of under-action of the circulation and a depressed nervous condition are present, the general health of the patient should be built up. Phosphorus, quinine, iron, cod-liver oil, and similar articles may be employed.

[1] Wolff, J: "Treatment of Writer's Cramp and Allied Muscular Affections by Massage and Gymnastics," *N. Y. Med. Record*, Feb. 23, 1884, pp. 204, 205.

[2] Riggs, C. Eugene: *Op. cit.*

TRAUMATIC NEUROSES.

By MORTON PRINCE, M. D.

Synonyms. — Traumatic hysteria; Traumatic neurasthenia; Traumatic hystero-neurasthenia; Railway spine; Railway brain; Spinal concussion; Accident neuroses.

Definition.—The term "traumatic neurosis" has lost its original signification, which was intended to apply to a peculiar condition which was supposed to be a distinct affection, a clinical entity, and to be caused by physical concussion (trauma) of the brain and cord. This conception has been abandoned, and the term is now used only as a general expression for a group of affections—viz. neurasthenia, hysteria, and certain localized nervous symptoms—when caused by an accident. Trauma, in the sense of a physical shock, is also not an essential factor in the neurosis, which may equally follow any accident that is made up of only a psychical shock. In the vast majority of cases, if not all,[1] the affection is either neurasthenia or hysteria, or, perhaps more commonly, a combination of both (hystero-neurasthenia) or allied functional nervous disturbances. It must be borne in mind that we are obliged clinically to class under neurasthenia and hysteria certain allied types of functional nervous derangements which pathologically are not exactly either. The symptoms are of the neurasthenic or hysteric type, and, being functional, it is convenient to classify accordingly the neurosis. To be more explicit, the symptoms may at first be the expression of a simple nervous shock, and later of the action of the mind on the body, or they may be the continuance of a nervous shock by which a derangement of the harmonious workings of the nervous system is brought about; or they may be pure habit neuroses, paralysis, or sensory disturbances from idea, etc. For practical purposes it is more convenient to include all such functional disturbances as neurasthenia or hysteria.

These affections do not essentially differ from the same nervous condition when not due to an accident, and therefore would not require a separate description from that already given them in this work, were it

[1] This qualifying expression is used because there are probably a few clinicians who are still unwilling to give up the idea of a special organic disease in a minority of cases. I think the weight of the evidence is against this view; but, even if it be correct, there is no way of clinically distinguishing these exceptional cases from the others, nor have we any knowledge upon which we can base a pathological distinction. We have no real knowledge of the occurrence of any neurosis that is not neurasthenia or hysteria, unless it be a local neurosis like "traumatic lumbago" or a local paralysis dependent on idea. But such local neuroses are strongly related to, and generally are a part of, the others. It is probable that future analysis will show that neurasthenia is a composite group in which several functional affections will be distinguished, and it may be that one of these will be found to be particularly related to accidents. At present it is not practical to make this distinction.

not that the cause, especially where there has been either severe physical injury or a great psychical shock, tends to give them a peculiar stamp by which they acquire certain clinical peculiarities, and that they are liable to become complicated by local injuries.

Further, the fact of the frequency with which they follow railroad and allied accidents gives them a medico-legal importance which makes it necessary that they should be studied in respect to their etiology, pathology, and course.

The type is often a mixed one, made up partly of the manifestations of hysteria and partly of neurasthenia, but in many cases the peculiar stigmata of hysteria are wanting, so that we may make two distinct groups. When the marks of hysteria are at all prominent, the individual case is usually classed with that affection.

For a clear appreciation of the clinical picture it is desirable that the symptomatology of each type should be described separately, but, as they have a common etiology and pathology, these may be discussed once for all in reference to both.

Besides the affections just mentioned, it is believed by some competent writers that trauma, without causing gross lesions, may cause different kinds of scleroses of the cord, posterior and disseminated, and have included the latter in the "traumatic neuroses." But it is better that disseminated sclerosis, even if it has this origin, should not be considered apart from the same disease when due to other causes, any more than should tabes. As to gross lesions, like hemorrhage, laceration, myelitis, etc., they, of course, are distinct from traumatic neurosis.

Etiology.—Of the various etiological factors the most important for consideration are the kinds of trauma which most frequently cause the different neuroses, and the part played by each of the various elements of the trauma itself; that is, by the physical and psychical elements. As to the first, it is with railroad accidents that traumatic neuroses are obtrusively associated in the medical mind, both on account of the frequency with which these neuroses occur after these accidents, and the great notoriety given them by litigation. Probably the worst cases—or, perhaps more correctly, the greater number of the severest cases—occur from this cause. This is to be attributed both to the peculiar circumstances attending such accidents, and the after-conditions to which the patients are subjected when litigation follows. The effect of litigation, an important topic, will be discussed later. But railroad accidents are not by any means the chief form of trauma, although some of the terms, "railway brain," "railway spine," have been derived from this kind of accident. Any form of injury is capable of causing a neurosis, but it is a fact of importance, and one which throws light upon the pathology, that the same kind of accident—that is to say, for example, a blow upon the head or a fall which would be followed under certain conditions or in a certain class of people by a neurosis—under other conditions or in other people would be harmless.

The accidents most likely to lead to injurious results are such as are calculated from their nature to be of a terrifying character. Thus, electric shocks, particularly from the high voltage street-currents, elevator and bad runaway-carriage accidents, are frequent causes. The possible kinds of trauma of course are multifarious, and it must not be supposed

that only some accidents of this kind can cause traumatic neuroses. Sometimes the actual trauma, whether looked at from a psychical or physical point of view, is apparently comparatively trivial, or such as, when happening to most people, for the most part results in purely local injuries, yet in certain susceptible individuals induces nervous symptoms. For example, I find in my notes the following kinds of accidents recorded in individual cases: Slipping and falling on the back; falling off a step-ladder or down a few steps; bumping head against a doorway; spraining back and other muscles by lifting; slight surgical operation without anæsthetics; being thrown against another person by sudden stoppage of street-car. Somewhat more severe was a blow on the head, in one case from a descending railroad gate, and in another of an elevator, but in neither case was the local injury of importance.

Sometimes the accident causes a purely psychical shock, no physical trauma at all having been sustained, as when a person imagines he received an electrical shock, or, as in one of my cases, where a person in a brown study, while in a street-car, was suddenly startled by the conductor calling to him to get off at the point of his destination.

Thus it will be seen that the "trauma" may be of all degrees of severity, but it should be said that of the above minor accidents the resulting neurosis was in the greater number of a mild type. This is not always so, as, for example, the following rather humorous case: A young woman was trying the high kicking act with some of her companions. While one leg was in the air she slipped and fell backward. She immediately had some sort of an hysterical attack, and was taken to the Emergency Hospital, from which she was transferred to the Boston City Hospital. On entrance she had complete hysterical paralysis of one leg, with painful contraction of the same, hemianæsthesia, etc.

In many cases an important factor in the generation of these neuroses is pre-existing ill-health. Neurasthenics and hysterics are particularly liable, as a result of any slight accident, to have an accession of symptoms or a return of symptoms temporarily allayed. Similarly, a person who has previously suffered from these neuroses, but who has recovered from them, is apt to become the subject of a traumatic neurosis, which then manifests itself by a group of symptoms identical with those from which the patient previously suffered. Thus Mrs. H——, an hysteric with monoplegia, anæsthesia, and trance states, has a return of these manifestations after an accident, and Mrs. D——, a neurasthenic with headache, paræsthesia, dyspepsia, etc., again breaks down with these symptoms.

Similarly, a person whose resisting power is weakened by organic disease of the nervous system is easily upset by accidents, as in the case of the man above mentioned who was startled by the conductor of the car. This patient previously suffered from tremor of long standing, but to this typical neurasthenic symptoms were added.

The next important point in the etiology, and one which has been the subject of extended discussion, is the relative influence of psychical factors in the production of the neurosis. The matter is not without practical importance.

There is still considerable difference of opinion in this matter, though much less than there has been. In the earlier stages of the con-

troversy the tendency was to lay the greatest weight upon the physical element, but of late the tendency is in the opposite direction and to attribute the greatest influence to the emotional or psychical factor.

The physical theory may be traced to the influence of popular medical notions of the severity of railroad accidents, to the tremendous physical injuries sometimes inflicted, and to the doctrine of "spinal concussion" propounded by Ericcson and for a long time accepted by writers on the subject. Under the influence of these dominant ideas it was overlooked that not infrequently the physical trauma was insignificant, and of a nature that would be considered, under other circumstances, as entirely inadequate to produce a disintegrating molecular disturbance or meningo-myelitis (Ericcson) of the cord or of the brain. The doctrine of spinal concussion, which was of necessity purely speculative and not based on anatomical findings, later gave way to the German theory of "traumatic neurosis," by which was meant a special disease, *sui generis*, a clinical entity which had a distinct pathology of its own—namely, disseminated capillary hemorrhages, numerous foci of inflammation, and sclerosis and other degenerative changes scattered throughout the brain and cord, and which, like the molecular disintegration of earlier writers and the meningo-myelitis of Ericcson, were caused by the physical blow or concussion to the spine or cord. The influence of the notion derived from the observation of the severe cases of physical injury was still dominant, and inferences that were not extravagant when drawn from a special class of injuries were applied over a wide domain of nervous accidents. The idea of "traumatic neurosis" as a clinical or anatomical entity has now, as has been said, been discarded by the best observers, and the affection is regarded as a form of hysteria and neurasthenia respectively, with the same pathological basis which these affections have when occurring under other circumstances. Consequently, the question has ceased to be, What anatomical changes are brought about by physical concussion, blows, etc.? and has become, Are the manifestations of hysteria and neurasthenia excited by the physical shock, blow, jarring, shaking, etc., or by the psychical and emotional shock or other mental process?

The difficulty in determining the exact agency is that in most accidents there are both physical and psychical shocks, and, while an accident may be solely and exclusively made up of a psychical shock without any physical trauma, still, when trauma does occur, there necessarily must always be some mental shock also; consequently, while in the first class we can exclude all physical injury, in the second class we cannot exclude all emotional element.

Attempts have been made to obtain information in the matter by experiments on animals, in consequence of which the inference has been made that trauma, such as occurs in railroad accidents, may induce in the brain and cord various degenerative processes which are the physical basis of the traumatic neuroses. All such experiments have little relevance to the conditions under investigation, and the inferences drawn from them have been far-fetched and untenable. The pathological findings in man are limited to a few autopsies, the changes found being mainly those of arterio-sclerosis and minute disseminated sclerotic patches. At one time much stress was laid by some upon these findings,

and it was inferred that they were not only the disease processes underlying traumatic neurosis, but, reasoning backward, that they must have been caused by physical concussion. Many objections have been made to the acceptance of these conclusions, and, according to the more recent and best criticism, these changes cannot be looked upon as the basis of traumatic neuroses.

In addition to the more common objections (Dana, Knapp, Oppenheim), it seems to me that it may be said that it is very likely that in a certain proportion of cases physical trauma may produce degenerative changes of the kind found by Krönthal and Friedmann, but it is exceedingly questionable whether such cases can be assumed to be neurasthenia or hysteria, or whether they should be included as forms of the neurosis under discussion. It is well understood that if the brain is sufficiently shaken all sorts of lesions may be produced, from gross hemorrhage and laceration to more minute changes of the same kind. But when this occurs they can hardly be regarded as cases in point, even when the symptoms are of the type in question. Such cases might well simulate the neuroses, or the organic disease may be complicated by a neurosis, as frequently occurs (*e. g.* tabes and traumatic hysteria), in which case the organic changes may as well have been of ancient date as of traumatic origin. The autopsies are too few in number, in view of all possibilities, to allow of generalization. But it is too frequently overlooked that when systematized organic diseases like multiple sclerosis, myelitis, and tabes occur in persons who have been subjected to trauma, such diseases do not become in consequence a traumatic neurosis.

With the almost universal acceptance of hysteria, neurasthenia, and allied states as the essential components of traumatic neuroses, this experimental evidence and that derived from the autopsies, together with the consequent conception "traumatic neurosis" as a disease *sui generis*, has dropped into the background. It is exceedingly difficult to demonstrate that a blow or concussion by its purely physical (not physiological) effect can generate these neuroses. On the other hand, there is direct evidence to show that the psychical factor can be, and in certain instances is, the sole agent involved; and there is strong circumstantial evidence to show that the physical factor does not act unless the circumstances are such as to allow of the introduction of an emotional element. This, however, does not exclude the possibility of the physical shock acting through its physiological channels—that is, by excitation of sensory nerves—without the introduction of any mental element. This, in a certain proportion of cases, is probably what occurs. It is not, strictly speaking, psychical, nor is it physical as that term is ordinarily used, but rather physiological.

It is well to have a clear and definite conception of the questions at issue, and for this purpose the various possibilities of the modes in which trauma may act may be formulated as follows:

First: It may, as under other circumstances and in other parts of the body, produce by its pure physical effect, as contrasted with its physiological effect, solution of continuity of the nervous tissues, which solution may be microscopic or otherwise, and may be in the form of minute disseminated hemorrhages or disorganization of nerve elements, or some similar process, whether brought about by a compression or

squeezing of the brain, in accordance with Horsley's theory, or by a general shaking of the organ, or (Duret's theory) driving of the cerebro-spinal fluid into the ventricles. Strictly speaking, this is the true interpretation of the thesis of physical trauma acting as a physical cause, and the one which, when we think accurately, we have in mind.

Second: Trauma may act physiologically by abnormal excitation of sensory nerves, but the pathological effect of such excitation would be expended upon the various centres, particularly the lower levels of the cerebro-spinal system, without the intervention of any psychical, emotional, or other element. As an example of what is meant by such an excitation may be taken the effect upon the heart and vasomotor system which trauma is undoubtedly capable of bringing about in this way, and which is seen in surgical shock. It is within the bounds of possibilities that a widespread quasi-physiological effect of this nature may be expended upon extended areas and all levels of the nervous system.

Third: Trauma may act by its psychical or emotional effect. This would include any suggestive influence, for which some writers make a claim, that might take part in the process.

Of these three processes, the third alone has been proven to be the actual mode of generation in individual cases, while there is every reason to believe that the second plays a co-operative part. The only contention is whether the first is ever the real etiological agent or plays any part whatever.

When speaking of a psychical factor it will conduce to clearness of thought if we define precisely what we mean, and analyze, briefly, its composition. A mental shock from external causes involves, first, a sensory stimulation, and, second, a cerebral reaction of which the mental equivalent is an emotion, and of which the corporeal sequence is various vasomotor, cardiac, secretory, motor, and other phenomena. The sensory stimulation may be through the visual or auditory apparatus, as when we are startled by the sight of an impending danger or by the sound of an earthquake. Probably the great mass of the minor psychical shocks that occur in the course of ordinary life arise through visual or auditory impressions. But, similarly, emotional effects may be excited through the nerves of common sensibility by blows and other forms of physical injuries. In accident without trauma we have emotional effects excited by impressions through the first two channels; in accidents with trauma, by impressions from all three. Thus there is a part which trauma plays even when the exciting cause of a neurosis is an emotion or other mental factor. Going still farther in the analysis, we find that the mental state which has primarily been excited by a sensory impression may be made up of, first, certain ideas, vague or defined, of danger or possible consequences to the individual, or there may be a confusion, incoherence, or inhibition of ideas; and, second, of certain emotions which may be definite, like a particular fear, or indefinite, like dread, apprehension, anxiety, etc. Important is the fact that to these mental states are subjoined various quasi-physiological concomitants, such as palpitation of the heart, pallor or flushing of the face, tremor, muscular weakness, sensory thrills, etc., which may be regarded as the physiolog-

ical manifestations of emotion. In a greater or less degree they accompany all emotion, and are without doubt due to the extension of the excitation to associated lower centres of the cerebro-spinal system.

There seems to be good reason to believe these lower centres may be, and customarily are, excited from above by, or synchronously with, that part of the brain which subserves the emotions, so that the whole forms a symptom-complex. Or they may be excited directly from below from the ingoing centripetal impression, without the intervention of the cerebral equivalent of the emotions or even of self-consciousness. Finally, there may result instinctive automatic muscular contractions which aim at protecting the individual from the threatened danger, such as raising the arm, springing away. The way in which emotion or its cerebral correlative acts upon the highest levels of the brain, so as to bring about the hysterical stigmata and other neurotic manifestations, is unknown to us. That it does so is an unquestionable fact, as evidenced by common observation in non-traumatic hysteria.

Now, when we speak of the psychical factor of an accident exciting a neurosis, we must mean, if we take the trouble to form any clear idea at all, that the generation of this whole symptom-complex—ideas (and mental confusion), emotions, physical accompaniments of emotions, instinctive muscular action—in some way interferes with and upsets the normal working of the brain so as to produce a neurosis. And it may be said here—a point to be later elaborated—that in many instances an integral element in that neurosis is the persistence of the original "emotion-complex" in a more or less complete form. The various physical manifestations comprising it tend to become segregated, and, instead of subsiding, continue in association with the other phenomena of the neurosis. A moment's reflection will thus show that even the psychical theory allows an important rôle for trauma, for, by powerful stimulation of the sensory nerves, this largely aids in the excitation of the emotion-complex, which in turn resolves itself into and excites the traumatic neurosis.

Let us consider the third mode first. The effectiveness of the psychical factor rests upon the following facts: Many cases follow pure psychical shocks. Examples of this are well known and are the objects of constant observation. Witness a case of the writer which was that of a woman who, while sitting at an open window, thought she had been struck by lightning. This psychical shock was followed by a typical hysterical paralysis and anæsthesia with hysterical "attacks." As a matter of fact it was proved that the lightning struck a house a mile away. The case of a man who was simply startled while riding in an electric street-car has already been mentioned.

In the literature are to be found examples enough of cases which followed narrow escapes from trauma which did not actually occur. In numerous other cases the trauma has been insignificant, while the emotional shock has been violent.

One of the worst cases, for instance, in the experience of the writer was that of a man who was thrown out of his seat by the sudden stoppage of an electric street-car to avoid a collision with a car in advance. The car could not have been going at a rate of more than twelve or fifteen miles an hour, and, although a physical blow did occur, it must

have been very trivial. Many similar instances might be cited. Probably a large proportion of cases are of a similar character.

The converse of all this is not without point. Many forms of trauma which are followed by the development of a neurosis if occurring under circumstances that are attended by fear or emotional excitement or apprehension of danger, whether conscious or subconscious, are harmless under the opposite circumstances. An inquiry which the writer made into the effect of football accidents may be cited in illustration of this statement and in support of the psychical theory. Any one who has watched a football game must have been struck with the severe physical shocks to which the players are subjected. A player weighing one hundred and sixty to one hundred and eighty pounds and running at full speed must, when suddenly tackled and thrown, strike the ground, often on his head, with a force equal to that inflicted in many railroad accidents. Sometimes the physical blow must far exceed that experienced by the victims of the latter class of accidents, in which, as is well known, the trauma may be very slight even when a severe neurosis has resulted. Observing this, it occurred to me to write to the surgeons or managers in charge of the principal college teams, asking whether traumatic neurosis had ever been observed to follow football accidents. Answers were received from six colleges.[1] In not a single instance was any such injury reported. In every instance the reply was negative. It would seem exceedingly probable that if a physical injury, *per se*, could produce a traumatic neurosis, we should meet with it after football accidents. But this does not seem to be the case. Similarly, I have never heard of a traumatic neurosis caused by blows of the fists in sparring or prize-fights, but, it is true, I have never made systematic inquiries into this class of injuries. It would seem, then, that there was needed another element which is present in one set of accidents and is absent in another.

Corroboration of this is found in the statistics of the relative infrequency of traumatic neurosis in railroad employees as compared with passengers. According to Van Outen, out of 18,275 *injured* employees of a railroad there were only 8 cases of traumatic neurosis, or 1 in $2284\frac{3}{8}$; while of 844 *injured* passengers there were 11 cases, or 1 in $76\frac{8}{11}$. This difference probably is too great, as may be inferred from the fact that of the injured employees there were 767 with injuries to the back of various kinds. Many of these were probably in reality examples of hysteria, and should be classed as neuroses. Still, after making all allowance for such errors, a great difference remains in the liability of employees, or those who by occupation are habituated to the dangers of railroad travelling, and that of the travelling public.

Dana states with confidence that "persons who are injured while asleep or intoxicated are less liable to severe neuroses." If this be true, it must be regarded as evidence in the same line, as a physical blow upon the head or spine must have the same physical effect whether a man be awake, drunk, or asleep.

[1] The replies were made by the surgeons in charge of the teams of Harvard, Pennsylvania, University of Michigan, and Cornell, by the captains or managers of Dartmouth and Williams. All of those replying had had an experience covering many years.

Finally, it may be repeated that the symptoms of hysteria and neurasthenia when following trauma are similar in kind to those met with in the same affections when not of traumatic origin; from which it would seem reasonable to infer that it is most probable that the anatomical substratum in all cases is the same, and that there is no more reason in the one case to suppose the existence of organic lesions, such as result from physical injury, than in the other; so that we cannot infer backward from any supposed anatomical condition present to a probable physical cause.

As has been said, the few cases that have come to autopsy have not aided in the solution of the matter. Where symptoms of disseminated sclerosis are present and this condition is found after death, the case is manifestly taken out of the category of the class of cases under discussion, and is pertinent only to an inquiry into the etiology of disseminated sclerosis.

An apparent objection to the psychical theory may appear to some to lie in the fact that some persons state that at the time of the accident they were not conscious of any feeling of fear, and hence that there cannot have been any psychical shock. But this is taking too narrow a view of the matter. In a work of this kind it would carry us too far to enter into any extended analysis of the psychical condition of persons at the time of an accident, but a short explanation may be permitted. In the first place, the statements of patients regarding the conditions of their emotional states are unreliable. To describe with anything like accuracy the disturbances of mental equilibrium which occur in one's self during an accident is a task for which the human mind is not fitted. Faults of memory occur as well as faults of mental insight and feeling. Nor is fear the only emotional state that is the equivalent of psychical shock. A person may, so to speak, be tremendously "startled" without so definite a mental condition as fear being present. A few moments' reflection will probably recall instances of this to every one.

Then, again, investigations made of recent years into hysteria show that self-consciousness is very far from being coextensive with cerebral activity, and that impressions may enter the brain and result in cerebral and subconscious processes of which the self-consciousness of the individual does not take cognizance. There may be a subconscious or subemotional fear, or emotion of which the dominant mind is ignorant. Or, in other words, the effect which a mental impression may make upon the brain and nervous system of an individual cannot be measured by the amount of conscious reaction which it provokes (Putnam). In fact, sometimes the effect is the greater the less the impression enters into consciousness, as when, for example, in consequence of being absorbed in thought, we are startled by some familiar noise or touch which at the moment did not enter into consciousness, and therefore was not recognized. Had we been able to give our attention to the sensory impressions, they would have passed without effect.

I was able in one case to demonstrate the existence of a subconscious fear. The subject suffered from hysteria in consequence of a slight fall. After hypnotizing her I carried her in imagination through the accident and the events immediately preceding. When I came to the moment of the accident her face suddenly assumed an expression of terror, tears

came into her eyes, her face flushed, etc. In the waking state she exhibited no such emotion in connection with the accident, although speaking of it always excited pain and made her uncomfortable.

It is probably of etiological significance that these activities which go on in the lower levels of the brain seem to be in more intimate association with the organic functions than the activities of the highest levels; that is to say, with the vasomotor, visceral, and lower motor and sensory centres. So it would seem that impressions may excite the lower cerebral activities, and through them the centres for the physiological manifestations of emotion, without the emotional state itself arising in consciousness. In this way we may observe the apparently paradoxical phenomenon of the physiological expression of an emotion without the emotion itself—a not uncommon clinical observation.

Suggestive illustrations of this might be drawn from pathological material, but something very akin to it is a common experience with people with "weak nerves" or persons who have lost their self-control from one cause or another. Such persons commonly have very disagreeable feelings at the time of slight mishaps, such as the shying of a horse while riding or the close passing of another carriage while driving. Such accidents, without exciting such a definite mental state as can be called fear, in some people at least (with others it may be different) will "send the heart into the mouth" and "a thrill up and down the spine," make the heart palpitate, "catch the breath," and excite various muscular contractions, etc., and this over and over again on every repetition of the experience, although the subject knows there is no danger and makes every resolve to control the neurosis. In some cases it would be a stretch of language to say that fear was present. The subjective state is best described as that of being "startled," of which the manifestations are almost entirely corporeal.

The fact that in some cases consciousness has been almost instantaneously lost at the moment of the infliction of the trauma has seemed to some an objection to the psychical theory. But in view of what has just been said this objection cannot be said to be well taken. The absence of consciousness does not forbid the entrance into the brain of impressions which shall be effective in setting up processes in the lower brain levels, which are the physiological equivalent of subconscious states, and which are capable of giving rise to mental and corporeal shock. Then, too, the known rapidity of thought is so great that it is exceedingly difficult to exclude the possibility of thought entering the mind even when the supervention of unconsciousness seems instantaneous.

As to the first of the above modes of action regarding the efficiency of trauma acting as a pure physical agent, by jarring or concussing or shaking the body, in producing neurasthenia and hysteria, it must not be lost sight of that there is still considerable difference of opinion amongst competent observers. Nevertheless, although some writers (Oppenheim, Knapp, Putnam) hesitate to give up this notion, the tendency is to give a subordinate place to this factor in the great majority of cases, and attribute a preponderating influence to it only in a minority of instances. This conservatism of belief would seem to be an example of the survival of a theory after the supposed facts which it was originally proposed to explain were supplanted.

If it were true, as was not long ago believed by some, that the symptoms of traumatic neuroses were the manifestations of organic changes in the brain and cord like hemorrhage and necrosis, then little other etiological explanation would be tenable than physical trauma; but now, with the almost universal acceptance of the doctrine of hysteria and neurasthenia, and failure of satisfactory evidence for organic disease of the above sort, the pathological basis for the neurosis has dropped out of sight, and therefore, logically, the theory proposed to explain the supposed anatomical condition loses its reason for survival.

If organic necrotic lesions are the basis of traumatic neurosis, then this affection is not hysteria and neurasthenia; if we accept hysteria and neurasthenia, then we must give up the anatomical theory. This does not preclude that in some cases the effect of severe physical blows may be to cause hemorrhages and wounds of the cerebral tissues, by which certain symptoms simulating neurasthenia and hysteria may be caused; but such cases are not true "traumatic neuroses," but must be placed in the same class with those in which gross cerebral injury occurs.[1]

A critical analysis of the condition which we have to meet would seem, then, to show that the physical theory of causation rests, in the main, upon the ground of frequent association of violence and sequent neurosis; but, as this association is not absolutely constant, and as other factors are even more frequently present, the inference is not a necessary induction from the facts.

The second mode by which, theoretically, physical trauma may produce its effect is by its physiological action in distinction from its physical or psychical action. We have already seen that an important concomitant of the mental state induced by the psychical shock is the group of physical symptoms (cardiac, vasomotor, muscular, etc.) which accompany emotions. There is good reason to believe that these and other phenomena of a traumatic neurosis may be directly excited through the sensory nerves without the mediation of any conscious state whatever. In fact, it is highly probable that this process is a co-operating factor in the development of most cases, but it is exceedingly difficult to estimate the influence this factor may have. It is possible that sensory impressions may further diffuse themselves, so to speak, through those parts of the brain which are not accompanied by conscious ideation or other conscious states, and thus bring about that disturbance of the harmonious action of the brain which is a neurosis. An elementary instance of this diffusion is seen in the following incident which happened to the writer: He was once accidentally and suddenly struck on the ulnar nerve by an Indian club which a companion was swinging for exercise. There was no pain, but he was instantaneously seized with a violent rigor lasting some minutes. The intense rigor, without any other abnormal sensation whatever, was a most curious experience and made a deep impression on his memory. A similar process may take part in the accidents under discussion, but that the rôle played by it is a subordinate one is shown by the absence of traumatic neuroses in football accidents, in prize-fighting, etc. to which reference has been made above.

[1] It is open to question whether even in these cases a true neurosis does not complicate the organic injury just as it may complicate tabes.

If physiological shock were alone sufficient, it should be met with under these circumstances.

The second element which has already been referred to as a part of the make-up of the psychical factor is the antecedent condition of mind in consequence of which an injury occurring under one set of circumstances, as football-playing, is without evil consequences, while an equivalent injury under other circumstances is followed by most grave results. This element is a sort of preparedness of the mind in the form of fixed belief in or expectation of danger and evil consequences. The exact psychology of this state of preparedness is not easy to define, and it would take us too far out of the way to enter into an extended discussion of its nature, but its existence and influence are easy to recognize. The prize-fighter knows by his own experience and by that of others that he may receive terrible punishment about the head and body—that he may even be "knocked out" without any further evil effect than a temporary unconsciousness (which is not painful), and a little lameness and stiffness which will soon be recovered from. Violent physical concussion is not associated in his mind with any after-effects that tend to excite terror or anticipate danger. Expectation, one of the most powerful influences for self-suggestion, that ill effects will follow is not present. Consequently, when the physical blow is given it does not arouse a prearranged emotion or psychical state. On the other hand, I think we have more than theoretical grounds for believing that the travelling public is in the condition of mind of apprehension or expectation regarding the evil consequences of railroad accidents. The mind is so prepared by preconceived fancies, by public experiences in the past, and popular beliefs regarding dangers of this kind, that even a slight accident is liable to excite latent ideas, subconscious mental states, and emotions which have been already set in form, so to speak, and prearranged in the mind. This broad fact is, I think, patent; into further details it is not necessary to enter. An analogous experience, although trauma is not an element in the case, is observed at times of earthquake shocks, when the public becomes panic-stricken, and some people become affected with neuroses from the rumble and vibration of the shock, although a similar noise and jar from the passing of a railroad train would pass unheeded. The principle is a broad one, and if pursued in the opposite direction would be found to include considerable of the underlying basis of the nobler virtues, of courage, of fortitude, of martyrdom—in fact, of that disregard of personal danger which many callings and social customs have required in all ages.

Heredity—The teaching of the French school is that hysteria always depends upon heredity, which is the real cause, while accidents with trauma are merely exciting causes. The necessary corollary would be, No heredity, no hysteria. By heredity is meant that one or more of the preceding generation have been affected by a neurosis of some kind, not necessarily hysteria, such as insanity, neurasthenia, hypochondriasis, epilepsy, etc. Briquet traced 1103 near relatives of 351 hysterics. Of these relatives, 25 per cent. were affected by hysteria, epilepsy, insanity, convulsive diseases, or other nervous afflictions. On the other hand, he found a similar history in only $2\frac{1}{8}$ per cent. of healthy persons.[1]

[1] Quoted by Lloyd: *Textbook of Nervous Diseases by American Authors.*

Charcot insisted, as do his pupils, that the one real cause of hysteria is heredity. On the other hand, observers outside of France, while admitting the very great influence of heredity, do not find, in the traumatic cases at least, so large a proportion with an hereditary taint; but, on the contrary, it is the experience of most observers that in a very large number of cases no history of this kind can be obtained.

Aside from the fact that it is often a matter of opinion as to just what shall be considered as a taint, or what evidence thereof shall be accepted, this diversity of experience probably depends upon the fact, if we can judge from published cases, that the opinion of the French school is for the most part based upon non-traumatic hysteria and what is almost a select class of traumatic cases—that is to say, subjects in whom hysteria has been excited by comparatively trivial accidents of every-day life, whereas in the almost wholesale production of the disease in this country by railroad accidents a very large number of persons have had their nervous systems shattered by shocks of such intensity that the soil of heredity is not necessary for the effect.

It is hardly surprising that a railroad accident should excite hysteria in one without predisposition, while a slight accident, like pricking one's self with a hair-pin or even a blow upon the shoulder, should require an hysterical soil. At any rate, in this country it has not yet been shown that a neurotic heredity exists in a preponderating number of cases. The same may be said for traumatic neurasthenia, which likewise frequently may be traced to the existence of one or more of the neuroses in a previous generation; but in many traumatic cases this is not the case.

Age and Sex.—The greater number of cases naturally occur in males between the ages of twenty and fifty. This is simply because men are more exposed to accidents than women, and especially at this time of life. But traumatic neuroses occur at nearly all ages with the exception of very early youth or infancy.

Relative Frequency of Hysteria and Neurasthenia.—Dana makes the statement that traumatic hysteria is the less frequent of the two; but this is not borne out by the latest statistics of Knapp,[1] taken from his own experience. Out of 200 cases of trauma affecting the nervous system, but exclusive of local injuries, such as meningeal hemorrhage, epilepsy, neuritis, etc., 120[2] were either hysteria or neurasthenia, or 60 per cent. Of these 120, 70 were hysteria and 50 neurasthenia. But it appeared that in the cases which were not subject to litigation neurasthenia and hysteria were of equal frequency—viz. hysteria 20, neurasthenia 21—while in the litigation cases hysteria was much more frequent—viz. hysteria 50, neurasthenia 29. But these figures are not sufficiently large to be more than suggestive—*i. e.* to eliminate chance. Again, another interesting fact is that of the whole 200 cases (about equally divided between litigation and non-litigation cases, 97 and 103 respectively), 79 per cent. of the litigation cases were hysteria or neurasthenia, but only 41 per cent. of the non-litigation cases were such.

PATHOLOGY AND PATHOGENESIS.—There is no reason to believe that the general pathology of neurasthenia and hysteria differs from that

[1] *Brain*: Autumn number, 1897.

[2] It is not clear what kinds of organic disease were included in the remainder.

of these affections when not arising from accidents, and therefore what has been said elsewhere in this work need not be repeated here.

Why the same accident should at one time result in neurasthenia and at another in hysteria is a question to which, with our present knowledge, we are unable to give an answer, excepting that in a certain proportion of cases the development of the neurosis is determined by the influence of heredity.

But in many cases the distinction between neurasthenia and hysteria is more apparent than real; that is, is clinical rather than pathological. It is scarcely disputable that pure neurasthenia is a distinct affection from hysteria, but in a very large proportion of cases of the former, notwithstanding the clinical manifestations, the pathology is to be regarded as that of hysteria.

The difficulty is that most, if not all, writers tacitly assume that neurasthenia is a distinct pathological entity—that it represents a definite morbid condition of the nervous system; whereas it is a *clinical* conception and not a pathological one. It represents a collection of symptoms only, which may be the expression of quite distinct and varied pathological processes. Neurasthenia as clinically recognized may be the expression of persistent physiological fatigue, and nothing more, of the nervous centres, or it may be this plus various secondary symptoms (pain, dyspepsia, etc.), or it may be the expression of real organic changes, such as effusion into the ventricles (cerebral atrophy, multiple sclerosis, etc.), or—and this is a fact not as yet generally recognized—it may be a pure form of hysteria. Hysteria may express itself by a group of symptoms which are typically neurasthenic without the usual stigmata. That is to say, just as hysteria may at one time appear under the form of paralysis, at another of ataxia, at another of chorea, and so on, in each case simulating some other affection, so it may present a group of symptoms which are identical with those of true neurasthenia, and which, in this case, simulates that disease. An hysteric exhibiting such symptoms when looked at from a clinical point of view is said to have neurasthenia, although pathologically the disease is really hysteria. It would be more correct to classify such cases with hysteria and to speak of them as forms of hysteria. This would give us a clearer conception of the conditions with which we are dealing, and do away with much useless controversy; but custom hath it otherwise, and, as a consequence, it is usual to speak of all such symptom-groups as neurasthenia. In other cases, again, along with typical neurasthenic symptoms there are found one or more of the stigmata. There may be contracted fields of vision or cutaneous anæsthesia or muscular weakness. These cases are very properly classed as hysteria. Pathologically, they are, if this interpretation be correct, the same as hysterical neurasthenia. Now, it is evident that whether or not hysterical neurasthenia shall include any of the stigmata is purely a matter of arbitrary agreement. Of course hysterical stigmata may be present without neurasthenic symptoms. When the mixed form is present some writers speak of hystero-neurasthenia, but with the same confusion of thought as was manifested when we used to speak of typho-pneumonia. The discovery of germs has changed all that, just as it has given us a new conception of pathological diphtheria as distinct from clinical diphtheria. Unfortunately, in the

field of functional nervous disease we are still largely limited to making clinical distinctions instead of pathological ones. It would carry us too far to enter here into a discussion of this matter, even if it were necessary, but the following well-known facts may be taken as evidence for the hysterical nature of much that clinically passes for neurasthenia:

There is a well-known group of cases which at first, and for a considerable period of time, present purely neurasthenic symptoms, and then later break down with paralysis or anæsthesia. For example, a man after a railroad accident suffered from neurasthenic symptoms for a year. He then suddenly developed hysterical hemiplegia and anæsthesia, which persisted a considerable period of time, but finally disappeared to leave him in his original condition of neurasthenia. Belonging to this same group we frequently meet with cases, plainly neurasthenic, in which careful inquiry elicits a distinct history of some of the stigmata at some previous time in the course of the affection.

Then some cases, although free from the usual stigmata, exhibit prominently symptoms which are plainly hysterical. Sometimes hysterical symptoms alternate with neurasthenic symptoms, and sometimes the course of the disease, the rapid or sudden recovery after prolonged invalidism, is consistent only with hysteria. Some of the last-mentioned peculiarities are more often observed in non-traumatic cases.

Genesis of Symptoms.—The mode of development of individual symptoms of the traumatic neuroses has received very little attention at the hands of writers. While in organic disease the relation of symptoms to their underlying causes has been for the most part worked out, very little attempt has been made to do this in so-called functional diseases of the nervous system. This will prove to be a fruitful field for future research. At present we are not in a position to refer most of the symptoms of the traumatic neuroses to a pathological basis. For instance, we know little of the nature or origin of the general *feeling of fatigue* which occurs in neurasthenia and hysteria, or, for that matter, when occurring normally after exertion. We merely know that it is a feeling that is generally induced, but not always, by expenditure of effort, and that in most cases it is an index of what, for want of precise knowledge, we call exhaustion. But I think every clinician of experience must have observed that in many cases it cannot be an index of this condition, but is of pure psychical origin—a false sensation, as it were, which stands in the same relation to true fatigue that psychical pains stand to pains which have their origin in physical changes in tissue. It would seem that the sensation of fatigue is capable of being excited centrally by association with other feelings; for example, as a quasi-hallucination, like many other subjective symptoms.

One symptom is so common and often plays so dominant a part in the clinical picture that an understanding of its pathology is desirable if we would understand the neurosis itself. This symptom is *pain*. An extended discussion of it may therefore be permitted.

The painful sensations complained of are of various kinds, but conspicuous among them are general pains without very special location, such as up and down the spine or indefinitely through the head. Of the origin of some of these pains we know next to nothing. But more often, as shown by a study of the author's cases, pain is localized, as, for exam-

ple, in the small of the back, and then is known as "traumatic lumbago," or over a particular rib, or in the neck, or a particular part of the head, or along the course of certain nerves. In such cases careful inquiry generally elicits the fact that these painful localities were originally the seat of bruises or injuries, such as strains or blows.

At the time of examination, some months or possibly even two or three years after the accident, the most careful scrutiny fails to detect any evidence of local injury. Nevertheless, these pains may be such a source of suffering as to be the most prominent cause of disability and to occupy the patient's mind to the exclusion of all other symptoms. What is their pathology? Are they due to the persistence of local injuries, or are they symptomatic elements in the neurasthenic or hysterical state? I think that either view is erroneous. The customary statement to be found in writings on the subject is that these pains are due to strains of ligaments or muscles. This explanation is insufficient. While plausible when applied to the so-called traumatic lumbago—that is, pain in the muscles of the back and analogous regions—it will not explain similar pains in regions where there are no ligaments to sprain or where the nature of the injury was such as not to sprain, but simply to bruise; for example, pains of the scalp or cranium following bloody contusions, pains over the shins or ribs or arms following similar kinds of trauma; nor will it explain the persistence of pain following injuries which under ordinary circumstances would be well in a few days or weeks.

The true explanation, in the writer's opinion, is that these pains are of a psychical nature; originally excited by violence, they persist, after the subsidence of the original injury, *as pure psychical phenomena.* While I do not wish to deny that sprains in individual cases *may* persist, nevertheless, in the great majority of instances, this is not the case. They are the manifestation of the concentration of the mind on the injured part. The evidence of this is to be found in the analogy of such pain with similar sensory phenomena in non-traumatic cases, and the absence of all objective injury in parts subject to examination. Reynolds[1] as far back as 1869 called attention to this pathology in non-traumatic cases, ascribing them to the influence of idea. The point is, that any sensory phenomenon, particularly pain, which has been excited by any disease process, whether inflammation, functional disturbances, traumatism, or anything else, tends to persist, after the subsidence of the exciting causes, so long as the mind dwells upon it and imagines the persistence of the disease process.

It would be easy to cite illustrations of this drawn from non-traumatic cases. Every one of experience must have observed the tendency in neurasthenics and hysterics for pain and other disagreeable feelings to persist long after the local cause has ceased to exist.

It may almost be laid down as a law that in such subjects the tendency of pain is not to subside with the subsidence of the exciting cause, as in healthy persons, but to continue even without any fresh excitation from mental attention. If the mind is concentrated upon the local condition, the pain persists indefinitely; but it would seem as if, even when no such psychical factor can be detected, there was a tendency for

[1] *Brit. Med. Journ.*, Nov., 1869.

the pain vibrations once started to continue for a long period of time as a result of a single excitation; much as one hears the continuous vibrations of a musical instrument or sees the oscillations of a galvanometer after the cessation of the single mechanical or electrical shock. It would seem as if the defect was an absence of an inhibiting or damping apparatus. An analogous phenomenon in healthy people is the persistence of the sensation of the motion of the sea after landing from a ship or sailboat. This is a distinct persistence of a sensation as a quasi-hallucination after the cause has ceased to act.

When the mind is concentrated on the affected portion of the body the intensity and persistence of pain are even more marked. Such pains become, therefore, in time pure psychical phenomena, a sort of "fixed" feelings or quasi-hallucinations without objective source. Perhaps it may be that in some of these cases where there is no apprehensiveness or self-concentration of the mind this absence in only apparent, and that there is, to use the common phraseology of the day, a subconscious attention from out the lower depths of consciousness of which the patient has no waking knowledge. Be that as it may, the clinical phenomenon is indisputable.

In one subject now under observation during a long period of time I have observed this phenomenon over and over again. Exquisite pain and tenderness at the site of former perineal stitch, long-continued pain following the pressure of fifteen to twenty minutes, duration from a bicycle saddle, continuing local tenderness following a slight bruise of the arm or leg,—these are a few examples of what I have repeatedly observed in the same subject. In each instance the pain persisted long after the slightest trace of local injury has existed. Lately this same patient struck the globe of her eye with the corner of a sheet of note-paper. Not a trace of inflammatory action that could be detected followed, but troublesome pain sufficient to demand treatment persisted for some weeks. In this and other patients I have been able to prognosticate this phenomenon at the time of slight illnesses or accidents.

Most of the localized pains experienced in parts that have been the seat of blows or other injuries by subjects of traumatic neurasthenia and hysteria are explicable in this way, and there is little probability that they have other genesis. Thus a man, for instance, with hysterical stigmata suffered from long-continued severe pains in both hips and groins. Absolutely nothing objective was to be made out. The foci of pain corresponded to the track over his body of the wheels of a fire-engine which ran over him in the deep snow, without doing any serious injury to the bones or soft parts. Another exhibits a similar diagonal line across his body corresponding to the passage of the wheel of a light wagon. Another receives a blow on the head from a descending gate, and suffers afterward from continued localized pain in the head in the same place without objective signs of injury. Another, the subject of extreme hystero-neurasthenia, still suffers, several years after a railroad accident, from tenderness over the shins where originally were bruises. Nothing else objective is to be made out. The psychical genesis of such pain is self-evident.

Very instructive in this connection is an interesting class of cases in which pain is not persistent, but comes on in attacks which are excited

by various (external) agencies, particularly mental agitation. Thus, one patient suffered attacks of pain in a part of the body originally bruised, with other symptoms, when startled by loud noise, as from snow falling off the roof. Such a noise in his mind perhaps simulated the crash of the railroad accident. Another similarly suffered, even at times when free from pain, if in conversation the accident was referred to. The law of association here excited pain of a purely psychical origin.

As a convenient expression to designate this psychical phenomenon —the persistence of pain, or its revivification under the influence of attention—I venture to suggest the term *algogenesia* (*algos*, pain; *genesis*, generation). As a clinical fact it is not an isolated one, but has its analogies in other sensory phenomena belonging to each of the senses. In other words, it is the consequence of a general physiological law. In truth, the phenomena of a true functional disease are merely the expression of the same physiological law which under ordinary conditions determines the habitual and agreeable processes of the body, and, under extraordinary conditions, determines disagreeable and so-called pathological ones.

The same phenomenon may be observed in connection with the visual, auditory, gustatory, and olfactory senses, though perhaps not with each in the same degree. Thus, Galton, as is well known, has shown that some people have the power of seeing distinctly as an hallucination certain objects of their thoughts. Taking Galton's test-question, for example, many persons by concentrating the mind can see the morning's breakfast table before them with each object upon it as distinctly as if it were a vivid dream. The same is true of anything else upon which such people intently think. Some when thinking of numbers actually see these before themselves. This is the case with some mathematical prodigies.

The degree to which this faculty of visualization is developed in some people is extraordinary. Bastian in the development of his views of the mechanism of aphasia calls those persons *visuals* who speak by means of visual memory-pictures of words.

In visualization, then, there is a revival, as a quasi-hallucination, a memory-picture, of a past experience. These memorial images are revived by thought alone, by concentration of the attention, either directly upon the visual sensation itself or indirectly on an associated sensation.

Similarly, we have *auditives*. It is well known that a trained musician can hear the sound of the orchestra and each instrument of the orchestra from simply reading the score. Some people use auditory sensations for the use of language. I suppose, to use a familiar illustration, most people distinctly hear the sound of the speaker's voice when reading the address of a person with whom they have a personal acquaintance.

As to the senses of smell and taste, the revivification of past sensations of each of these is less vivid than with visual and auditory images, but, nevertheless, the faculty exists with some people in a greater or less degree.

Now, the same law must hold true for general sensibility as for special sensibility—for tactile, thermal, and pain sensations: the law is

general, not special. Pain sensations are, in some people, particularly easily revived as memory-images or quasi-hallucinations. Some perfectly healthy people, as is well known, have only to think of a pain or of a disease in a certain part to have the feeling. This is especially easy if previously that part has been the subject of painful disease. From my own clinical experience I could cite numerous illustrations of this. Sometimes these disagreeable or painful sensations are excited not by the thought of the sensation itself, but by associated images, as in one patient who was taken with nausea on the sight of a rubber mat which recalled a similar mat on board ship, or, in another case, when the thought of a possible cancer of the ovary in another patient excited pain in that region. These are all true memorial images of past sensations. Persons who exhibit the faculty may be called *algogenetics*, and the phenomenon when it constitutes a factor in disease *algogenesia*. We have, then, *visuals*, *auditives*, *algogenetics*, etc.

After psychical pains of this kind have persisted for any length of time they become, by the law of association, so united with other sensations and movements that they tend to become excited by mere association alone, with or without mental concentration—that is, they become *habit pains*.

Traumatic Lumbago.—A very common location of pain and tenderness is the lumbar region. These symptoms then simulate lumbago. The commonly accepted explanation of traumatic lumbago is that it is a persisting sprain of the muscles and ligaments. While it is impossible to disprove that this may at times be the pathological condition present, it is extremely unlikely that in the majority of instances the pain felt in this location differs from that just described and manifested in other regions of the body. In many instances the purely psychical character of such a lumbago is obvious. The exquisite hyperæsthesia of the muscles, extending even to the skin, is such as is never met with in true sprains. Not only the slightest movement, but the lightest touch over the skin, causes intense agony and throws the patient into great mental agitation. All analogy would seem to indicate that with certain exceptions—which, however, it must be difficult to make practically—the more moderate cases have a similar pathology. The origin of such a "lumbago" is undoubtedly to be sought in some trauma, but the persistence of the painful sensations can only be ascribed to psychical influences. If traumatic lumbago is really a persisting sprain, it seems inexplicable that it should not be met with amongst football-players, who should be particularly subject to such sprains.

Very often the original trauma can only be inferred from the nature of the accident, inasmuch as the victims of severe accidents are frequently too excited to remember much about the details, or loss of consciousness may have prevented this. But often enough the patient remembers quite clearly the circumstances of the accident, or the presence of bruises or wounds, which at the time were never felt, tells the story.

Ocular Pain, Headache, etc.—We are perhaps on less sure ground when we seek to explain painful sensations other than those associated with or originally excited by trauma; but some of them, such as pain and headache brought on by mental and physical exertion, are, in certain

instances at least, intelligible. Pain or discomfort from using the eyes is frequently complained of, as in non-traumatic cases. This pain is usually in the eyes themselves, or it may be in the head, and then may be felt as localized pains or simply as a headache. Why should use of apparently uninjured organs like the eyes cause pain? If the eyes were in a state of inflammation or had been the direct object of trauma, such sensations might be intelligible on general principles, but in the absence of such conditions it seems at first sight paradoxical that the use of a healthy organ should cause pain in itself or in adjoining parts. Yet this is one of the commonest of symptoms in neurasthenia. An intelligible, even if not easily demonstrable, explanation of this is to be found in the law of diffusion of energy under excessive mental or physical effort. When the mind is in a perfectly healthy state we are not conscious of any effort in concentrating the attention and fixing the eyes (including accommodation) while reading, but in doing this when the mind is tired we are conscious of a distinct effort, which increases proportionately with the increase of fatigue. Now, all analogy with similar processes elsewhere in the body shows that we cannot increase the expenditure of nervous energy to one centre without diffusing a greater or less amount of energy to associated centres. Consequently, when the mind is in a condition of fatigue the increased mental effort demanded to work the visual apparatus, to contract the muscles of the eyes, to bring about the necessary amount of accommodation, to fix the attention, and to unite together the various mental processes involved diffuses itself as nervous discharge to associated cerebral centres of the sensory region. These sensory centres may be those for the eyeball or closely associated centres, for the scalp, the cranium, the back of the head, the neck, etc. Painful sensations in each region would respectively result, and we should have eye-pain, headache, occipital neuralgia, etc. A similar explanation would account for not only the eye-pain, but the headaches, the pain in the neck, and even in the back, which are so often met with in eye-strain from errors of refraction, etc. An element in the causation of the more distant pains in this affection may be the fact that to fix the eyes it is necessary to fix the head; the latter act involves the innervation of the muscles of the neck and even of the back. Hence the increased effort required to overcome the effect of defective refraction would probably diffuse nervous discharge to all the sensory centres associated with the muscles involved. Evidence in corroboration of this explanation may be drawn from analogous processes of which we have definite knowledge. For example: in an epileptic fit the discharge starting in one centre spreads successively to adjoining centres. In contracting a single group of muscles, as in flexing a finger, with increasing effort more widely distributed groups of muscles are successively thrown into contraction, so that finally, when the whole force of the individual is exerted, all the muscles of the forearm, upper arm, shoulder, and even face and trunk, are violently contracted in the effort intended to contract simply a single group of muscles.

Physiological researches have shown that the diminished contractile power of a fatigued muscle is due to the accumulation in the muscle of chemical substances that poison the muscle. If these substances be

washed out, the muscle regains its power of contraction, but in fatigue, in consequence of this accumulation, the muscle responds with increasingly diminished force to a given stimulus or requires a stronger stimulus to induce a given contraction. Mosso[1] has further shown that the blood also of a fatigued animal is defiled by the accumulation of poisonous substances. These may play a part in the symptomatology of neurasthenia and hysteria, and may be a factor in the causation of the *sensation* of fatigue, which may consequently be central rather than peripheral in origin.[2]

Thus we have two factors contributing to the demand for increased effort in neurasthenia—diminished contractility of (fatigued) muscles after use, and diminished cerebral power. On the sensory side we have the researches of Head, who has shown in disease of the viscera the very common diffusion of the afferent impressions from these organs to the associated sensory centres for the corresponding cutaneous regions, producing hyperæsthesia, pain, etc. in definite cutaneous areas.

The phenomenon described by Möbius as akinesia algera has probably the same pathology, and exemplifies the excitation of painful feelings by nervous energy intended for muscular contractions alone. This is a group of symptoms which consists of pain excited by the voluntary contraction of certain groups of muscles. This may become so excessive that every movement may create pain, which thus may invade nearly every region of the body, and an incapacity for movement result that may simulate paralysis. The basis of the phenomenon is neurasthenia, hysteria, or hypochondriasis. Pains of this nature are evidently analogous to the ocular pains just described, as has been recognized by Oppenheim, who also points out that the pains observed by Erb associated with hearing, as well as the pain excited in some patients by the ingestion of food, are of an identical character.

It is evident that after a while such pains, like those of algogenetics, by the law of association become habit pains, or what I have ventured to call an association neurosis. They, then, are excited by association rather than by diffusion of excessive effort. I have been able to demonstrate experimentally the possibility of exciting the sensory centres by diffusion of energy intended for muscular contraction. The experiment, performed on myself, consists in forced flexion and extension or elevation and lowering of the arms. When these gymnastic exercises are carried to the point of fatigue a sharp stinging sensation, somewhat widely diffused, is felt in the skin of the chest area. The intensity of this feeling has been sufficient to make it a matter of some effort to continue the exercises. This observation has been repeated many times. I have found that the sensory areas involved roughly correspond with the muscular groups exercised.

The phenomenon of producing a pain in one region of the body by slight irritation (as by scratching) of the skin in a distant area is not unfamiliar ; likewise the intense pain in the back sometimes excited in

[1] Mosso (*La Fatigue, intellectuelle et physique*) has shown that the blood of a dog whose muscles have been tetanized by electrical stimulation if injected into another dog causes increased heart action and increased respiration.

[2] It is possible that the peripherally referred sensation of muscular fatigue may be of central origin, and due to the increased effort necessary to contract the chemically fatigued (poisoned) muscles.

neurasthenics by simply tapping the patellar tendon. The writer is familiar with one case, a man, in whom during mental fatigue the forced effort of reading causes an intensely disagreeable tickling feeling in the larynx. This may be explained, possibly, by the close association of the larynx with the faculty of speech. In normal individuals[1] the excitation of pain areas by muscular effort is of necessity, on principles of evolution, uncommon, but in neurasthenia and hysteria examples of this are of common experience.

The most probable explanation of this, it seems to the writer, is this diffusion of nervous energy under effort. This may be the cause of the pain in torticollis and some of the occupation neuroses in which muscular spasm plays a prominent part. Why in certain cases special sensory areas should be excited in preference to others must depend upon various intricate conditions which we are not yet in a position to define, but among them will be found the natural anatomical and physiological associations of the brain on one hand, and such antecedent pathological conditions on the other as may have originally excited directly these sensory areas and brought them into close association with the other centres in question.

Habit Symptoms.—It is very common for secondary symptoms of this kind, as well as original primary symptoms, to persist long after the pathological conditions which give rise to them have ceased to exist. They then continue as habit symptoms, and may complicate the original neurosis, or, persisting alone, may give rise to a pseudo-neurasthenia or hysteria. It is not inconsistent with probabilities that the way in which this comes about is as follows: The frequent excitation of nervous processes of whatever nature (motor, sensory, vasomotor, mental, etc.), in association with each other or with any physiological act, tends to group them together, and so unite them that the excitation of one of them or the performance of the physiological act excites all the others. At first the nervous processes (symptoms) are pathological, but with every repetition of the excitation the union of the different members of the group becomes firmer, until finally anything that excites one member excites the rest. For example, let us take the pain in the eyes (above discussed) brought on by effort in neurasthenia. At first this depends on fatigue and the increased effort needed to overcome this condition. By frequent repetition this pain becomes associated with contraction of the muscles of accommodation and of the eyeball, etc. The final result is that after fatigue has passed away any use of these muscles brings on the same pain, which then becomes a habit pain. Habit symptoms or association symptoms of this kind are in their nature truly functional, and, if this interpretation be correct, depend upon the same physiological law upon which all education depends. It is owing to the same law that we are able to acquire the faculty of language, the mechanical arts, the arts of music, painting—in fact, all accomplishments and knowledge. The musician who sees a bar of musical notation before him hears at once the sounds of various orchestral instruments necessary for the score, and his fingers automatically strike the keys of the instruments that will reproduce the sounds. The auditory images, the visual images, and the

[1] The writer is able to excite in himself sharp stinging sensations in the skin by gymnastic exercises carried to the point of fatigue.

motor impulses are all associated in one group. These are all physiological habits. The essential difference between physiological habits and pathological habits is that one is the result of voluntary education and is agreeable, and the other is the result of involuntary education and is disagreeable.

But the sensations that accompany physiological habits may have an unpleasant character, their purpose evidently being to aid in the preservation of the species. Among such habits may be mentioned the feeling of hunger and the local gastric feelings that are felt at a particular hour of the day—that is, in association with various daily actions. If the dinner hour is changed, say from one to two o'clock, these feelings after a time are correspondingly delayed. Likewise the feelings in connection with a desire to urinate, which may become associated with various social habits, and from this cause may become so frequently excited as to become a neurosis.

SYMPTOMS OF TRAUMATIC NEUROSIS.—The immediate consequences of the accident vary largely with its nature, according to the amount of physical violence that has been inflicted, and according to whether the shock has been purely psychical or has been accompanied by physical injuries. The final consequences, aside from local injuries, are very slightly influenced by such considerations.

By immediate consequences are meant the symptoms which immediately follow upon the accident. When one takes into consideration the great variety of accidents that may happen to an individual, the great variation in their intensity, from a mere tripping on the sidewalk to a fearful railroad collision or an electrical shock from a powerful dynamo, the different proportions in which the physical shock and psychical shock are mingled in the accident and the different ways in which individual nervous systems react to such influences, one perceives that the histories of the condition immediately following an accident vary greatly, and that it would be impossible to describe all varieties met with. This is, however, unnecessary, as the permanent condition, the real neurosis, is the later state and follows the immediate consequences. It is important, however, to have a knowledge of the more common early symptoms as they make the connecting links between the accident and the finally developed neurosis, and also because, from the point of view of pathogenesis, the final symptom group is often largely modified by and made up of symptoms which have persisted as a sort of parasites from the early stage.

In the following description reference is made mostly to serious accidents :

Loss of consciousness may or may not ensue, and is naturally much more likely to occur when there has been a direct blow upon the head, either from some missile or by the subject falling or being thrown violently to the ground. When it is due to a pure psychical shock, it is more of the nature of a faint or swoon, while that from physical trauma is naturally that which commonly follows blows on the head.

The testimony of patients is not reliable as to whether consciousness was lost or not. If the accident was at all severe in its terrifying effects, there is always so much excitement that the patient is apt not to be able to give a connected account of himself at the time. It is quite

common for the patient to say that, following the first crash of the accident, which is distinctly remembered, the first thing he calls to memory was finding himself doing this or that—extricating himself from his position or helping some one else, etc. Between the two events there is an interim in his consciousness which he is unable to fill up, and which he is liable to regard as a loss of consciousness, but which interval must have been occupied by conscious acts.

In other cases, however, there is a dazed condition of the mind. Consciousness is confused, so that the subject imperfectly takes in his surroundings.

From this instant the immediate effects differ so in different cases that no single description will suffice for all cases. Some persons are at once completely "knocked out," as it were, and are obliged to be at once transported to their homes and put to bed, to which they are confined for periods varying from a few days to several weeks, generally in consequence of surgical shock and local injuries. Nausea and vomiting are not uncommon in such patients, and pain from bruises and sprains prevents their leaving the bed.

In other cases, after the first shock and the patient has extricated himself from his dangerous position if it be a railroad accident, the subject, although in an excited state of mind, quite commonly is entirely unaware that he has sustained any injury, nervous or physical. He employs himself helping others, caring for the wounded, etc., and finally makes his way home. It is only after the accident is over, after the bracing excitement of the events of the day has subsided, that he begins to experience the beginnings of grave nervous disturbance, or these may be postponed for several days or a week. They then develop with an intensity that may be fully as grave as in cases that exhibit physical injuries from the first instant. Perhaps, if an examination could be made of subjects of severe accidents at the time, evidences of severe psychical and physical shock would always be detected which were not noticeable to the patient. Probably, besides local pain, tenderness, contusions, wounds, etc., various combinations of vasomotor and cardiac disturbance, tremor, muscular weakness, mental confusion, and emotionability, such as always or quasi-physiologically follow great emotional excitement, would almost always be found.

In other cases, again, the patient suffers from slight or moderate surgical shock; he is dazed or confused for a moment or two, feels weak, perhaps nauseated. After recovering his presence of mind he remains light-headed, physically weak, and experiences headache and dizziness, and some soreness and pain in some part of the body where he has been struck. He may feel sufficiently bad to at once go home, or may do so by the advice of friends, or he may continue trying to pursue his occupation the rest of the day or giving assistance if others are involved in the same accident.

From this time on, again, the progress of individual cases differs. Some of the last class of patients complain of the above symptoms for a few days, and exhibit considerable apprehensiveness regarding the seriousness of their condition. The symptoms, however, subside in a few days, a termination which is greatly favored by the assurance of the physician that no serious injury has been sustained. Others, on the contrary,

go on with greater or less rapidity to the development of the neurasthenic or hysterical state. Where this is to be the course of events, even at this, which might be called arbitrarily the intermediate stage, there is still great diversity in the condition of individual cases according to the nature of the accident. It will conduce to clearness to describe the results of the severer accidents first, meaning by this not necessarily severer forms of trauma, but accidents which, by their nature or in consequence of certain peculiarities in the make-up of the individual, produce a profound psychical or physical effect upon the individual. Railroad accidents notoriously are of this nature.

In cases where there has been severe surgical shock the patient is, of course, as has been said, confined to his bed and exhibits the well-known symptoms of that condition in addition to those about to be described. But whether surgical shock is apparent or not, or whether the subject is conscious of having been injured or not, perhaps even under the impression that he has gotten off "scot free," he is apt to be troubled with insomnia or restless sleep. He is haunted by nightmare and tosses about unable to sleep, and lives over again the accident. The scenes he has gone through are pictured with distressing vividness in his mind. He has a feeling of anxiety, apprehension, and general weakness not easy to describe. Perhaps the popular expression that he feels nervously shaken describes best his condition.

In some cases nausea and vomiting occur, and sometimes fainting spells at this stage. The next day pain and soreness in different parts of the body are added to the clinical picture. These localized painful feelings probably represent strains from sudden wrenches or violent muscular efforts or bruises of which the subject may or may not have been conscious. In this state these feelings are similar to what every one has experienced in the trivial accidents of every-day life. From this time on they play one of the most important rôles in the course of the neurosis, and when the accident has been of the kind to produce local injuries, like being run over by a wagon, they are the most prominent symptoms at the beginning.

Pain may of course be localized in any part of the body, but perhaps the most usual situations are the spine, especially the lumbar region (when it is known as "traumatic lumbago"), the neck, and head. When in the head it is generally the expression of the effect of blows. At first pain and tenderness are localized and confined to the region injured, but later it is apt to spread to neighboring parts and become more or less diffused, as all through the head and up and down the spine.

The patient feels weak and complains of lassitude, which is both mental and physical. His mind feels "muddled," and he is unable to concentrate it for any extended application. Physical exertion tires him as well, and he is unable or indisposed to make exertion, although he may attempt to carry on his usual vocation, but with poor success. Headache is very common, and may from this time on be more or less persistent. The symptoms now begin to crystallize themselves into one or the other of the different types that the neurosis is going to take. If *hysteria*, then anæsthesia, paralyses, and other stigmata develop, if they have not already done so; if *neurasthenia*, the characteristic manifestations of that condition, or mixed types, partaking more or less of each

condition may be the final outcome. But, as has been said already in the section on Pathology, many cases of neurasthenia are in reality hysteria without stigmata, and it is merely a question of terms whether a case of neurasthenia with one or more stigmata shall be classed with one or the other affection.

Traumatic Neurasthenia.

As traumatic neurasthenia resembles in general idiopathic neurasthenia, it is not necessary to describe the symptoms with any great extensiveness, but the reader is referred to the article on Neurasthenia in this work. Nevertheless, there are certain peculiarities of the traumatic form which tend to give it a stamp of its own. While most of the symptoms of the idiopathic form are met with in the traumatic form, still the grouping is usually somewhat different, and certain symptoms dominate the picture and stand out in such a way as to overshadow the others.

The essential features of neurasthenia are an incapacity for prolonged effort—whether this be mental or physical—and an increased irritability of the organism. There is an incapacity for sustained effort, which, if persisted in, brings on abnormally early and with abnormal intensity the sense of fatigue. This is what is called in athletic parlance "lack of endurance," and is shown on both the motor and sensory side. The patient who could before walk several miles can only walk a block or two without exhaustion. A few gymnastic exercises that would only refresh a normal person has the same result. He may be able to lift as great a weight as before, but a much less number of times. It is highly probable, however, that if careful measurements were made which could be compared with similar ones made before the accident, the maximum single effort would be found to be less. But such observations are rarely possible. Practically, there is no diminution of strength of this kind recognizable. After sustained physical effort the succeeding exhaustion is much more intense than that which ordinarily follows the effort necessary to fatigue a normal person, and persists a longer time, so that a few hours of effort are paid for by several days of suffering. The same thing is true of mental effort. There is the same incapacity for persistent application, which brings on a sense of mental fatigue and confusion of thought. But, besides fatigue, persistent effort brings on a train of symptoms varying in their character with the case; among them may be mentioned headache, insomnia, vasomotor disturbances, pain, and so forth.

As has been pointed out in the discussion of the question of the pathogenesis, some of these symptoms may be regarded as the effect of diffusion of nervous energy, in consequence of forced effort, to associated nervous centres and tracts. In this way feelings connected with different regions of the body are aroused by mental or physical effort directed to some other part.

Many of these effects are usually spoken of as manifestations of increased irritability, which is the second peculiarity of neurasthenia. By this is meant that the subject is abnormally sensitive to the influences of his environment. Heat and cold cause excessive discomfort or distress. Loud noises or noises not ordinarily noticed excite the patient, and in

some cases bring on attacks of associated local symptoms. General nervousness may be complained of, but in my experience this has been less common than in the idiopathic form. Slight causes excite the various local or diffused pains which are so frequently present. Likewise, he is mentally excited and angered by trivial contradictions and disappointments that ordinarily he would not mind.

In almost all cases of neurasthenia, with few exceptions, this tendency to fatigue and increased irritability, or lack of inhibition, is present, but special inquiry must often be made to determine the presence of these phenomena, as they are frequently overshadowed by other local and more objective symptoms which occupy the patient's mind to the exclusion of the more general symptoms. Besides what has just been said, only those symptoms of neurasthenia which are more commonly met with in the traumatic form need be mentioned here. They may be mental or physical, and either may predominate.

Mental Symptoms.—With the exception of the incapacity for persistent mental effort, the mental symptoms in the majority of cases are of slight character, and for the most part limited to a certain amount of irritability of temper, nervousness, and lack of energy. The memory is complained of as not being as good as before, and there is apt to be a certain amount of depression and apprehension regarding the patient's condition; but usually the amount of intellectual work which the patient can perform for one reason or another is diminished. In a smaller number of cases, on the other hand, the mental symptoms are more important: an inability to fix the attention and to concentrate the mind is noticeable; the patient finds difficulty in keeping his thoughts in hand and controlling them and in arriving at a decision in matters which ordinarily he would settle without difficulty. Small matters, in reality of little moment, seem of momentous importance and disturb his equanimity. Molehills become mountains in thought. The quality of intellectual work is apt to be decidedly inferior to what it was previously, although it is not easy in such matters to estimate exactly the amount of the difference. The mind becomes easily confused, and the thoughts do not arrange themselves with precision. If obliged to do accounts, the patient makes many mistakes or fails entirely in severe cases. The memory is apt to be bad, so that the patient finds himself resorting to various devices to prevent forgetting important matters. On the affective side equal mental changes are often recognized. The relatives point out that the character of the patient has changed. Although before amiable, of an even temperament, and cheerful, he has become irritable, depressed, and emotional. In extreme cases these mental symptoms may stand out in bold relief and dominate the picture.

But, as a rule, the most severe mental symptoms are met with in hysteria in which stigmata are present or have occurred in some part of its course. In such cases the patient may be so irascible that the slightest contradiction excites him to anger, and putting him to the inconvenience of an examination has the same result. It is not rare that this peculiarity exposes him to the suspicion on the part of the physician of exaggeration or malingering. He may be emotional and easily excited to tears. In some cases he breaks down from merely being questioned about the accident or his disabilities. Pain is not easily borne, so that tests

for tenderness excite him likewise to tears. He may be suspicious, apprehensive, and melancholic. Bad dreams are often a tormenting symptom, and insomnia, crying, and laughing spells may be noticed.

It must not be imagined that all cases show such extreme mental symptoms. On the contrary, the great majority are of a milder type. Every degree of mental change, from slight fatigue, mental confusion, and irritability to the most exaggerated types, are met with. As neurasthenia shades into, and often is, hysteria, the latter class, more properly perhaps, should be classed as hysteria.

Sensory Symptoms.—These are, for the most part, limited to pain and hyperæsthesia of the general and special senses. Pain plays a very important part in the clinical picture, and in a large proportion of cases is the source of the greater part of the discomfort complained of. It may be situated, as has already been said in the preceding part of this article, in almost any part of the body, but the most common situations are the back and head. When in the lumbar region it is known as traumatic lumbago, and is usually attributed to sprain of the muscles and ligaments. While injury is probably usually the exciting cause of this pain, yet probably in the great majority of cases, after the neurosis has continued any length of time, such physical injuries have long ceased to exist and the pain persists as a pure psychical phenomenon. It is probable that only in rare cases traumatic lumbago of long standing really represents any local organic change. The same may be said of pain in the cervical and dorsal spine, which is also common. Pain along the whole length of the spine, such as is seen in idiopathic neurasthenia, or what used to be called spinal irritation, is often met with. Pain, either localized or diffused through the cranium, is frequent in cases in which blows upon the head have occurred. In fact, it may be said that wherever a local injury has been sustained, whether a bruise, a sprain, or a fracture, there pain is likely to afterward persist so long as the neurosis continues. These pains may be more or less constant or they may be brought on by attempts at physical and mental effort. In some cases the patient is comfortable so long as he remains quiet, but any attempt to carry on his ordinary vocations results in paroxysms of pain. Quite commonly, worry or anxiety, anything that disturbs the mental equilibrium, likewise induces attacks of pain in these regions. The painful areas are also apt to be the seat of tenderness even when absolutely no objective condition exists that would reasonably account for them. The persistence of tenderness has probably the same pathology as that of pain—that is, it probably has a psychical genesis.

The *hyperæsthesia* above referred to is not, as a rule, very marked, but it may be present. *Anæsthesia* does not occur in neurasthenia. Sometimes *paræsthesia* in the form of numbness of one side of the body, of an entirely subjective nature, is complained of. A very common sensory symptom is *headache*, which may be more or less constant or brought on by various exciting causes. *Dizziness* is sometimes an annoying symptom.

Motor Symptoms.—Paralysis does not occur, and the motor symptoms are almost entirely limited to a lack of endurance and to a slight diminution in muscular strength. Twitchings and tremor are sometimes

observed. Both the *tendon and skin reflexes* are very frequently exaggerated, although with equal frequency they are normal.

Ocular symptoms are limited to asthenopia, and in some cases slight limitation of the field of vision. This asthenopia is manifested by early fatigue on use of the eyes (although this is more properly a part of the mental fatigue already described) and pain and headache after similar efforts. In severe cases very slight use of the eyes in reading, writing, drawing, etc. excites confusion of thought and a sense of prostration, severe pain in the eyes and parts of the head, as well as headache.

Slight limitation of the field of vision undoubtedly sometimes occurs, although this is not admitted by some writers. This is merely a question of classification, it being only a matter of opinion as to whether the existence of this stigma should take the case out of the class of neurasthenia and place it with hysteria. As has been said, many cases of what is clinically neurasthenia are pathologically hysteria. The distinction is of little consequence. This limitation of the field of vision may exhibit itself after fatigue from using the eyes, so that when the field is taken two or three times with the perimeter the second and third fields will be more contracted than the first. It has been my experience that when the test is not quickly made and the patient obviously becomes tired, apparently contradictory results are obtained when the observer attempts to verify his observations by repetition. A similar condition is the so-called "shifting type" (Förster) of contracted field.

The muscles of accommodation show also a tendency to early fatigue, as do those of other parts of the body. The pupils are apt to be unusually dilated, and may exhibit the oscillating phenomenon on exposure to light—that is, they first contract and then dilate.

The *auditory symptoms* are insignificant, and are limited to hyperæsthesia, but the dizziness which is not uncommon may possibly be looked upon as an auditory symptom.

Vasomotor and circulatory symptoms are important, and often a cause of distress. Feelings of heat and flushing, especially after effort, are often complained of, as well as cold hands and feet. This coldness of the extremities may be objectively apparent to the touch. There may also be excessive perspiration or the reverse. Nutrition may be impaired in a way that, after the neurosis has persisted a certain length of time, a diminution in the body weight occurs. Degenerative atrophy does not take place. In this connection the physician should be warned not to hastily assume the existence of atrophy of the limbs because of a moderate difference in the circumference of the two sides by measurement. Normally, according to Walton, there is a difference in the two sides of the body in about two thirds of all persons, and the maximum difference in the circumference of a limb may be as much as five eighths of an inch. The limbs of either side may be the larger.

The *pulse* is usually more frequent than in health, and not uncommonly ranges from 90 to 100. These figures may be considerably exceeded, but it is difficult to eliminate the effect of the examination upon the pulse-rate. An ordeal of this kind is sufficient to send the pulse up many beats, as is exemplified by the experience of those who make physical examinations for pensions, etc. According to Oppenheim, this rapid cardiac action may lead to insufficiency, hypertrophy, and

dilatation. The accuracy of this statement, it seems to the writer, is open to question, but it is very possible that the cardiac muscle may share in the general fatigue, and thus from imperfect contraction of the circular fibres fail to close the mitral orifice, and give rise to murmurs and temporary dilatation. Palpitation, dyspnœa, præcardiac pain, and distress may occur, but in the writer's experience these symptoms are less common in the traumatic than in the idiopathic variety of neurasthenia.

Digestive symptoms, although met with, are also less common in traumatic neurasthenia, which in this respect shows a marked contrast to the same affection when brought on by the debilitating effects of every-day life.

Irritability of the urinary mechanism, leading to increased frequency of micturition, may occur.

Impotence in its various forms is common, and makes the usual impression on the mind of the patient if a male.

An interesting phenomenon, and one which often stands out in prominent relief in the clinical picture, is what may be called the segmentation or association of symptoms; that is to say, there is a tendency for a certain number of the symptoms, apparently as a result of frequent repetition, to become grouped together and segmented off, as it were. Originally, such symptoms may have been simply the physiological expression of fear or fright, or may have been caused by physical injuries, such as pain and various distressing feelings. Later, they become grouped or associated together and form a quasi-neurosis by themselves, which may be excited by various external conditions which would not ordinarily induce such symptoms. For example, any attempt at mental or physical exertion induced in one patient the following train of symptoms, which were of marked severity and always occurred together, although when at rest he was comfortable: 1, a girdle pain; 2, lumbo-inguinal pain; 3, a tingling over the whole right side; 4, insomnia; 5, injection of eyes; 6, headache; 7, a nauseous testicular pain. This same group would also be excited by a loud startling noise which evidently recalled the noise of the railroad accident. This patient was entirely free from these symptoms unless he submitted himself to certain special exciting causes, but the symptoms themselves had previously been prominent at the beginning immediately after the accident, and for the most part were then due to local injuries.

In another patient nervous spells and feeling of fright, extreme tremor affecting the whole body, were excited simply by thoughts of his condition. In this case these symptoms were the primary ones originally excited by a psychical shock, and were the expression of fear. Later, they subsided, but only to be re-excited by thoughts connected with the injury.

In still another case during the continuance of the neurosis, and long after the patient was substantially well, talking about herself or the accident still brings on the attacks of pain, flushing, mental confusion, nervousness, and other symptoms. In all of such cases the symptoms thus excited are always the same, and the excitation of one ensures the accompaniment of the others, although they were originally referable to mental or local physical conditions. They persist long after such condi-

tions cease to exist. They often play an important part in the symptomatology of the affection, but to be recognized require careful examination on the part of the physician. Another set of symptoms worthy of mention is the reproduction of neurasthenic symptoms from which the patient previously suffered before the accident; that is to say, it sometimes happens that a person who has been subject to neurasthenia or hysteria, but has substantially recovered from these conditions, meets with an accident, which then has the effect of reproducing all the old symptoms in their original form.

Traumatic Hysteria.

As has been explained already, it is not always possible to draw a sharp line of distinction between neurasthenia and hysteria, for, while hysteria is not neurasthenia, yet, clinically, neurasthenia may be hysteria, and one condition may develop into or complicate the other. The French school of writers, whose views at present dominate in this field of neurology, tend to make the distinction depend upon whether or not the so-called objective stigmata are present. By stigmata are meant such defects as paralysis, anæsthesia, contractions, visual troubles, and hystero-epileptic attacks. Some would insist that the presence of one or more of the stigmata is requisite for hysteria, or at least for so-called hysteria major as distinguished from hysteria minor. But this is a mistake, as, in the first place, the distinction between major and minor hysteria is an arbitrary one, one condition shading into the other; and, in the second place, hysteria may be quite as well marked and quite as severe without stigmata as with them, unless amongst stigmata are included the whole symptomatology of hysteria.

Every clinician of experience was familiar with hysteria without these manifestations before the work of Charcot and his pupils had, by their brilliant studies, given such prominence to the objective manifestations.

The tendency at present is to leave too much out of sight those forms of hysteria in which paralysis, anæsthesia, and similar troubles are not observed. Nevertheless, considering the frequency with which these troubles occur and the distinctness with which the different types are defined, it is convenient to describe hysteria with stigmata by itself, as will be done here. Traumatic hysteria of this type does not differ essentially from non-traumatic hysteria, excepting that, as with neurasthenia, it is apt to be complicated by the effects of local injuries, and certain features are likely, but not necessarily, to be stamped upon it by the accident, so as to give it a peculiar clinical picture of its own.

Traumatic hysteria may occur—1, without marked neurasthenic symptoms, or 2, with marked neurasthenic symptoms.

Hysteria without marked neurasthenic symptoms is more apt to develop after slight accidents, such as moderate blows or slight falls. But whether the case be of the neurasthenic type or not, the physical manifestations (paralysis, etc.) may come on at once or after a slight interval—a period of preparation or meditation, as it has been termed by Charcot. This period varies from twelve hours to as many days.

As an instance of immediate development may be mentioned that of the case of my own, already referred to (p. 615), in which an "attack" and paralysis of the leg with contraction immediately followed a fall backward due to an attempt at high kicking.

Miura[1] collected 31 cases of brachial monoplegia, including three of his own. Of these 31 cases, 15 were caused by a traumatism of some kind, from a simple prick of a hairpin to a fracture of the arm. Of the 15, the time of development of the paralysis was noted in 9. It was immediate in 3, but after an interval varying from fourteen hours to eight days in 6.

Very slight blows may induce hysterical manifestations. In one of Charcot's cases a paralysis of the hand came on in a woman who slapped her child with the back of the same hand; but in such cases there is almost always a bad history of heredity.

Hysteria with neurasthenic symptoms is the form taken in the great majority of instances (hystero-neurasthenia). Especially is this the case after severe accidents in which there has been profound physical or psychical shock. The immediate consequences of the accident and the neurasthenic condition are the same as has already been described, and it is not necessary to repeat what was said. There is the same tendency to fatigue and nervous irritability. The local and general pains which have such prominence in traumatic neurasthenia play an equal part. The mental symptoms are also the same in kind, but in hysteria the most severe disturbances are met with. There is greater emotionability, and the alterations of personality are more marked. Of course there are, as well, mild cases with little change of this kind, the manifestations being largely limited to the physical troubles. But in the severer cases the subjects are described by the popular term hysterical. There may be uncalled-for attacks of crying and laughing, these occurring without provocation or from slight causes. The patients may be irascible, irritable, suspicious, and there may be ideas which are more or less fixed and mental depression. Sometimes such patients give the impression of being broken down by suffering, but the condition is only in part a consequence of this kind. The change in character is noticeable to the friends. The lack of self-control is marked. Incapacity for application, poor memory, etc. are present as in simple neurasthenia.

All this has already been described. All the symptoms that occur in traumatic neurasthenia may be present in traumatic hysteria, but they may be masked by more dominant manifestations. On the whole, it may be said that as the changes in the mental condition are greater, and there are physical defects in addition, the *disability* in hysteria is greater than in neurasthenia.

To these symptoms—the neurasthenic and mental—are soon added one or more of the stigmata. The most common of these are cutaneous anæsthesia and analgesia, paralysis, contractures, tremor, contracted fields of vision, amblyopia, defective color vision and hysterical "attacks." These may develop early, immediately or soon after the accident, or they may occur at any time in the course of the neurasthenic state, so that they may develop synchronously with neurasthenic symptoms or

[1] *Archiv. de Neurologie*, May, 1893.

be consecutive to them. They may, in the course of the disease, disappear, leaving the neurasthenia persistent.

For the practitioner to understand traumatic hysteria he must have a thorough knowledge of hysteria as a whole. Without such a knowledge it will be impossible for him to comprehend the meaning of much that is of common observation in the traumatic neuroses, and, although certain symptoms are more common in the traumatic form, he must be prepared to meet with every symptom that has been observed in non-traumatic hysteria.

It is only necessary here to describe the stigmata which are more commonly met with after traumatism.

Anæsthesia is one of the commonest of the stigmata, and follows the same laws as in non-traumatic cases. It may involve all forms of sensibility (touch, pain, temperature, muscular sense, special senses), or it may be limited to one or more, so that various combinations may be found. Pain, touch, and visual fields are most frequently affected. Analgesia with normal tactile sensibility is the most common of the different varieties. In intensity it may be of all degrees, from a failure only to perceive the lightest touch to absolute loss of tactile or painful sensibility. A fact of considerable diagnostic importance, and one that must not be disregarded, as is too often the case, is that sometimes, while the lightest touch may be felt, it is not so distinctly felt as in the sound regions of the body—much as, when an electric battery has run down, pressing the key will ring the bell, but not so loudly as before. This fact is so common in non-traumatic cases that it cannot be doubted in those of traumatic origin. Its importance lies in the evidence it affords of the existence of a stigma, and consequently of hysteria.

The distribution equally varies. It may be limited to the skin or may involve the mucous membranes, muscles, tendons, bones, and joints.

So far as concerns the skin, the most common form of distribution is that of a hemianæsthesia or of a limitation to one limb. Continental writers describe as common the islet form, where the anæsthesia is distributed in patches over the body like islets, but in the experience of clinicians of this country this has, I believe, been comparatively rare. This has been my experience, which is in agreement with that of Knapp, Dana, Preston, and others. In the hemianæsthetic form the anæsthesia is limited by the median line of the body, but I think I am right in saying that this limit is not so fixed as it is usually described to be, as it may be one or two inches on one side or the other. I think it is extremely probable that in consequence of the highly suggestable state which characterizes the hysterical condition the limiting line of the anæsthesia has too frequently, and all unconsciously, been manufactured[1] by the

[1] In three cases I have been able to verify this statement. In one case the examining physician, thinking the limiting line should be two inches from the median line on the anæsthetic side, demonstrated this boundary, but when erroneously told it should be on the opposite side, corrected, as he thought, his faulty observation and demonstrated the line in the new situation. In another case, not being convinced of the real existence of a hemianæsthesia which had been found, I offered to demonstrate anæsthesia in any part of the body, allowing my collegue to draw a limiting line anywhere. In this I was successful, the limiting line having been drawn diagonally on the abdomen. The mode was simply to suggest a line by touch. Another case was demonstrated in my clinic before the class.

examiner by suggestion. With the mind on the median line as a limit it is difficult not to suggest this boundary to the subject by the mode of touching or pricking the skin.

When the anæsthesia is confined to one limb, it takes the so-called stocking or glove form—that is, includes the whole circumference of the limb, and is limited above by a line running bracelet-like around the limb. I also think this line is not so sharp as it is usually stated to be, and that, although one can make out a bracelet-like line, this is confined to the existing profound anæsthesia, and that a second zone of slight loss of sensation extends upward and shades into normal sensibility. When the anæsthesia is nowhere profound, it is not easy to determine a limiting line, but the anæsthesia involves the whole circumference of the limb, and is not limited to the distribution of individual nerves or the areas supplied by spinal segments.

In paraplegia the anæsthesia may affect both legs.

Special Senses.—The most important of the disturbances of the special senses are those of vision, and of these the most common are *contraction of the visual fields*, *amblyopia*, and *color-blindness*. Contracted field is a common accompaniment of cutaneous anæsthesia, but may occur alone, without other impairment of sensation, and even be the sole sensory stigma. It is regarded as valuable evidence of hysteria. While it may be in one eye alone, in the great majority of cases it occurs in both eyes, but is most marked on the same side as the anæsthesia and

FIG. 65.

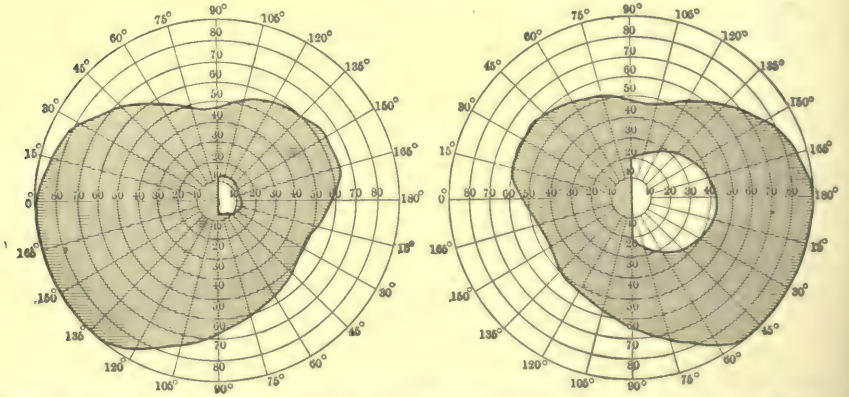


Diagram of fields of vision in a case of hysterical homonymous hemianopsia, from Hendrie Lloyd's clinic at the Philadelphia Hospital. Contraction of the sensitive half-field. Colors correctly named at fixing-point in the left eye. Colors correctly named within temporal side in an area 10° wide in right eye.

paralysis. The contraction is concentric, but usually is irregularly so. True heminanopia, if it occurs at all as an hysterical defect, is rare. It is denied by some writers. A few cases in hysterical subjects, irrespective of trauma, have been reported, but the fields usually are not of the pure hemianopic type, but show a contraction of the opposite (retained) portion of the field, so as to give the appearance of the heminanopsia having been produced by complete contraction in one half and incomplete in the other. In such cases the heminanopsia is usually associated

with hemianæsthesia and impairment of hearing, smell, and taste, and is then always on the anæsthetic side.

The color-fields may also be contracted, and one color may be more affected than another, as in non-traumatic hysteria, so that the red field may be larger than the blue, the reversal of the normal condition. Contraction of the color-fields probably occurs before that for form.

Impaired acuteness of vision, or *amblyopia*, is not uncommon. Rarely the loss is complete (amaurosis). Both eyes are usually affected, but the impairment of vision, like the contracted field, is most marked on the paralyzed side, while on the opposite side it may be slight. It is usually associated with anæsthesia elsewhere.

Parinaud was the first to describe a remarkable peculiarity of hysterical amblyopia which distinguishes it from that due to organic disease—namely, in some cases the amblyopia which is found on testing one eye *alone* disappears when the other eye is opened ; or, in other words, vision returns to the affected eye when both eyes are used, so that we have monocular amblyopia coexisting with normal binocular vision. These observations have been confirmed by Pitres[1] and Bernheim in France, and I have had the opportunity to do likewise in two cases.[2] This fact has great medico-legal importance, as the failure to recognize it may lead to the charge of malingering, as actually occurred in one of my own cases. As the usual tests for malingering (screen, "boite de flees," prism) would show normal vision in the alleged amblyopic eye, the charge is natural if the peculiarity of this form of amblyopia is not known. In one of my cases the circumstances were such as to render the *bona fide* character of the phenomena beyond question.[3] Charcot also found similar characteristics regarding color-vision ; that is, that certain hysterics with monocular color-blindness see colors normally with the affected eye in binocular vision. P. C. Knapp[4] in this country has confirmed this observation.

Monocular polyopia has also been observed coexisting with this form of monocular amblyopia. It was present in the first of my cases. Its pathology is not settled, but it is probably of psychical origin.

A withdrawal of the near point and approach of the far point in these cases was also observed, so that the patient saw relatively clearly at a certain fixed distance, in my case at about eight feet. Parinaud and Charcot explain this by a contracture of the ciliary muscle. By reason of this contracture the near point and far point coincide, and the eye cannot accommodate for objects placed either nearer or farther than determined by the contractures.[5]

Partial and complete color-blindness, already referred to, is most marked, when present, on the anæsthetic or paralyzed side.

[1] *Leçons clin. sur l' Hystérie*, i. 193.

[2] *Amer. Journ. Med. Sciences*, Feb., 1897.

[3] The subject, otherwise in good health and the applicant for a position on the police force, had better than normal vision with both eyes open, but each eye tested separately exhibited marked amblyopia.

[4] *Nervous Diseases by American Authors*, p. 164.

[5] I was led to criticise this explanation (*loc. cit.*) on the ground that, notwithstanding such a contracture, the subject should be able on optical grounds to read large A, Snellen, at one foot, which was not the case in one of my patients. But this criticism overlooked the fact that while this may be true for an eye with normal acuteness of vision, it may not be true for an amblyopic eye.

Hearing and smell may be impaired or abolished on one or both sides, but the impairment is most marked on the side of the paralysis and anæsthesia when these are present.

In contrast with deafness from disease of the auditory apparatus the hearing is better to aërial conduction than bone conduction, inasmuch as the deafness may be only for high and low notes, more rarely the latter. A. Galton's whistle is useful for detecting the former. A very common combination, therefore, is that which also occurs in capsular (organic) hemianæsthesia—viz. loss of cutaneous sensibility, smell, taste, and hearing on the same side, the chief difference being that in capsular hemianæsthesia the visual defect is hemianopsia, while in hysteria it is amblyopia, concentric limitation of the visual fields, and impaired color sense.

It remains to be pointed out that hysterical anæsthesia differs in one particular from that of organic disease—namely, although the subject is not conscious of the sensory impressions, nevertheless these reach the sensorium, and there enter into relation with certain cerebral processes in such a manner that they play a part with certain other psychical processes, the so-called subconscious states. It has been shown that in some hysterics, at least, the blind eye really does see and the anæsthetic hand does feel, for if such persons are hypnotized they will in the hypnotic state describe the impressions which were not seen or felt in the waking state. This means that, as Janet puts it, there is a "limitation of the field of consciousness" by which certain impressions are excluded from all the other mental states which make up self-consciousness for the time being, and are therefore not felt—much as in extreme absent-mindedness certain tactile feelings are excluded from the mind. Nevertheless, these feelings may play an automatic guiding part in co-ordinating muscular action. A consideration of this aspect of anæsthesia belongs to the general subject of hysteria, but it is mentioned here, as, unless it is borne in mind, certain apparently paradoxical phenomena might be misunderstood. For example, a patient of my own could finger various objects with her anæsthetic hand even when a screen was placed before her eyes. Unless sensory impressions had guided her fingers this would have been impossible.

Hyperæsthesia is common; it is more particularly found over the spine. In the lumbar region it is apt to be associated with the so-called lumbago, and in some cases the lightest touch to the skin apparently causes intense pain. It may be localized in points or patches in different parts of the body where bruises originally existed.

Paralysis.—In traumatic hysteria, as in the same disease due to other causes, paralysis is very common, probably ranking next in frequency to anæsthesia, which it generally accompanies. In fact, it is uncommon, though it does sometimes occur, for paralysis not to be accompanied by some one of the different forms of anæsthesia. The paralysis may be in the form of *hemiplegia*, *monoplegia*, *paraplegia*, and *quadriplegia*, or it may be localized and confined to one or two groups of muscles, as of the larynx or bladder. Of these varieties, hemiplegia and monoplegia are decidedly the most usual types following traumatism. Quadriplegia (all four extremities) is distinctly rare, and paraplegia is not common. I have seen quadriplegia once only, and this was not complete, and should perhaps have been regarded as

paralysis from idea. There was no anæsthesia at the time of examination.

The paralysis may be of all degrees from a slight weakness, so-called amyosthenia, up to absolute loss of power. The presence of simple amyosthenia is important from the point of view of diagnosis, and should not be overlooked.

One important respect in which hysterical hemiplegia differs from capsular hemiplegia is that the face is rarely involved. It has been a much disputed question whether the face is ever paralyzed. Charcot at one time maintained that this was never the case, but later observations have placed the matter beyond question, and it is now admitted[1] that exceptionally the face may be paralyzed in hysterical hemiplegia. The recognition of this fact is important from a medico-legal point of view. I have observed one undoubted case in which a moderate palsy of the lower facial muscles accompanied paralysis of the arm. The condition was caused by a street-car accident.

In degree the paralysis may be of all grades, but usually it is not as profound as in capsular paralysis, and the asymmetry of the pain is less. It may be limited to a few muscles of the face, but ordinarily the whole lower half of the face is involved, the upper half escaping, as in the capsular type. According to Richer, a constant accompaniment of this paralysis is anæsthesia of the skin and mucous membrane occupying a region corresponding to the paralyzed muscles; but anæsthesia almost always accompanies paralysis. In the writer's case, just cited, there was hemianæsthesia.

Paralysis of the tongue, which so frequently occurs in capsular hemiplegia, does not seem thus far to have been observed; nevertheless, Féré[2] has shown that there may be a weakness (amyosthenia) of the tongue similar to that which occurs in the limbs.

A condition simulating paralysis of the face and tongue may be brought about by a spasm of the muscles of the opposite side which draws the face and tongue away from the side of the paralyzed limbs. Charcot attributed at one time all supposed cases of facial paralysis to this cause. Such a contraction may coexist with a true paralysis of the opposite side and aggravate the distortion.

In monoplegia a careful examination will generally disclose some weakness of the other limb of the same side, and when the arm is paralyzed the face may be involved.

In one case I observed paralysis of the muscles of the back, so that the patient could not hold herself upright without support; she could not even sit. No paralysis of the limbs could be detected, but there was slight hemianæsthesia. The statement is met with that the paralysis in monoplegia and hemiplegia is on the same side of the body as was the injury, and considerable stress has been laid upon this point for purposes of differential diagnosis; but the statement is open to question. The possible fallacies are obvious. It is quite common for a blow to a limb to induce a paralysis of that same limb, a result which is quite

[1] See Richer: *Paralysies et Contractures hystériques;* Decoux: *Paralysie faciale hystérique;* Gilles de la Tourette: *Traité de l'Hystérie;* Pitres: *Leçons cliniques sur l'Hystérie, etc.*

[2] Cited by Richer: *Paralysies et Contractures.*

a natural consequence of psychical influences; but the matter is very different in paralyses due to injuries of the head and other distant parts. There would seem to be no satisfactory explanation of a blow to one side of the head producing by predilection a paralysis of the same side of the body, nor is there clinical evidence that it is a fact.

The paralysis may affect only certain movements, while the same muscles may be able to contract for other purposes. Thus an hysteric may be able to move the leg lying down, but not for walking. Extreme degrees of this constitute astasia-abasia, which consists in an inability to stand or walk, but a control over the legs for other purposes (hopping, jumping, etc.). This has been observed after an accident (Pel,[1] Bremer [2]). A failure to recognize this peculiarity has led to the charge of malingering. It is also well to remember that the strength of the grasp may be greater as measured by the dynamometer when the eyes are fixed upon the hand than when they are closed. This is analogous to the corresponding phenomenon of anæsthesia.

Also voluntary movements may be impaired, while the same muscles may contract for automatic movements; thus the muscles of the face may contract from emotions, though paretic for voluntary impulses.

Local Palsies.—Ptosis, strabismus, defective convergence, and similar motor troubles of the muscles of the eye in hysteria[3] are not so rare but that frequent instances have been reported, particularly in France, where much attention has been given to the subject owing to the writings of Charcot. But there has been much discussion and difference of opinion as to the interpretation of these defects. The question is whether they are to be regarded as due to paralysis or to contractures. The matter is not an easy one to determine. That many or most instances of these deformities are due to contractures is generally accepted by the best observers, and hence there is an inclination to explain all or most cases by this pathology, although a good many cases have been reported as instances of paralysis. Gilles de la Tourette,[4] who has given a careful critical analysis of recorded cases, and particularly those collected by Borel,[5] is disposed to look upon all reported instances of isolated third-nerve palsies as in reality cases of contracture, and also most cases of sixth-nerve palsies and combined palsies of all the ocular nerves; but he allows that palsies of the last two kinds, particularly of the sixth nerve, do, though rarely, occur, and he cannot exclude the possibilities of hysterical paralysis of the third nerve alone. He thus sums up his analysis: "From this long discussion we conclude that, like the muscles of the face, the muscles of the visual apparatus are so frequently affected by contracture that the question must be asked whether we ought to admit the paralysis of them. This, when it exists, besides being almost constantly accompanied,

[1] *Berl. klin. Wochenschrift*, 1893, p. 561.

[2] *Journ. of Nerv. and Ment. Dis.*, Jan., 1893.

[3] This statement, as well as the following description of the rarer ocular phenomena, strictly speaking, is based mostly on observations of non-traumatic hysteria. But as any phenomenon of hysteria may also occur in the traumatic variety, the distinction is not made here, as it is well that the practitioner should be prepared to meet the unusual manifestations, and be able to correctly interpret them.

[4] *Traité de l'Hystérie, etc.*

[5] "Affections hystériques des Muscles oculaires," *Archiv. d'Ophtal.*, 1886; "Affections hystériques des Muscles oculaires, et leur Reproduction par Suggestion hypnotique," *Ann. d'Ocul.*, 1887.

like contracture, with amblyopia, is accompanied more frequently than are organic palsies by secondary contractures. Besides this, the paralyses are associated, and more frequently alternate."

On the other hand, other writers (Richer, Parinaud, Pitres, Borel, Bristowe, Pansier, Kniess) more positively report or assert the occurrence of paralysis of the oculo-motor nerves, though it is admitted that it is rare.

Dilatation of the pupil (mydriasis [1]) is more rare, and still more so is *contraction* (myosis [2]). Both are almost always, if not always, due to spasm, and not paralysis.

Defective accommodation also occurs, which is also usually interpreted as due to spasm.

But, whether due to paralysis or spasm, the important clinical facts are that different forms of strabismus, closure of the eyelid, abnormal pupils, defective accommodation, may occur in hysteria.

Further, the weight of the evidence, on the whole, is that while these defects are usually due to contractures, exceptionally one or all may be caused by paralysis.

The clinical facts are, from a medico-legal point of view, important, as a failure to recognize them may lead to erroneous diagnosis and induce the expert to mistake serious structural disease for hysteria. The subject, however, requires further investigation.

One further peculiarity of hysterical ophthalmoplegia, according to Parinaud,[3] and confirmed by the observations of Bristowe [4] and Raymond,[5] is that while the patient may not be able to move the eye in one or more directions voluntarily, these same movements may be performed involuntarily and automatically. This phenomenon, if not known, might give rise to the suspicion of malingering. Gilles de la Tourette observes that it is analogous to the motor phenomena of astasia-abasia. This observer, in conjunction with Borel and Babinski, has succeeded in producing conjugate deviation, strabismus and blepharospasm by hypnotic suggestion, these conditions being due to contracture, not paralysis.

Paralysis of the adductors of the vocal cords, producing aphonia, has been observed, and paralysis of the detrusor of the bladder (retention), rarely of the sphincter (incontinence), occurs.

A case of *facial paralysis* of the peripheral type, involving the upper as well as the lower branches of the nerve, has been observed by Richer[6] in an hysteric who received a blow in the left eye during an "attack." It was accompanied by anæsthesia of the same side of the face and amblyopia of the same eye. The patient had also other stigmata previous to the accident. Although it is difficult to explain this case excepting by hysteria, still a single case affords too slight evidence for a general statement.

The development of the paralysis (hemiplegia, monoplegia, etc.) may be gradual or it may be sudden, and when of the hemiplegic type may,

[1] Galezowski: *Progrès médical*, t. vi., 1878: "A Case of Myosis and Myopia (due to Spasms)."

[2] Giraud Teulon: cited by Gilles de la Tourette.

[3] *Compte rendu du Service ophthalmologique*, 1888; *Archiv. de Neurologie*, 1889.

[4] *Brain*, 1885, p. 544, and 1886, p. 313.

[5] Quoted by Gilles de la Tourette: *Traité, etc.*, p. 430.

[6] *Paralysies et Contractures hystériques*, p. 192.

in the latter case, occur with loss of consciousness and simulate organic apoplexy, for which it is often mistaken by those unfamiliar with the phenomena of hysteria. The recognition of this mode of onset is important. Sometimes it follows a so-called "attack" in which convulsive movements take place. The mode of development is thus various.

The character of the paralysis usually presents certain peculiarities which distinguish it from that of the organic paralyses.

The muscles, unless contracture is present, are flaccid. This is the most common type. There is none of that rigidity which is almost always present in organic hemiplegia because of the descending degeneration along the motor tracts, and in paraplegia when the lesion is above the lumbar enlargement. The joints may be passively moved without exciting resistance. Sometimes, when the paralysis is absolute the arm, for instance, hangs like a flail from the shoulder-joint and may be passively swung about at will.

In other cases, however, *contractures* may be present, and then the paralysis may be made to simulate that of organic disease. But the consequent posture taken by the limbs is different, and it is not difficult to distinguish the immobility of contracture from the general muscular rigidity of descending degeneration.

These contractures are the same as when occurring in non-traumatic hysteria, and are described in the article on Hysteria (p. 689). I would merely point out that they are sometimes painful—that is, any attempt to move the limb causes excruciating pain. This pain must not be mistaken for that of joint disease: it is merely a sensory stigma.

The contractures of the ocular and facial muscles have already been referred to. They are of importance in that they simulate paralysis of the antagonistic muscles. A blepharospasm causes inability to raise the lid; a glosso-facial spasm causes an asymmetry of the face and draws the tongue to one side. The latter, instead of pointing to the side of the paralyzed limbs, as in hemiplegia, is drawn away from them and assumes the form of a hook. These contractures are rare.

The *gait* of hysterical hemiplegia is characteristic. Instead of swinging the leg outward or raising the body so as to clear the ground with the toe of the paralyzed leg, which in organic hemiplegia the spastic condition of the muscles compels the patient to do, the foot is dragged, the foot or toe scraping the ground, but the toe may be turned in or out.

There is no true degenerative atrophy of the muscles, such as occurs after injuries to the nerves and spinal cord.

In some instances, according to the observation of Charcot and Babinski (confirmed by others), a rapid wasting occurs in an hysterically paralyzed or contracted limb. In a few days the muscles are largely reduced in size, and after this the process remains stationary; but it is important to bear in mind that this wasting differs from real degenerative wasting in that there is no reaction of degeneration, the only electrical change being a diminished contractility. I have never myself observed this kind of atrophy in hysteria.

Notwithstanding these exceptional cases, in the great majority of instances the only wasting of the muscles is that which comes from dis-

use, in consequence of which one limb may become smaller than its fellow. The general practitioner must be cautioned against mistaking this, as well as the atrophy of Charcot, for degenerative atrophy, as is frequently done. The distinction must be based on the electrical changes.

Reflexes.—The skin reflexes from anæsthetic areas are diminished or abolished corresponding to the completeness of the loss of sensation. The same is true of the conjunctival and pharyngeal reflexes. The skin reflexes from non-anæsthetic areas are unchanged.

The tendon reflexes vary somewhat according to the type of hysteria, whether paralysis is present or not, and the variety of the paralysis. The statements of writers exhibit considerable variance in consequence, and often are not as definite as could be wished. In hysterical hemiplegia my own experience corresponds with that of French writers (Charcot, Pitres, Gilles de la Tourette); that is, that on the paralyzed side these reflexes are not exaggerated, as in organic hemiplegia, but are either normal or diminished; nevertheless, some writers speak of them as being frequently exaggerated.

In monoplegia Miura[1] states that of 31 cases the deep reflexes in the paralyzed limb were reported as more or less increased in 7 cases, diminished or lost in 7, normal in 3, while there was no record in 14. But the increase in some of the 7 was slight.

In paraplegia there is also a diversity of statement. According to Gilles de la Tourette, the deep reflexes are never exaggerated, but are frequently normal and may be diminished. This is undoubtedly too strong a statement, as occasionally they are somewhat increased, but the increase never equals that seen in disease of the cord. Ankle clonus is extremely rare in any form of paralysis, but occasionally has been noted.

In hysteria without paralysis the deep reflexes are probably normal in the majority of cases, as stated by Gowers, but in the neurasthenic type, especially when there is much spinal tenderness and evidence of lack of inhibition, there is a tendency to exaggeration.

On the whole, it may be said that in hysterical hemiplegia and monoplegia the frequency with which there is an absence of marked myotatic irritability is in striking contrast with the almost constant and marked increase met with in organic cerebral disease, and consequently the condition of these reflexes affords information of great value in diagnosis.

It will be seen, from what has thus far been said, that the characteristics of hysterical paralysis are the involvement of one or more limbs, with flaccidity of the muscles, without atrophy or degenerative electrical reactions, but combined with cutaneous anæsthesia of peculiar distribution, and contraction of the visual fields, and, perhaps, amblyopia and partial color-blindness.

Tremor is a common motor symptom and occurs in various forms. It may resemble the tremor of senility, alcoholism, paralysis agitans, or multiple sclerosis. It may, therefore, be rapid or slow, fine or coarse, cease or continue during rest, be originated or increased by voluntary effort (intention tremor) or not. It may be limited to the paralyzed limbs or be more generally distributed. It may precede the paralysis,

[1] *Archiv. de Neurolgie*, May, 1893.

and later cease on the development of the latter. It is apt to play an important rôle in the symptomatology of the traumatic cases, and may exist without anæsthesia or paralysis, and be almost the sole stigma present.

The Attacks.—The so-called "attacks" are occasionally met with in traumatic hysteria, but much less frequently than in the cases due to non-traumatic causes. The reason for this probably is that attacks, especially the major kind, are much more apt to be observed in persons of a distinctly hysterical "diathesis" with bad heredity, while hysteria from traumatism affects a much wider group of persons. The grand attacks are comparatively rare in all classes of cases in this country, and when they do occur, it is agreed, still more rarely copy the type described by the Salpétrière school. The probability is that the so-called grand attack with its four stages is largely a matter of education by surroundings and other modes of unconscious suggestion, just as the different stages of hypnotism of the same school were artificial products.

When attacks occur in traumatic hysteria they are almost always of the minor type. Probably the most common forms are attacks of crying, laughing, or trance-like conditions. Here belong also probably, certain temporary fits of depression, changes of mood and temper, together with various corporeal symptoms, such as cardiac pains and distress and pains and unpleasant sensations in different parts, which ordinarily usher in and constitute the prodromal stage of a major attack.

Nutrition is not usually affected in traumatic hysteria, but after a disabling protracted illness patients often lose body weight. This is particularly true of litigation cases, where anxiety and worry are superadded. The changes that occur in the paralyzed and contracted muscles have already been spoken of.

The skin of the paralyzed limb may be a few degrees colder or warmer than of its fellow, or one portion may be colder and another warmer. But the extreme vasomotor changes seen in organic paralysis do not occur. It would appear that trophic changes are more common in monoplegias than in hemiplegias, for out of the 31 cases collected by M. K. Miura[1] (before referred to), muscular atrophy was reported in 9. In 2 of these there was also osseous atrophy; in 1 there was an elevation and in 2 a diminution of temperature. In 4 others there was a simple lowering of temperature. Most of these cases were traumatic. On the other hand, the fact that these cases were reported by different observers renders some of them open to doubt, as in the absence of electrical changes it is not easy to determine, and must be a matter of opinion, whether a difference in the circumference of a limb should be attributed to real atrophy, to lack of use, or to a normal difference. In one of my cases this difference, as shown by the measurements taken before and after a year's interval, was due to a lack of growth, and not atrophy.[2]

Hysterical Affections of the Joints.—These are not as common as neurasthenia or paralysis or some other forms of traumatic hysteria, but when they occur they usually have their origin in an injury. At the

[1] *Archiv. de Neurolog.*, May, 1893.

[2] The case was one of hysterical knee-joint, but not of sufficient intensity to prevent the patient from walking about. *Boston Med. and Surg. Journ.*, 1891.

outset there is probably some local injury of a mild character, such as a bruise or a sprain. This later subsides, leaving no trace of its earlier existence, but the joint symptoms of pain, tenderness, hyperæsthesia, and muscular spasm continue as hysterical symptoms. The *modus operandi* probably is, in brief, as I have elsewhere[1] tried to show, as follows: At first, from physical reasons, every movement of the joint excites these symptoms, so that they become associated with simple movement and the idea of injury. Later, under the influence of this idea, which tends of itself to keep them alive as psychical expressions, mere movement alone excites them by association.

Other Symptoms.—Numerous other symptoms may occur in traumatic hysteria, but individually each is rare as compared with those already described. Among those occasionally met with are various forms of spasms (such as chorea and different forms of tic), ataxy, and nutritional changes. Of the last Knapp mentions a case where the hair temporarily turned white over a portion of the scalp which had been the seat of pain. Œdema, including the "blue œdema" of Charcot, has been observed. In one case of my own there were attacks of hemorrhage from the bowels, which were difficult to account for on any other theory than that of hysteria. The other symptoms, mental and physical, were typically hysterical. In non-traumatic hysteria hemorrhage from mucous membranes is known to occur. I have known it in one instance to take place from the nipples. Retention, more rarely incontinence, of urine may occur.

As I have said at the beginning, the practitioner must be prepared to meet occasionally with every symptom in traumatic hysteria that is known in the non-traumatic form, and a thorough knowledge of hysteria as a whole is necessary to comprehend exceptional cases, or, for that matter, any case of hysteria.

Prognosis of Traumatic Neuroses.—The frequency with which litigation involves cases of traumatic neuroses makes the question of prognosis one of immense practical importance. Unfortunately, it is a matter about which we have the least definite knowledge. Opinion varies between two extremes—between those who believe that the disease is of little gravity, and those who take a pessimistic view, and with equal positiveness hold that the outlook is most grave and that few cases recover. It is a regrettable fact that those who take the optimistic view are in litigation cases apt to be consulted by defendant railroad corporations and others, while the pessimists are sought by claimants.

Many difficulties attend the determination of the question. Most litigation cases, after the settlement of the claim, are lost sight of by expert neurologists, to whom the matter as a medical question is left for determination. A committee, of which the writer is a member, is at present engaged in an endeavor to learn the outcome of these and other cases. But at most such an inquiry cannot determine the curability of traumatic neuroses, but only the outcome of the disease after it has been subjected to influences which it is conceded must modify its course and intensify its character.

But even statistics of non-litigation cases do not necessarily give us reliable information as to the natural course of the disease, its duration,

[1] *Journ. Nerv. and Ment. Dis.*, May, 1891.

or the possibilities of recovery, but only as to the disease when subjected to chance conditions of environment. What I mean is that neurasthenia and hysteria are not self-limited diseases in the sense that diphtheria or typhoid fever is. The course of each is very largely determined by its environment. By environment is meant surroundings, treatment, and all moral, psychical, and physical influences which act upon the mind and body of the patient. It is well known that non-traumatic neurasthenia and hysteria are largely dependent upon such external influences as to whether they pursue a favorable or unfavorable course. Given a case of either which is capable of rapid recovery, and it may under unfavorable conditions be prolonged indefinitely. A neurasthenic or hysteric is often an unconsciously educated invalid, and it is not infrequent that unwittingly the physician by his treatment has taught the patient to continue an invalid, although his wish was to teach him to get well. It would not be difficult to cite cases in illustration of this.

Cases that are the subject of litigation are particularly exposed to evil influences of this sort, and, it is universally admitted, suffer thereby. Physicians, overlooking the greatest evil, are apt to shift the responsibility from their own shoulders and place it upon those of their patients. The delayed convalescence during the long delay preceding the trial and for protracted periods after it is ascribed to anxiety and worry on the part of the patient. There is every reason to believe that far worse are the unfavorable opinions given out of court and in court by the family physician and skilled experts who have been engaged to aid the claim of the patient for damages.

What would we think would be the prognosis in a case of non-traumatic neurasthenia that was submitted to such influence? Suppose such a patient was told by the family physician that his case was a most serious one, that the exact nature of his trouble was a little doubtful, but that he, the physician (having read Oppenheim and Dana), was afraid that the case was one of "traumatic neurosis," and that there were vasomotor and sclerotic changes in the brain, in consequence of which there was more or less degeneration; and, at any rate, whatever the trouble was, he, the patient, nevertheless was ruined for life and would never recover, etc. etc. Then suppose two specialists, skilled experts, perhaps professors of neurology, are called in for an opinion just to show that the family physician is right, which they proceed to do. They think perhaps there are no organic changes in the brain, at least of a gross sort, and that it is a case of "traumatic neurasthenia," but nevertheless the physician in attendance is right, and that the patient will never recover, but that he is disabled for life and perhaps will grow worse and worse. In order that our patient may thoroughly understand this, and may not be ignorant of any detail of his disease, we take him into a large room full of strange people and then let him listen to his fate. Each expert in turn publicly describes, in a most minute way, every symptom and sign the patient has (many of which he was unconscious of); one or more experts graphically describe, for the comprehension of the patient, the appearance of hemorrhages, vasomotor changes, broken spinal cords and brains, and what not, and then again formally states his belief, under oath, that the patient is affected with a most

serious disease with a long name, and that he is destined to a life of hopeless invalidism. Treatment, it is said, will have no effect. These opinions are given by the friendly advisers and well-wishers of the patient solely for his benefit. What would be the chances, under these influences, of our non-traumatic patient recovering? And yet this is what most litigation cases are subjected to. The wonder is not that few (as some think) get well, but that so many get well.

Nor are the cases that are not subject to litigation as far from evil influences of this kind as is generally assumed.

During the past ten years and until very recently the prevailing views, in this country at least, regarding the nature of these neuroses has been that of a particular German school which has regarded them as pathologically the result of structural changes in the brain, and the prognosis has often been correspondingly gloomy. With the physician controlled by opinions of this sort the influences tending to encourage recovery must be largely inhibited. The susceptibility of these cases to suggestion, conscious or unconscious, is notorious. Until all cases of traumatic hysteria and neurasthenia are given the benefit of the most intelligent treatment, which shall include the same expectation of cure, and the same therapeutic resources as non-traumatic cases, our knowledge of the possible prognosis must remain unsatisfactory.[1]

Nevertheless, it must not be inferred from what has been said that the outcome of these cases is entirely dependent upon medical and other influences. Many patients recover in spite of everything that is detrimental, and some cases obstinately remain uninfluenced in spite of the most favorable treatment. In France, where the views of Charcot have prevailed, not a few non-litigation cases which have persistently continued unimproved for years are to be found in the literature.

Reports of recoveries not based on expert examination of patients are unreliable, so far as absolute recovery is concerned; for in such cases, though the individual may be improved to the extent of being able again to engage in his occupation or not to give the appearance of invalidism, yet it may be found on careful examination that he is not the same mentally or physically as he was before the accident. Even traces of the physical defects, such as amyosthenia or slight anæsthesia, may persist.

Practically, it is important to distinguish between a degree of recovery which is sufficient to enable a subject of one of the neuroses to enjoy life and resume his vocations, and the absolute disappearance of all neurasthenic or hysterical manifestations. It is evident that the persistence of a slight degree of anæsthesia or a mild weakness of the left hand would not constitute a practical disability, although the case could not be claimed as a recovery. The time element is also important. A given case, as in non-traumatic cases, may continue unimproved or slightly improved during a long period of time, and then more or less rapidly recover. Therefore, for the proper classification of the final result the length of time that has elapsed since the accident must be

[1] An analogous effect of influences of this kind can be seen by looking back to the numerous bed-ridden cases of hysteria which apparently existed forty to fifty years ago, before our modern knowledge of hysteria was acquired. These cases were then thought to be organic.

known. It is also of immense forensic importance to know what length of time is necessary for recovery to take place. In litigation cases the question is always asked, What will be the probable time required if recovery occurs? To answer this question with the desired exactness requires more information than we at present possess. Until the desired information is forthcoming we must rely upon our general knowledge of hysteria and neurasthenia. But there is some little statistical evidence of more or less value.

In Miura's collection of 31 cases of hysterical paralyses 15 were traumatic. Of the whole 31, irrespective of causation, 22 were either cured or were improved, while of the 15 traumatic cases 3 were cured, 5 improved, and 3 were unimproved. The result in the remainder (4) was unknown. But these statistics are of little value, as the length of time that elapsed is not given nor the amount of improvement.

In a recent paper P. C. Knapp[1] gives the results in 28 cases of traumatic hysteria: 18 of these were complicated by litigation, 10 were not. Of the former, 1 recovered, 5 were considerably improved, 7 not improved, 5 died. (Of the deaths, 2 were due to pneumonia, 2 were attributed to exhaustion, and 1 was unknown.) Of the 10 non-litigation cases, 3 recovered, 4 improved, 2 not improved, 1 died (pneumonia). The time elapsed since the accident is not given. Nor does the expression "improved" give very much information.

It is probably safe to lay down the following general rules:

1. In traumatic cases the tendency is to recovery, unless prevented by injudicious treatment or other influences.
2. The earlier appropriate treatment is begun, especially in neurasthenic cases, the better the prognosis.
3. Many cases entirely recover.
4. Some cases only partially recover, so far as the complete disappearance of the symptoms is concerned, but yet recover sufficiently to enjoy life and to resume their vocations.
5. A minority of cases do not improve at all, or not sufficiently to make any material difference in their life.
6. The time necessary for recovery varies with the peculiarities of the case, the surroundings, the existence or not of litigation, etc.
7. While litigation is in progress little improvement can be expected, though it may occur.
8. The longer neurasthenic symptoms have persisted the more firmly established they become (habit symptoms), and the more difficult they are of cure.
9. Hysterical stigmata may disappear after existing a long time (many years).
10. Severe mental symptoms make the prognosis less favorable for a complete cure.
11. Litigation prolongs and intensifies the disease by suggestion on the part of the physician and auto-suggestion on the part of the patient.

TREATMENT OF TRAUMATIC NEUROSES.—The treatment must be mostly in the form of mental therapeutics, and all resources of that kind should be employed. First and foremost, in order to get rid of the

[1] *Brain*, Autumn, 1897.

restrictive influences of auto-suggestion on the part of the patient, it should be explained to him that no physical injury has been sustained, and that his trouble is either neurasthenia or hysteria, as the case may be. This should be impressed upon him as soon as his confidence has been gained, and he must be made to accept and believe it. It is also well to explain the nature of the pains, hyperæsthesia, and other distressful feelings, making him understand that the former are not due to local injuries when it is the case (as it usually is after the first week or two), but are of a psychical nature or due to habit, etc. Explanations of this kind always require tact, and rarely can be made with benefit before the patient's confidence has been gained. Patients, especially those who are claimants for damages, rarely welcome information of this kind, but generally resent it as in some way reflecting upon their personality. They do not wish to hear, whatever they believe, what might be interpreted as prejudicing their case. Even those who are not litigants are apt to infer that if no physical injury has been inflicted, it will be thought by others that they do not suffer. Nevertheless, it is desirable that the patient should understand his disability if possible. When it is not possible he must, at any rate, be made to understand that it is curable, and that whether cure or not takes place largely depends on the co-operation and attitude of himself.

When litigation is in progress little can be hoped for until this is finished, but sometimes improvement occurs up to a certain point. It is not a bad plan to make the patient further appreciate the risk which he incurs of delayed or incomplete recovery if he is exposed to the evil effects of litigation. He can then take his choice between riches and health.

In severe cases isolation—that is, separation from a bad environment and removal to a beneficial one—is of the greatest value. The principle is the same as in non-traumatic cases.

The "rest-cure," *per se*, excepting so far as it secures isolation and has a mental influence, is illogical and useless, and aside from a certain class of cases is not to be recommended. As a form of mental therapeutics it might be of benefit.

If a patient has become physically "run down," of course he must be well fed.

The local symptoms must be allayed by various expedients. It is very desirable that pain should be inhibited as soon as possible, and habits of this kind nipped in the bud before formed, or, if formed, broken up. For this purpose there is nothing better than electricity, especially the static variety. In using electricity its suggestive power should be taken advantage of and used with all the influence of which it is capable. The static spark is very effective in this respect. By its means pain can often be kept in subjection or dispersed. Also for the treatment of paralysis and anæsthesia electricity is of great value. Faradism for this purpose acts well; its effect is probably mental. The patient must be taught to exert himself without consequent fatigue or other painful results. It is not uncommon that a patient refrains from doing, not because he dreads pain or other kinds of distress induced thereby, but because he is afraid he will do himself permanent injury. Such persons should be assured that no matter how much they suffer

from doing anything, they cannot do any real harm to themselves (when it is the case, as it usually is). This mere assurance sometimes has surprising effects.

Hypnotic suggestion has been used with good effect, and some cases cured, but in my experience equally good results can be obtained by suggestion in the waking state by use of the static machine.

It is important to distinguish the sensation of real fatigue from that of false fatigue, which is, as has been explained, a pseudo-hallucination like hysterical pains. When this sensation is of the latter kind, it is not benefited or relieved by rest. The more rest is given to it the more it demands, until the patient becomes bed-ridden, and then, after nothing but rest, the patient feels as fatigued as before. Many a patient has thus been educated to feel fatigue on the slightest exertion. Such cases should rather be cautiously and gradually taught to exert themselves without suffering fatigue. The same is true of many pains and digestive disturbances.

Self-control must also be inculcated, and again the patient must be taught. He must learn not to be upset by his own symptoms, not to be awed by them, but to treat them with contempt and to hold his emotions in check. Some patients, figuratively speaking, kneel down before their symptoms and worship them as an idol on an altar. To speak disrespectfully of one is to insult the individual. Isolation is here of value, in that the patient is not subjected to disturbing influences, and therefore not put under constant strain, and for many other reasons.

Hydrotherapy has given good results, and when facilities are at hand it may be used.

Some cases must, on account of their weakness of character, their egoism, or (extreme cases) the shattered condition of their minds, be blindly led, without any attempt at co-operation on the part of physician and patient. For such cases the "rest cure" may be suitable.

It is rarely of any use to force patients, but rather cure must be gradual. Although some cases rapidly, almost suddenly, recover, it is not usual.

The possibility of cure after long periods of time is attested by the well-known cases of hysterics with paralysis or contractures who have been "miraculously" cured at Lourdes and other places.

On the lines here laid down a systematic plan of treatment should be instituted and rigorously carried out; and, above all, the patient should not be left to his own devices and the management of himself, but be under the constant influence and control of a physician.

SEASICKNESS.

By W. GILMAN THOMPSON, M. D.

Synonyms.—Naupathia; Kinesia; Motion-sickness; Mal de mer (French); Seekrankheit (Ger.).

Definition.—Seasickness is the name given to a group of symptoms characterized by nausea, vomiting, great mental depression, and more or less complete bodily exhaustion. Vertigo is usually present. Although the statement may not be unquestioned, it seems best, in the light of present knowledge to classify seasickness as a neurosis of motion. Gihon[1] defines it as "primarily and essentially a marine vertigo."

Etiology.—The susceptibility to seasickness is wellnigh universal. Natural exemption exists in only from 3 to 5 per cent. of all travellers. Gihon[2] says: "While perhaps 5 in every 100 persons are practically immune, 25 per cent. will be but little sick, 60 per cent. a great deal so, though for a comparatively brief period, while the remaining 10 will be distressingly ill; 1 or 2 of these, if the weather be cold and stormy, passage long, and their prior condition bad, becoming critical." He found that out of an annual average enlistment roll in the United States navy only 5 per cent. became seasick, and during fifteen years only 12 men were seriously enough affected to require hospital care. The average duration of the seasickness was but three days.

Motion.—Seasickness, as implied in the name, is brought about essentially by the unwonted motions imparted to the body when at sea, but an identical condition may arise in sensitive persons by similar motions produced in other ways. Thus "car-sickness" is very common in this country, especially among women and children of from four to twelve years of age. Car-sickness is commonly produced by riding backward in an ill-ventilated railway car which is going at rapid rate around sharp curves. It is accentuated by the use of the eyes, either in viewing stationary objects out of the window which seem to be moving or by fixing the eyes upon a printed page. In some instances car-sickness becomes so serious an affliction as to practically interdict the taking of long journeys by rail. Those who have car-sickness are usually also made ill at sea.

Symptoms akin to those of seasickness are experienced by many persons who travel repeatedly in the modern quickly-falling "express" elevators, although here the exposure is but momentary and the symptoms may not proceed beyond vertigo or nausea.

Other forms of motion, such as are derived from a child's "seesaw"

[1] *Twentieth Century Pract. of Med.*, vol. iii. p. 176.

[2] *Loc. cit.*

or "swing," whirling in a "merry-go-round," etc., are very apt to produce seasickness in adults. The up-and-down, rapid motion of toboggan sliding may give rise to a "sinking" sensation, vertigo, nausea, etc. Rapid whirling of the body in a constant direction will do the same.

Admitting some unwonted form of motion as the primary cause of seasickness in the great majority of cases, it remains to discover, if possible, in what manner the motion acts upon the body to produce the peculiar complex of symptoms.

Constant effort to maintain equilibrium when the body is suddenly thrown out of balance becomes extremely wearisome, and the exaggerated muscular tonicity which results is probably not without some influence, although it by no means explains all the phenomena of seasickness. In severe cases the repeated acts of vomiting produce complete lack of tone in all the muscles, and yet the vomiting may continue indefinitely thereafter. It will be recalled that before the days of anæsthesia emetics were given prior to surgical operations to produce muscular relaxation.

Van Valzah[1] believes in a mechanical hyperæmia of the nerve centres, due to exaggerated tonicity of the muscles used in maintaining equilibrium, which empties the intramuscular veins. In opposition to this view is the fact that in tetanus, tetany, hydrophobia, and other forms of spasm of the skeletal muscles, which is often much greater than in seasickness, muscular tension does not produce the condition under discussion. This author also differentiates an anæmic variety of seasickness, and argues that the whole process is primarily a disturbed sense of equilibrium and of position in space, causing vertigo, consecutive cerebral anæmia, and cerebro-spinal irritability, with the attendant gastric and mental symptoms.

Observations of the facts just stated in regard to the influence of body motion in producing seasickness have received various other explanations. An old theory, which merits less serious discussion, is that the fluid in the semicircular canals of the ears is made to press unequally upon its boundaries, very much as water is thrown against the sides of its containing cup if the latter be made to swing or revolve rapidly. In this manner the sense of equilibrium is disturbed, space perceptions are distorted, and sympathetic vertigo and gastric disorder result (Bénard[2]).

J. A. Irwin[3] also in 1881 wrote in favor of the influence of the semicircular canals, and in a subsequent paper,[4] while admitting the influence of visual impressions, he refers again to kinesia "as a disturbance of the special function of equilibration located mainly within the semicircular canals."

Another theory is that of Dastre and Pampoukis,[5] who made an interesting series of experiments several years ago to determine the effect upon the respiration of swinging motions. When dogs and rabbits are swung rhythmically their thoracic respiratory movements follow the rhythm of the swing, but become out of rhythm with the abdominal movements. Moreover, the abdominal viscera are thrown alternately

[1] *New York Med. Journ.*, Aug. 13, 1892. [2] *Étude sur le Mal de Mer*, Paris, 1879.
[3] *Lancet*, Nov., 1881.
[4] "The Literature of Seasickness," *N. Y. Med. Record*, May 20, 1893.
[5] *Annales de Physiologie, norm. et pathol.*, v. ii. p. 277.

against the abdominal walls and the diaphragm. The pressure is offset to some extent by a reflex partial contraction of the opposing muscles. The authors suggest that the visceral pressure stimulates the Pacinian bodies of the mesentery, thereby exciting not only the modifications in respiration, but the sensation of nausea and the vomiting.

Brunton [1] has shown how closely the respiratory and vomiting centres are associated. Hypervenosity of the respiratory centre often produces nausea and vomiting, and the favorable influence upon seasick patients of getting them into the fresh air and compelling them to take repeated deep inhalations is an every-day experience.

In cases where the stomach is filled in part with fluids and in part with gases of indigestion the motions described will change the position of fluid in the stomach in such manner as possibly to irritate the gastric mucosa and cause vomiting. Yet seasickness is equally common when the stomach is wholly empty. Currie attributed seasickness to the pressure caused by the intestines and other abdominal viscera against the diaphragm. Gilchrist found the cause in similar oscillations of the brain, which excite central nerve irritation that radiates outward along peripheral nerves; and Skinner [2] adds to this view the mechanical displacement of the viscera and of the blood in its vascular distribution. The result is a neurosis of the sympathetic nerves of the heart and vessels, ending in general vascular paresis and consequent reduced arterial pressure.

An explanation which, to me at least, seems most plausible in accounting for many cases is that the liver, being by far the heaviest viscus, and being not so firmly attached but that it is capable of an up-and-down movement of from one to three inches in active breathing, acquires a different momentum from the rest of the body or from that of its immediate environment, and by this momentum it tends to slightly continue its motion beyond that of other organs. In this manner it is led to press upon or drag or irritate the solar and splanchnic plexuses, from which, by reflex sympathetic nerve mechanisms, the various symptoms of seasickness are initiated. In support of this hypothesis is the fact that in almost all cases it is the long downward "swoop" of the body, as in a deep roll at sea, the downward rush of an elevator, the descent of the swing, which produces the worst sensations. I know of no better illustration than the feeling produced in many by the rapid descent of an elevator which is suddenly checked and made to return to the floor above. One can almost imagine feeling the liver continuing its descent.

Martin and Howell note, in confirmation of the theory of visceral displacement, that recovery from seasickness is often accompanied by tenseness of the abdominal wall, caused by the endeavor of the muscles to restrict the free play of the viscera.

The conception of position in space is largely dependent upon the experience of muscular co-ordination and of the muscle sense. Disturbance of this faculty is believed by many to be causative of seasickness (Moussoir [3]).

Jolting motions, such as riding over a very rough road in a vehicle

[1] *Pharmacology and Therapeutics*, p. 323.
[2] "Recent Studies in Naupathia or Seasickness." *N. Y. Med. Journ.*, 1893.
[3] *Le Mal de Mer et le Sens de l'Espace*, Paris, 1889.

without springs; jarring motions, such as those produced by earthquake shocks; and swaying motions, such as are experienced in camel-riding,—are all common sources of seasickness in the hypersensitive. A projected French army camel corps was abandoned on account of the seasickness experienced by the soldiers.

As a general rule, in those who are affected by motion the resulting seasickness is proportional to the extent and, up to a certain point, the duration of the motion, but those who have become accustomed to one form of motion at sea, such as pitching, may be again made ill by the totally different form of rolling, etc.; and in many persons the jarring of the screw of a steamer is an additional factor. Seafaring men who have not been seasick for years may suddenly become so in an unusually severe storm or when some unwonted circumstance combines with exaggerated ship motion to unbalance them.

Visual Disturbances.—Many of the well-known normal optical phenomena have been cited as factors in producing seasickness. The visual impression of instability of objects sliding and moving out of place, the persistence of motor images upon the retina, and their subsequent overlapping the field of stationary objects, are all believed to contribute in many cases to the sickness. The late Graily Hewitt[1] attributed all seasickness to primary visual disturbances. It cannot be said, however, that such distortions of accustomed visual impressions are always provocative of seasickness, for viewing the undulations of the sea from the shore does not excite them. In the case of car-sickness it is the body which is moving while the objects external to the car are mainly stationary. In *mal de mer*, however, not only the body, but all external objects, seem to be moving, and the psychic effect is undoubtedly to intensify the feeling of insecurity of the body by seeing familiar objects in distorted positions.

The blind are often seasick, and attempts to keep patients who are apt to be very sick blindfolded at the commencement of a voyage, in order to abolish visual confusion, have not proved satisfactory.

In the article upon "Gastric Neuroses" in this *System*[2] Stockton and Jones state that "one of the most prolific causes of functional gastric disturbances is eye-strain, and almost any neurosis may be induced by it," but near-sightedness and other abnormalities of vision do not influence the general phenomena of seasickness one way or the other, and both men and animals confined below deck, and away from all moving objects and the sight of the sea, suffer alike from the malady. It may be equally severe in the blackest night or in the glare of the sun.

Psychic Influences.—In persons of highly sensitive nervous organization the over-stimulation or fatigue of any special sense is sometimes provocative of vomiting. High-pitched, rasping noises, flashes of light, disgusting sights, stenches, etc. may all excite it. In many persons an attack of seasickness will be precipitated at sea by the smell of food, by even the thought or suggestion of it, as in reading a ménu, etc., or by the sight or sound of others vomiting. In persons who are always severely ill at sea the mere recollection of past experience may develop seasickness, although the vessel be moored to the wharf.

[1] *Brit. Med. Journ.*, 1892. [2] Vol. III. p. 111.

Martialis reports the case of a woman made seasick by watching a swaying lamp, and Jobard mentioned the occurrence of the malady in a woman while looking at a vivid painting of the sea. Rosse refers to seasickness caused by the sound of a boatswain's whistle, and Gihon mentions a case caused by reading exceedingly vivid nautical pictures, and one of a naval officer made seasick by receiving orders to return to sea; but such extreme examples belong rather to the domain of hysteria, and are scarcely to be admitted as evidence in explaining the sufficiently manifold phenomena of seasickness.

Even those who have spent their lives as seafaring men may from time to time suffer from seasickness. Lord Nelson was a familiar example of this, and unusual causes may from time to time affect such persons, as, for example, the case of Gihon,[1] who relates how after twenty years of immunity from seasickness while at sea as a naval officer, he became seasick while eating on a vessel which was malodorous from a cargo of bone phosphate.

Strong mental emotion, other than that generated through the simple causes above recited, is often said to be responsible for seasickness. Extreme depression or worry or despondency may induce it, but the influence of dread of the sea and of terror is much exaggerated. The latter, in fact, is often curative, as, for example, the fear generated in the face of shipwreck or fire at sea.

Overeating and -drinking prove fruitful sources of seasickness, whether indulged in before going on board the vessel or at any time during the voyage. The strong, pure sea air greatly stimulates the appetite, and many persons eat and drink twice as much at sea as they do upon land. This, combined with indolence, produces indigestion and renders the stomach much more liable to nausea from slight causes.

Tobacco, even in those habituated to its use, may excite nausea by disturbing still more the equilibrium of an unbalanced nervous system.

Foul air, whether simply malodorous or distinctly noxious, has a very potent influence.

Constipation is a strong predisposing factor. I have seen many cases of persons who are not at all sick at sea for the first three or four days, but who suddenly become so after prolonged constipation, which results in part from the sea air; in part from the overeating which the bracing air invites by stimulating the appetite; in part also from lack of exercise. On a long voyage repeated occurrence of constipation is apt to be followed by renewed attacks of seasickness.

Age.—Infants in arms and children under three or four years of age are usually free from sickness at sea. It should be remembered that rocking motions are chiefly used in lulling infants to sleep, and young children are not subjected to many of the causes, like overfeeding, psychic influences, etc., which disturb adults, and they are much more accustomed to disturbances of equilibrium—bending, stooping, rolling, etc.—in their play.

At the opposite extreme of life seasickness becomes less common and severe, fortunately for those whose bloodvessels are highly atheromatous.

Blood-pressure.—The theory that irregularities in barometric pressure

[1] *Twentieth Century Pract. of Med.*, vol. iii. p. 184.

caused by wave movements react upon the expansion of the larger bloodvessels is clever, but improbable, and is ably controverted by Gihon [1] and Brewer. Moreover, aëronauts are subjected to much greater sudden variations in atmospheric pressure without the occurrence of nausea.

Idiosyncrasies too numerous to mention play an important rôle. One of the most confirmed dyspeptics I know, a gentleman past seventy years of age, eats heartily and with impunity almost everything set before him at sea. Many persons can eat and apparently digest food at sea, such as welsh rarebits, pastry, nuts, etc., which they are never able to take at home.

Some persons, who are never affected in small yachts, are always sick on large steamers, possibly because of the difference in motion and the reassuring sight of land obtainable from the smaller craft. But others have exactly the opposite experience, and conditions which have repeatedly caused seasickness may suddenly fail to do so without known cause.

Conclusion.—In concluding the discussion of the etiology of seasickness the following propositions seem justified:

1. Seasickness is not explained upon any single theory.

2. The majority of cases are primarily due to disturbances of equilibrium of the viscera, chiefly the liver, thereby causing reflex nervous irritation.

3. Other cases originate in optical impressions from seeing moving objects in unwonted positions, which through direct reflex action or fatigue of the visual apparatus give rise to the symptoms.

4. Muscular fatigue from increased muscular tension (tone) in the efforts to preserve equilibrium, or from unconscious effort to adapt the respiratory rhythm to the rhythm of the unaccustomed motion, must be reckoned as a cause of seasickness in some cases.

5. Contributory factors of great importance are overeating and -drinking, smoking, constipation, foul air, especially with bad odors, many psychic impressions, and idiosyncrasies.

Obviously, any or all of these factors may operate in combination in the same individual.

Symptoms.—The typical symptoms which belong to the malady seasickness are: Nausea and vomiting, nervousness, malaise, headache, prostration, vertigo, and syncope. In prolonged cases other more serious symptoms supervene, such as protracted constipation, hæmatemesis, extreme emaciation and collapse, involuntary passage of urine and fæces, or anuria. Exceptionally, convulsions have occurred, and sugar has been found in the urine (Rosse).

Nausea and Vomiting.—Clinically, seasickness is manifested in a variety of ways. There are those who, without preceding nausea, vomit a few times and are well again. Such are usually robust or plethoric subjects. Others, less fortunate, are incessantly nauseated, and have all the symptoms except vomiting, which they endeavor to induce for relief. Still others—and these are usually, though by no means always, neurasthenic subjects—become so violently ill with both incessant nausea and vomiting as to be utterly prostrated. Such attacks often begin at once on going to sea, but they may come on more gradually,

[1] *Loc. cit.*

with increasing severity, after several days of immunity. Anorexia and malaise, sometimes chilliness, a foul tongue, constipation, slight dizziness, and irritability are premonitory symptoms which are often succeeded by increasing nausea, retching, and vomiting. At first the vomiting causes prompt relief, and in the mildest cases this is permanent, but much oftener the symptoms return in half an hour, and the whole process is repeated again and again until the patient no longer recuperates between the acts of emesis. At first strong mental efforts to ward off the vomiting may succeed, but in such cases a violent fit of vomiting may surprise by its suddenness even the patient himself, who without premonition cascades over the first thing at hand. Salivation is excited, and the taste in the mouth has the bitterness of gall.

Protracted vomiting is both distressing and dangerous. The abdominal muscles become tender and sore from repeated strain. I have seen hæmatemesis occur on the second day from intense congestion of the stomach and violent efforts at retching. In severe cases bile is ejected, and the contents of the small intestine have been found in the vomitus. The nausea and vomiting prevent sleep, and, as no food is retained and often no fluid, emaciation may become extreme. The straining efforts may cause rupture of bloodvessels which happen to be already weakened by disease. The repeated emptying of the stomach results in a considerable loss of fluid which cannot be replaced, and hence thirst is complained of, though the patient may dread to swallow that which is sure to be promptly ejected again. A diminution of the urine from a similar reason is noted in extreme cases.

Nervous and Mental Symptoms.—Intense, racking headache, usually frontal, "light-headedness," mental unrest, apathy, and extreme depression of spirits and a desire to be alone are early symptoms. A feeling of dulness and drowsiness is also common, especially among those who do not vomit promptly. Light customary sounds and customary odors, like those of food cooking or tobacco, become alike offensive. The patient grows irritable and morose. As nausea or vomiting continues, mental hebetude succeeds the condition of irritability, and this is attributed to the cerebral anæmia succeeding hyperæmia. Hopeless despondency ensues, the patient utterly disregards personal appearance and habits of cleanliness, and says he longs to die, although suicide has almost never been attempted from this cause. In extreme cases, Gihon[1] writes, "mothers lose every sentiment of maternal affection, and modest women all sense of shame, exposing themselves with the most reckless abandon; and I know of instances where they have submitted to sexual approaches for which they afterward suffered the keenest remorse." Surely, no other malady may so strikingly obliterate all moral sense by a few hours of intense disgust with life; and another curious feature is that the mental symptoms depart as soon as there is decided improvement in the physical condition, and often disappear even more suddenly than they began. Reynolds[2] reported three cases of temporary insanity, and Rosse another, caused by seasickness.

The utter demoralization produced by the attack is very characteristic, and has been made the butt of much innocent satire. For the time

[1] *Loc. cit.*, p. 180.

[2] *Lancet*, 1884, i. 1161.

being, friends, home, everything, is forgotten in the wretchedness which makes life itself seem insupportable. This is particularly true of those cases in which nausea is unrelieved by vomiting or predominates, and it is highly typical of seasickness. Other forms of repeated vomiting, such as may accompany cardiac or renal disease, gastric ulcer, and the like, are often free from the extreme mental depression of true *mal de mer*. The explanation is in part furnished by the fact that with a long voyage in prospect there seems no possibility of abatement of the misery, and the mind is disturbed with the future as well as the present. The sight of land often has an immediate curative effect, although the ship's motion may be increasing.

Mental diversion, anything almost which arrests the attention from the absorbing contemplation of the nausea and misery, may be curative. Thus I have seen the sudden stoppage of the engines of a steamship in mid-ocean bring many to their feet cured (who had previously felt too ill to stir), in the fear that a collision or other serious accident was impending.

Vertigo and Syncope.—Modern writers lay much stress upon vertigo as essential in seasickness, and as often preceding and causing the vomiting. While this is true of many cases, it is not invariably so, and the vertigo when present may be merely the sequel of weakness from vomiting and the attendant vascular depression. In mild cases it is often not true vertigo or dizziness which is present, but an intense feeling located in the epigastrium which the patient describes as one of "hollowness," "sinking," "faintness," etc. There may be craving for food or drink, which is instantly vomited, or the mere sight of food may excite protracted emesis.

True syncope sometimes occurs, though this is less common than vertigo in early or mild cases, although common enough later from physical exhaustion. The pulse becomes feeble, rapid, sometimes intermittent; the heart sounds are weak; the vessels are relaxed, and the extremities are cold and bluish, perhaps bathed in clammy perspiration. The voice is feeble, and the features are strikingly pallid; no fever is present. The feebleness of the circulation, combined with vertigo, the loss of equilibration, and the inability to take food, all combine to produce muscular exhaustion. The gait, at first tottering, soon gives way completely, and the patient sinks down anywhere exhausted, often helplessly lying in a pool of his own ejecta.

Sequelæ.—The vast majority of patients are cured as soon as they set foot on land, and proceed at once to gratify a ravenous appetite and forget all their previous miseries. In some cases, however, more especially among women and victims of neurasthenia, certain symptoms persist after landing. It is very common for some instability in gait or confusion in walking, as if the ground were rolling, to last for a few hours, and the headache and vertigo may last for days or in exceptional cases for weeks after landing. Rarely vomiting continues on shore, and patients may become more and more prostrated, and finally die from asthenia. Fortunately, but very few cases of this kind have been recorded. In an otherwise healthy woman of thirty years whom I have lately seen vomiting persisted for three weeks after a brief European voyage, and it was a full month before the vertigo and prostration were completely over-

come. On an exceptionally long voyage to Australia a case has been known to last for six months.

Dangers of a Sea-voyage.—Fatal cases are not unknown. Fordyce Barker reported 3 such, 1 patient having died from inanition two days after landing, and the 2 others died from exhaustion. Andrews reported 4 fatal cases, Gonin 2, 1 of which was caused by hæmatemesis, and there have been a few others.

Victims of certain maladies ought on no account to hazard a sea voyage if there be any likelihood of suffering from severe vomiting. Foremost among such diseases are those in which the effort and straining of violent retching are liable to produce vascular rupture. Aneurysm, advanced arterio-sclerosis or atheroma (from danger of cerebral hemorrhage or cardiac dilatation), hæmoptysis, hæmatemesis, or gastric ulcer, rupture, advanced cardiac valvular disease,—are all imperative contra-indications. Epileptics are said to be made worse by seasickness. The vomiting of early pregnancy is, strangely enough, sometimes improved by it, and abortion is rarely induced.

This is not the place to discuss the advantages of a sea voyage, but it may be appropriately stated that it is a peculiar fallacy that seasickness is beneficial in "biliousness" and other complaints. The sea air, rest, change of scene, and freedom from responsibilities and cares are the real beneficial agents, and it is not necessary to go through a siege of nausea and vomiting to profit by them to the maximum extent.

TREATMENT.—*Prophylaxis.*—Many attempts to avoid seasickness have proved complete failures. Such are swinging staterooms, specially constructed vessels, going at once to bed and lying down before the vessel leaves the dock, remaining blindfolded, the practising of swinging motions before sailing, etc. Many drugs have been recommended, like the bromides, to be taken for a week before sailing, but they usually serve to disorder digestion and make matters worse. I have seen patients who had dosed themselves with patent bromide preparations until they induced a state of bromism which added greatly to their illness, but occasionally, when properly prescribed, the bromides are not without prophylactic value. They help some people very much at times, fail in the same persons at other times, and fail completely in the majority of cases. Dutton[1] employs a mixture of bromide of ammonium with spirits of chloroform and aromatic spirits of ammonia, to be taken for three or four days before sailing, with the object of calming the central nervous system.

Chlorobrom, recommended by Charteris,[2] is a palatable compound containing ½ drachm each of chloramid and potassium bromide to the ounce.

There are, however, certain simple precautions which are helpful, if not infallible, to many. Such are—avoiding fatigue and worry before starting upon the voyage; eating the simplest food for several days previously; lying down, but keeping in the fresh air on board ship, and naturally in a part of the vessel free from odors and excessive motion or jarring; avoiding looking at the water or straining the eyes in any manner; eating often at sea and but little at a time, and avoiding taking much fluid at one time. The wearing of belts and abdominal lacing

[1] *Seasickness, Cause, Treatment, etc.*, Plymouth, 1890. [2] *Lancet*, Mar. 6, 1892.

may help some persons by supporting the viscera, but as a rule they interfere with free respiratory movements, and loose clothing is much better. Faradization and massage of the stomach are alike useless, and rank with hypnotism as possible mind-cures of very uncertain effect.

Medicinal Treatment.—As a rule, the less medicine taken the better, although the remedies which have been lauded as specifics are too numerous for mention. In general, the treatment should be stimulating and supporting to the circulation, rather than depressing to the nervous system, and the simplest sedatives, if any, should be used. Such remedies as creasote, chloroform, ether, etc. are commonly worse than useless, and phenacetine and antipyrine are too depressing. Oxalate of cerium 3 gr., bismuth 30 gr., codeine $\frac{1}{2}$ gr., trional 15 gr., and moderate doses of the bromide of sodium are the most useful sedatives for relief of vomiting or headache. Danvers [1] strongly recommends an effervescing hydrobromate of caffeine, and Dujardin-Beaumetz the combined use of caffeine with sodium salicylate. The citrate of caffeine in doses of 1–2 gr. is a useful remedy for both the nervous and vascular systems. A combination of drugs much in vogue of late consists of atropine gr. $\frac{1}{150}$, hydrobromate of hyoscine gr. $\frac{1}{150}$, nitrate of strychnine gr. $\frac{1}{60}$. These remedies may be given hypodermically two or three times a day, but they are far too strong for indiscriminate use by patients, and I have known instances of dangerous poisoning from the combination. Strychnine and caffeine are the best vascular tonics for use in seasickness.

W. W. Skinner [2] believes that seasickness follows lowered arterial tension, being a neurosis of the sympathetic nervous system, and accordingly recommends for its treatment,

℞.	Atropinæ sulphatis,	gr. $\frac{1}{150}$ – $\frac{1}{64}$;
	Strychninæ sulphatis,	gr. $\frac{1}{64}$;
	Aquæ menthæ destillatæ,	♏xv.—M.

It is highly important to thoroughly evacuate the bowels. For this purpose mild salines are usually best, and are more apt to be retained. Such are the Rubinat water, less objectionable in taste than many bitter waters, Seidlitz powders, and effervescing citrate of magnesia, which is usually retained.

If nausea is constant and vomiting does not follow, it is well to unload the stomach of its fermenting food by a simple emetic, such as salt in lukewarm water, and violent non-productive retching is much relieved by drinking sufficient hot water to give the stomach something to contract upon.

External applications are useful in the form of hot-water bags or prepared mustard leaves to the epigastrium, and hot-water bottles to the feet. Gihon finds alcohol sponge baths useful, and regards the vapor of alcohol in the patient's stateroom as highly sedative.

Dietetic Treatment.—No special dietetic system is applicable to any large number of cases, and foods which may be suited to a patient in one voyage may fail to be retained by another. Patients who anticipate severe seasickness do well to live simply for several days before embarking, avoiding all richly cooked food, desserts, sweets, etc. Since it is often

[1] *Lancet*, 1892.

[2] *New York Med. Journ.*, Dec. 8, 1893.

difficult to obtain palatable invalid foods on crowded steamers, they will do well to provide themselves with some of the simplest forms of nourishment, to be used when vomiting commences. Such, for example, are—pasteurized milk, which will keep fresh for six or seven days, and which may be pancreatinized at time of using, or diluted with equal parts of Vichy or lime water, or taken with 10 grains of sodium bicarbonate and 3 grains of cerium oxalate to the tumblerful; the Swiss milk condensed and canned without sugar will keep longer and is very serviceable; koumyss, matzoon, or matzol; clam broth, which may be obtained bottled, and which seems almost a specific for nausea in some cases; one or two of the artificial foods, such as malted milk or the meat preparations, such as somatose, somatose biscuits, carnogen, Mosquera's beef meal, etc.

Some patients crave sour or pungent articles to relieve the constant bad taste in the mouth; as, for example, plain pickles, dried ginger, sliced lemon, lime-juice tablets, all which are acceptable. The juice of one or two sour oranges or lemons squeezed into iced Vichy or Apollinaris water, with a little sugar or saccharine and a pinch of bicarbonate of soda, makes a most refreshing and soothing beverage. Strong black coffee is excellent, and is beneficial for its diuretic action and stimulating effect upon the circulation. The extract of coffee may be carried if there is uncertainty of obtaining good coffee at sea.

The meat extracts, such as Liebig's extract, Johnson's fluid beef, etc., while not containing much nutriment, are certainly stimulating, and are to be recommended in some cases. A form of beef tea is now sold at the grocer's condensed in capsules which may be dissolved in a cupful of hot water.

It is, however, not well to ingest too much soup or broth at once, for if there be much motion at sea the weight of the fluid, tipping about in the stomach as the vessel rolls and pitches, may easily accentuate the nausea. For this reason it is best to take all fluid food in quantities not exceeding two or three tablespoonfuls at a time, if need be, at half hourly intervals.

It often happens that patients retain dry solid food much better than fluids, and they should try dry soda crackers, Huntley and Palmer wafers, lemon-gingersnaps, zwieback, and chipped smoked beef which may be obtained in boxes at the grocer's before sailing.

Many patients crave cracked ice, but it should be taken sparingly, or else, becoming lukewarm in the stomach, it excites nausea.

When the stomach rejects even the simple articles above enumerated, or if hæmatemesis occurs and the patient's strength is failing, recourse must be had to rectal feeding and stimulation. For this purpose pancreatinized milk, meat extracts, and brandy should be used.

If alcoholic stimulants are indicated, the best are very dry cold champagne, weak brandy and soda, or sparkling Moselle. As a rule, it is not desirable to take much alcohol in any form, and no non-diluted spirits should be drunk.

PARALYSIS AGITANS, AND TREMORS IN GENERAL.

By FREDERICK PETERSON, M. D.

PARALYSIS AGITANS.

Synonyms.—Shaking palsy; Parkinson's disease; Paralysis tremens, tremula, palpitans, jactitans; Rigor tremens (Peterson); Ger. Schüttelähmung, Schüttelkrampf; Fr. Paralysie agitante.

Definition.—Shaking palsy, first adequately described by Parkinson in 1817, is a chronic neurosis characterized by rhythmical tremor, considerable muscular rigidity, and some weakness, but without actual paralysis. Tremor and rigidity are the cardinal symptoms of the disease. The latter gives rise to the peculiar attitude and often singular gait (festination, retropulsion, etc.). The muscular weakness is more apparent than real, and has frequently been termed pseudo-paralysis by various authors. The name paralysis agitans, therefore, though now too well established by usage to be changed, is not wholly satisfactory, and rigor tremens would better define the syndrome.

Etiology.—The disease is essentially one of *late life*, though a number of cases have been reported as having their onset between the ages of seventeen and thirty years. In by far the greatest number the disorder begins between the ages of forty and sixty, but particularly in the latter half of this period. A diminishing number show an onset between sixty and seventy years of age, and a very few have their beginning in the succeeding decade. The following tabulated collection of cases, observed by myself at the Vanderbilt Clinic and in private practice, well illustrates the relation of age to the disease, as well as that of sex:

Age at onset.	Males.	Females.	Total.
30 to 40	4	3	7
40 to 50	18	6	24
50 to 60	30	23	53
60 to 70	19	7	26
70 to 80	2	3	5
Total	73	42	115

As regards *sex*, it will be seen from the above that men are almost twice as frequently affected as women.

It is a moot question whether *heredity* plays any particularly import-

ant part in the development of the disorder. Direct heredity is certainly extremely rare, and a neuropathic basis can be determined in less than 10 per cent. I have seen but one case of direct heredity, a father and son, in both of whom the disease began between the ages of thirty and forty. In the case of a woman in whom the disease had progressed to such an extreme degree that there was irremediable contractures of both hands and both feet, and in whom the general rigidity was so great that locomotion was impossible and speech very difficult, a brother became affected with shaking palsy. This seemed to me not to show an hereditary nature, but rather a condition analogous to communicated insanity and imitative hysteria major and imitative chorea. There had always been constant anxiety on the brother's part lest he should be afflicted like his sister, and he was prone to watch continually for the appearance of similar symptoms in himself. Perhaps this state of expectant attention actually led to the development of his own malady. Some support is given to this idea by the cases of a husband and wife under my observation some years ago. The wife, aged fifty-seven, presented the typical symptoms of a shaking palsy of four years' standing. The husband, ten years older, developed while he was under my treatment a tremor of his right hand precisely like his wife's. He had also been under the fear of being affected by the disease. Gowers mentions one case in which father and uncle also had the disorder, one where the sister was affected, and three where one parent presented the same symptoms.

The influence of *occupation* is assuredly not great. Undoubtedly, the majority of cases are from the common walks of life. If occupation has any distinct relation to the malady, it is probably such pursuits as subject the individual to undue exposure to extremes of heat, cold, and dampness. Thus, among my cases were 4 coachmen, 2 night-watchmen, 3 engineers, 1 iceman, 1 iron-founder, 1 messenger, and 9 day-laborers. Two men developed shaking palsy almost immediately after prolonged exposure in a great snowstorm.

Probably the most frequent exciting causes are mental stress, trauma, and physical illness. Among the *mental strains* which give rise to the disease are worry, anxiety, grief, excitement, and sudden fear. In the case of an illicit distiller the malady appeared soon after his discovery, trial, and the confiscation of his property. In several cases domestic unhappiness was assigned as a cause. One woman developed it during an anxious period of nursing her dying mother, one from seeing her son drown, and another during a period of worry over an inebriate son. In one man the disease began soon after great excitement over a religious discussion. Fright is not infrequently a direct precursor of the malady. The tremor of fear in these cases seems to persist and take on the character of paralysis agitans, and a noteworthy peculiarity is the localization often observed of the tremor in a particular limb when it is coincidently affected with the emotional shock. This is especially shown in cases where *trauma* and the emotional shock usually incident to sudden injury act together to induce the disease. It is usual for the tremor in cases of traumatic origin to begin in the injured member. Thus, a woman fell from a stepladder upon her right arm, and the disease began in the right arm. A coachman in a runaway was thrown from the carriage upon his left shoulder, and shaking palsy soon manifested itself

in his left arm. Falls upon the shoulder or arm are indeed so frequent as exciting causes as to be especially noteworthy. A woman sitting quietly at work was suddenly frightened by water flowing upon her left wrist from a faucet; the tremor of Parkinson's disease began immediately in the left hand (Gowers). Contused injury to the thigh has set up the disease in that limb, and dislocation of the jaw has induced the characteristic tremor in that part (Charcot). In one of my cases cellulitis in the right hand led to the tremor beginning there; in two cases injury to the fingers induced the disorder in that particular hand, and in one amputation of fingers of the left hand gave rise to paralysis agitans, beginning in the left hand. There is evidence that the mere concussion of severe falls or other accidents—and this has a medico-legal value—may so affect the nervous centres as to induce paralysis agitans. Among physical *diseases* which have in many instances immediately preceded the manifestation of the disorder are sciatica, fever and ague, articular rheumatism, grippe, gout, typhoid fever, pleurisy, dysentery, and remittent fever. Where the disease was local, as in the cases of sciatica, rheumatism, gout, and pleurisy, it is interesting to note that this has determined the localization of the tremor in the side or part affected. Other and rather doubtful exciting causes have been mentioned, such as muscular fatigue and sexual excess. Toxic tremors, such as those of lead, alcohol, tobacco, tea, and the tremor of neurasthenia, have apparently no tendency to induce this disease.

PATHOLOGY AND PATHOLOGICAL ANATOMY.—There has been considerable pathological study of this malady in late years by modern methods, and it must be confessed that no very definite results have as yet been reached. We must still assume that it is a functional disease, a species of neurosis in the aged, allied to chorea in the child. There are good reasons for believing the anatomical basis to be some disturbance in the motor cortical area at present unknown, but probably nutritional and degenerative in character. The frequent "hemiplegic" manner of onset is one argument in favor of the view that the disease is in the hemispheres. The idea entertained by some authors (Dana) that the seat of the lesion is lower down, in the spinal cord, medulla, and pons, would seem to be invalidated by such a fact as that an attack of hemiplegia puts an end to the tremor. The frequent sudden origin of the disease from mental shock or strain, such as fright, also lends support to the view of a cerebral cause. The position of the hands (analogous to that of hemiplegia, epilepsy, tetany), and the fact that other tremors (neurasthenia, chorea, fear) are surely cerebral in nature, are further arguments. Excellent pathological studies of paralysis agitans have been made by Dubief, Von Sass, Koller, Borgherini, Dana, and Ketscher. Ketscher's work is particularly valuable, as it is based upon the examination of the nervous systems of three cases compared with control studies of those of ten aged people. The changes found by Ketscher on microscopical investigation were mainly in the spinal cord, in two cases in the muscles, and in one in the peripheral nerves. In the nervous system of his cases he found indications of degeneration and atrophy, such as—in the cells of the brain and cord, pigmentation, indistinct outlines, absence of nuclei, granular degeneration, general increase of interstitial connective tissue and of the neuroglia of the brain and cord; in the vascular system, thickened

vessel walls, miliary aneurysms, small extravasations, pigment in the adventitia, dilated perivascular and pericellular lymph spaces, œdematous softening here and there in the neighborhood of these spaces; in the nerve fibres, swelling of axis cylinders, disappearance of nerve fibres; in the muscles, increase in the number of nuclei, loss of transverse striation, hyaline and fatty degeneration, disappearance of muscle fibres. Such changes are analogous to those which take place in old age, though they would seem to be probably somewhat more marked in Parkinson's disease. We are thus constrained to go back to the original hypothesis, that the disease must still be regarded as a neurosis with unknown anatomical basis, but occurring in individuals whose nervous systems are prematurely and markedly senile.

SYMPTOMS.—*Tremor* is the most important and usually earliest symptom of paralysis agitans, although, paradoxical as it may seem, a considerable number of cases have been recorded of true Parkinson's disease without any tremor whatever. Thus 10 such cases have been reported by Charcot, Berger, Wienskowitz, Buzzard, Hardy, Amidon, and Beevor, and I have observed myself 4, 1 of which I presented at the New York Neurological Society in 1895. Rigidity is, however, always present when the tremor is wanting.

The tremor usually begins in one extremity, nearly always a hand, and then spreads to the leg of the same side, then to the opposite hand, and finally to the opposite leg. It may sometimes begin in another part, such as the neck muscles or the shoulder, and the progress of the tremor is not always hemiplegic in character. In 102 of my cases, where the place of onset was carefully inquired into, the following was the result of the inquiry:

	Cases.
Tremor began in the left hand in	46
Tremor began in the right hand in	40
Tremor began in the left foot in	6
Tremor began in the right foot in	3
Tremor began in both hands (?) in	5
Tremor began in both feet (?) in	1
Tremor began in the head in	1
Total	102

The tremor may be so slight at first as to be noticeable only when the muscles are fatigued; later on it becomes constant, except during sleep and when momentarily arrested by any voluntary effort. Usually on waking from sleep there is no tremor for some little time. Rarely a certain amount of tremor may persist during sleep. The peculiarity that the tremor is going on when the limb is at rest distinguishes this from every other form of tremor, since the other forms are more or less "intentional" in character. Thus, the tremor of paralysis agitans ceases for a few seconds when the patient is asked to shake hands, and, even though the tremor may be extreme in degree, the patient is generally able to draw a perfectly straight line across a blackboard. Usually, after the momentary cessation on effort, the tremor is apt to redouble in intensity for a short time. The tremors of senility, multiple sclerosis, delirium tremens, and so on tend to grow more and more extensive the greater the effort of the patient to steady the part, as, for instance, in

attempting to draw a straight line. Very rarely cases are met with in which the tremor of paralysis agitans assumes something of an intentional character.

Charcot's statement that the head never takes part in the tremor, but is only moved by the movements of the contiguous upper extremities, is not borne out by the facts. Cases have been reported by Oppolzer, Clement, Jones, Westphal, Demange, Buzzard, and Huber where the head presented an intrinsic tremor. Gowers reported 8 of 35 cases as presenting tremor in the head; 26 of my 115 cases exhibited tremor of the head which depended upon tremor in the neck muscles. It is not uncommon for the disease to extend to the face muscles. There was marked facial tremor in 8 of my cases. The jaw muscles and tongue are occasionally affected, and very rarely the orbicularis palpebrarum.

The tremor consists of a regular oscillating and alternating contraction of opposing muscles, often varying greatly in the extent and rate of rhythm at different times, and even in different parts of the body of the same individual. Sometimes the tremor ceases completely for an hour or two daily, and in some cases there may be great diminution or increase for an indefinite period. In the hand the tremor usually is of such a character that it has been described as a "pill-rolling" movement. The rate of the tremor is ordinarily five per second. It is a kind of tremor that readily permits of being taken by a sphygmograph,[1] but it is much better studied by means of Ludwig's kymograph. The accompanying illustrations of the tremor of paralysis agitans were taken with a kymograph by the writer and published in the *New York Medical Journal*, March 10, 1894 (Figs. 66–70). The advantage of the kymograph is that rapid revolution of the drum may be made to magnify the waves, so that each may be studied in all its details, and the dicrotism of the waves in paralysis agitans be clearly brought out. Doubtless, all tremors are a modification of the rhythmic discharges of energy from the cortex, which take place at the rate of ten in a second. When there are fewer per second it is probably due to the fusion of two or three impulses. The rate per second of the tremor of Parkinson's disease, as determined by various authors, is well shown in the following table:

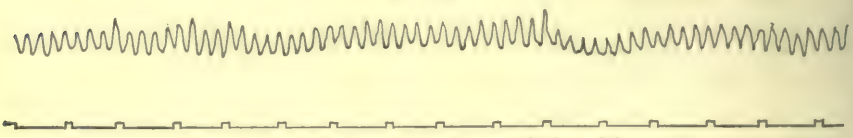
Author.	Publication.	Rate to the second in paralysis agitans.
Marie	*Contrib. à l'Étude*, etc.	5
Charcot	*Mal. du Système nerv.*	4–5
Ewald	*Berl. klin Woch.*, 1883, No. 32	5
Grashly	*Arch. für Psych.*, 1885	4.14–5.34
Huber	*Virchow's Arch.*, vol. 108, p. 45	3.43–5.57
Gowers	*Dis. of the Nerv. Syst.*, 1888, p. 1001	4.8–7
Wolfenden and Williams	*Brit. Med. Journ.*, May 19, 1888	5.1
Peterson	*Journ. of Nerv. and Ment. Dis.*, Feb., 1889.	3.7–5.6
Dana	*Medical News*, Dec. 17, 1892	3–6.1

In the accompanying kymographic tracings we see in Fig. 66 the tremor was taken with slow revolution of the drum from the thumb of a patient. It is similar to tracings made with a sphygmograph. Fig. 67

[1] See *Journal of Nervous and Mental Diseases*, February, 1889, article by the writer on the use of the Edwards' sphygmograph in making tracings of muscular tremor.

was also taken from the thumb, but in another patient where the tremor was very marked and the excursions wide. There was a remarkable

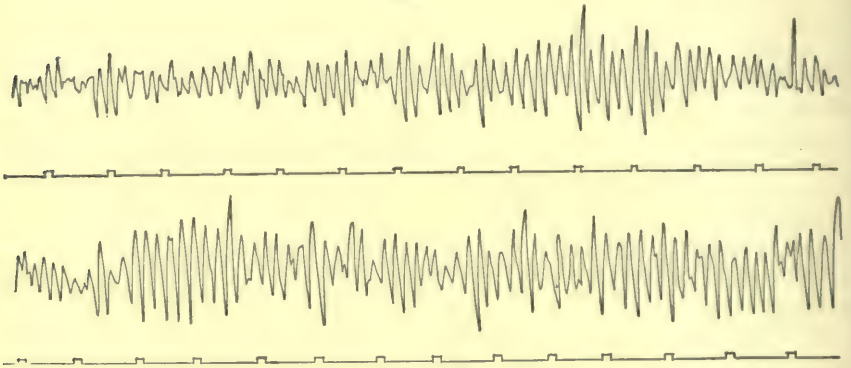
FIG. 66.



Tremor of paralysis agitans: slow revolution of drum.

feature in this tracing—viz. a manifest tendency of the waves to aggregate in groups of increasing and diminishing range about every four

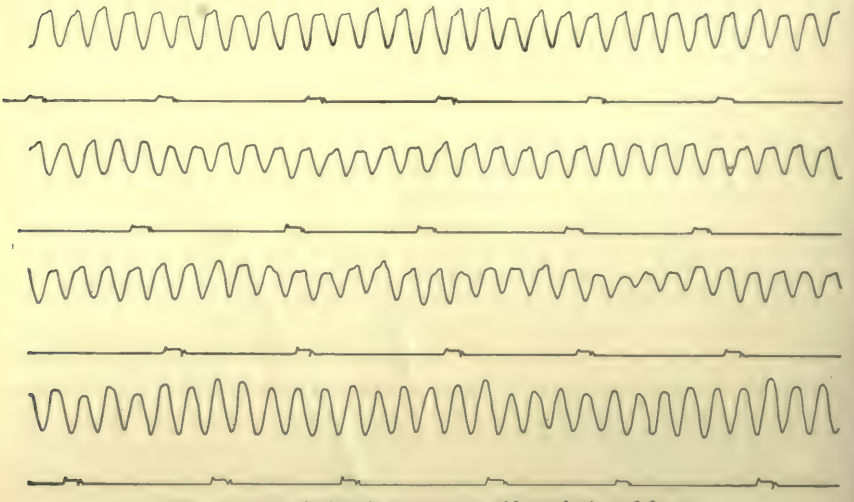
FIG. 67.



Tremor of paralysis agitans: slow revolution of drum; singular grouping of series of waves, each four seconds.

seconds. It seemed as if the innervation rhythms were invigorated every few seconds. This did not seem to be related to cardiac or respira-

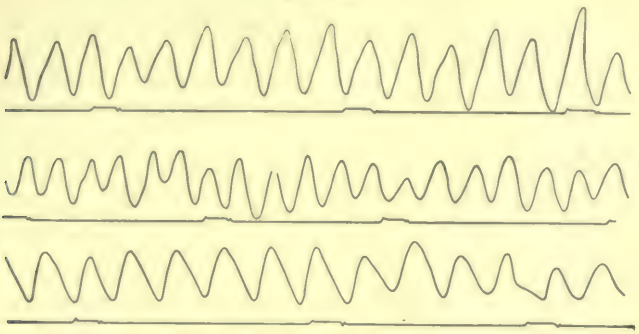
FIG. 68.



Tremor of paralysis agitans: more rapid revolution of drum.

tory influence, and may have been due to some unknown factor (such as nutritional rhythm in the cortical cells (?)). By a more rapid revolution of the drum we magnify the waves, as in the series of four tracings from the wrist illustrated in Fig. 68. In this the tendency to dicrotism becomes manifest, and the very exact rate of five per second is apparent. In Fig. 69 the tracing was taken from the ring finger in another case, and, though the drum revolved more rapidly, the wideness of the

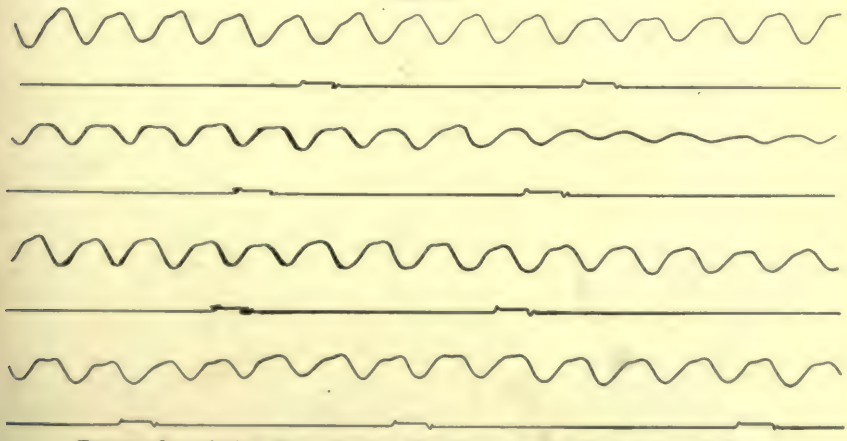
FIG. 69.



Tremor of paralysis agitans: still more rapid revolution of drum.

excursion hardly permitted of the development of dicrotism. By very rapid revolution of the drum of the kymograph, as in Fig. 70, where four tracings showing the movement of the extensors and flexors of the wrist are given, the waves are still further amplified, and the dicro-

FIG. 70.

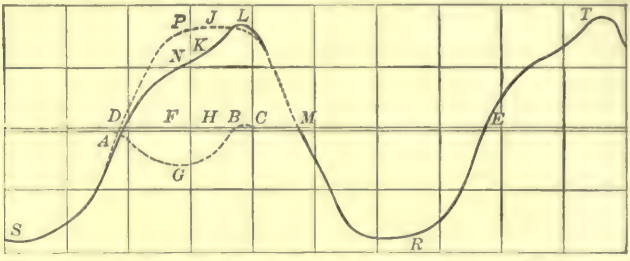


Tremor of paralysis agitans, showing dicrotism: very rapid revolution of drum.

tism and regularity become still more pronounced. Mr. Kennelly of the Edison Laboratory kindly made for me a geometrical analysis of a curve selected from the first of the tracings in Fig. 70, hoping to determine some feature that might prove of scientific value. If the waves were constantly the same, it would be possible to attribute a particular outline to a particular cause, and thus discover impulses and activities

not perceptible in the wave as a whole. In Fig. 71 we have a pair of these waves transferred by micrometer measurements to large scale paper. The curve *S D N K L M R E T* can be analyzed into components in its first half wave length. One of these is the second semi-wave, *M R E*, inverted, and the other a smaller opposite wave in

FIG. 71.



Geometrical analysis of paralysis agitans wavelet, taken from one of the waves in Fig. 70.

dotted line *A G B*. The opposite wave has two thirds of the main wave length and one third of the main wave amplitude, so that (neglecting as a possible discrepancy the wavelet *B C*, unaccounted for) there is a main train of waves, with a semi-train superimposed upon it, making a compound wave train. This analysis makes the dicrotism more manifest. In a series of waves, such as liquid waves through elastic tubes (the arterial pulse), such an analysis would prove of distinct value, because the current velocity, the elasticity, and the resulting series of curves are here nearly invariable. But where, as in paralysis agitans, variation is considerable, the analysis is difficult, and must be carried laboriously through a whole series in order to be productive of the best results.

Rigidity and muscular weakness tend to appear at the same time as the tremor, and in the same part, and are usually coincident in their onset. The rigor muscularum generally extends more rapidly and farther than the tremor, so that we often see it in the whole body when but one or two extremities take part in the tremor. As has already been stated, there are occasionally cases which present rigidity, but no tremor. In perhaps one sixth of the cases tremor and rigidity precede the tremor. Rigidity must be regarded as one of the cardinal symptoms of Parkinson's disease, for upon it depend the peculiar attitude, the characteristic gait, the expressionless face, and the monotonous voice. Rigidity is of varying degree, from slight stiffness of the muscles to almost utter helplessness, with contractures. This symptom was present to a marked degree in 85 of my 115 cases. It affects the muscles of the limbs, trunk, neck, and face. Even the tongue, palate, and laryngeal muscles may be implicated. Debove has reported cases with rigidity of the ocular muscles. The rigidity may be limited precisely to the parts affected with tremor, as in several cases with paralysis agitans of the hemiplegic type observed by myself, where the rigidity and tremor were so coincident as to implicate exactly one side of the body, even to one side of the mouth and tongue. Weakness is, as a rule, more apparent than real. The stiffness of the muscles so interferes

with motion as to present an appearance of loss of power. Undoubtedly, actual loss of power is sometimes observed, but it never amounts to paralysis.

The rigidity gives rise to a characteristic *attitude* in Parkinson's disease (Fig. 72). The postures in parts are determined by preponderance of rigidity in certain muscle groups, especially the flexors. The head leans forward and the shoulders are stooping. The knees are flexed to a greater or less degree and the thighs adducted. The elbows are bent, the wrists a little extended, while the hand tends usually to assume its ordinary position when at rest or the position when holding a pen. The face has the expressionless appearance of a mask. While a typical case will present such features as these, there are exceptional cases in which the attitude and postures of extremities are unusual. Thus, instead of stooping head and shoulders, there are instances of extension of the neck and spine; some distal joints of the fingers may be over-extended; sometimes only one or two fingers are contracted.

FIG. 72.

Case of paralysis agitans, showing the attitude, the position of the hands, and the facies (Gray).

Early in the disease, by effort, the characteristic attitude and postures may be overcome to a certain extent, but in advanced cases this power is lost, and often another cannot, by taking hold of the parts, restore them to a normal position. The contractions become frequently permanent and amount to actual *contractures*. I have seen ankylosis of all the joints of the hand and wrists in the characteristic position, and observed contractures in the toes and double talipes equino-varus in some cases. One case had such rigidity of the orbicularis oris that the mouth was motionless, open, and the patient was constantly drivelling. Protrusion of the tongue may be almost impossible.

It is not uncommon to note in old cases a certain amount of *muscular wasting*, but this may not attract attention unless the disorder is unilateral in type. One case of mine (Fig. 73) presented so striking an atrophy in the affected parts that I reported it in the *N. Y. Medical Journal*, Oct. 11, 1890. A man, aged sixty, had a typical Parkinson's disease of nine months' standing, confined to the left face, arm, and leg. The middle and ring fingers were strongly contracted into the palm. The atrophy involved especially the intrinsic muscles of the hand and adductors of the thigh, but was

also noteworthy in most of the other muscles. The faradic reaction in the atrophied muscles was normal. The measurements of the circumferences were as follows:

Showing the Amount of Wasting in a Case of Paralysis Agitans where the disease was limited to the left side.

Circumference of—	Right.	Left.	Difference.
Arms: 18 cm. below shoulders	25.5–23	22.5–20.5	2.5–3
Forearms: 15 cm. below elbows	21–19	18–16.5	2.5–3
Thighs: 15 cm. above patellæ	37–34.5	35.5–33	1.5
Legs: 15 cm. below patellæ	29–27	28–26	1

The illustration shows fairly well the wasting and contractures, but the rigidity was so great that the patient could not be photographed in the best position to show the defects.

FIG. 73.

Showing wasting of left arm and leg, with contractures of elbow, wrist, and fingers, in a case of paralysis agitans.

In many instances the rigidity gives rise to peculiarities in locomotion or movement of any kind. Patients find difficulty in turning over in

bed, in turning around when standing, in moving the head from one side to the other. Often several efforts have to be made in rising from a chair. The *gait* is frequently affected in a singular manner. In starting to walk the first step is taken with great effort and slowly, and the steps follow each other with increasing quickness. The steps are short and the patient seems to run. He is often unable to stop until he reaches some support. This phenomenon is known as *festination* or *propulsion*. Trousseau called this "running after the centre of gravity." Sometimes a similar tendency exists to run backward (*retropulsion*), and very rarely sidewise (*lateropulsion*). Patients who show the phenomenon of festination very commonly exhibit retropulsion also: 31 of my cases had the tendency to festination, 17 to both propulsion and retropulsion, and 2 to lateropulsion.

Benedikt years ago called attention to a *diminution in electrical irritability* in the affected extremities of old cases, and I have in several instances corroborated his observation with the faradic current.

As regards the *tendon reflexes*, they never present the marked exaggeration which we have as a symptom of organic lesion in the cortico-spinal segment of the motor tract. No significance attaches to their condition in this disease. All of my cases were examined as to the state of the deep reflexes in the upper and lower extremities, and the result in the 115 cases was that in 39 they were somewhat exaggerated or hypertypical, in 9 subnormal, and in all the others (67) normal.

The *voice* has frequently a high pitch and piping quality, and the *speech* in such cases shows the peculiarity of a sort of halting ejaculation of the words. Forty of my cases exhibited these characteristics of articulation and phonation. Doubtless the changes in speech and vocalization are due to a certain amount of rigidity in the muscles concerned with these functions. The changes may be summarized thus: 1, a condition of monotonia, as if there were difficulty in adjusting the vocal cords so as to vary the pitch of the voice; 2, high pitch and piping quality of tone, depending probably upon a minute degree of contracture in the crico-thyroid, posterior crico-arytenoid, and internal thyro-arytenoid muscles; 3, a species of what has been well termed festination in the speech, slowness of ejaculating the first word, followed by rapid pronunciation of the five or six succeeding words, then, again, a pause, with the ejaculation of a similar series. There is an analogy between this festination in speech and that of gait.

Among sensory and vasomotor symptoms are to be mentioned rheumatoid pains, thermal paræsthesia, paræsthesia of cold, and hyperidrosis. We will examine these in some detail. Patients often complain of miscellaneous paræsthesiæ, such as numbness, tingling, and pricking, and sometimes of actual *rheumatoid* or *neuralgic pains* in the extremities affected. Sixty of my cases presented paræsthetic symptoms of one kind or another. Probably only 10 per cent. suffer from pains. The pains vary much in character. Thus one man had shooting pains in his legs; another, a dull, aching pain in the three extremities affected; another, pain in both arms; another, burning pains in the parts implicated; a woman, rheumatic pain in the joints of the arm having the tremor.

An excessive *feeling of heat* over the whole body, or more rarely over limited areas, is not infrequently complained of by patients: 23 of my 115 cases called attention to this symptom. It is purely a subjective sensation, with a vasomotor basis, and there is no actual rise of the general temperature nor of the local temperature in the parts to which the thermal sensation is referred, as I and other observers have found by investigation.[1] Occasionally the localized feeling of heat may correspond to a patch of superficial redness, but this is rare. Usually there is nothing noteworthy in the appearance of the subjectively warm areas. Sometimes the sensation is so excessive as to be a source of the greatest discomfort, so that only the thinnest clothing can be borne, and warm weather adds to the misery of such cases. Not infrequently the heat sensation is limited to the affected limb or limbs, and it may be a very early symptom, preceding both rigidity and tremor.

A *feeling of cold* instead of heat is sometimes complained of, as in 15 of my cases; but this subjective sensation of cold is not accompanied by any alteration of temperature, nor necessarily even by any actual coldness of the extremities to touch. The chilly sensation may be general or local, and may alternate with that of heat.

As a rule, *hyperidrosis*, when it is observed, exists in association with thermal paræsthesia. This symptom was noted in 23 of my cases. It may be general in its distribution or limited to an affected extremity or side, and often the perspiration is very profuse. Undoubtedly, it has its origin in vasomotor disturbance, which sometimes makes itself also manifest by changes in the pupils.

The *small pupils* not infrequently met with in these cases are usually merely a senile myosis, but sometimes—and especially is this true when the pupils are unequal—this symptom may be justly ascribed to changes in the sympathetic. The reflex to light is never changed by Parkinson's disease.

Among the most trying of the symptoms complained of is *restlessness*, a species of general discomfort analogous to *anxietas tibiarum*, distributed over the whole body as a rule, but often particularly marked in the parts affected. Doubtless, it is due to fatigue to a great extent, but the feelings of heat and cold, the miscellaneous paræsthesiæ, the effort required in moving the rigid muscles, and the hyperidrosis all contribute their share to the production of this symptom.

Marie and Azoulay[2] have called attention to *tachycardia* as a frequent symptom in paralysis agitans, but it is, in my opinion, not as common as they report: 9 of my cases exhibited this symptom, manifested by palpitation and rapid pulse. In no case was the pulse above 120. *Mental symptoms* associated with paralysis agitans are not uncommon. They consist chiefly of a certain amount of depression, due to the hopeless and uncomfortable state of the patient, and of a varying degree of weakness of memory and other faculties which should be, perhaps, looked upon as essentially senile in character. I have observed 2 cases

[1] Grasset believes the thermal paræsthesia was accompanied by an actual rise of temperature, but this is certainly not true. Gowers mentions a case in which the affected side (in which was an intense feeling of heat) was .6° F. warmer than the other. But it is not uncommon in normal persons to find an axillary asymmetry of temperature as great as this, and it seems to me this observation is therefore not of great significance.

[2] *Progrès méd.*, 1885, No. 49.

in which there was actual insanity, but I should be disposed to consider this an associated condition rather than a psychosis having a pathogenic relation to paralysis agitans. Some authors, like Ball and Parant, believe in a close relation between the two, and the latter divides the psychical consequences of Parkinson's disease into three groups—viz. one in which there is irritability, suspicion, and depression; one in which there is enfeeblement of the mind, varying from simple blunting of the faculties to complete dementia; and a third group with symptoms of insanity, properly speaking, such as melancholia simplex or melancholia with hallucinations, delusions of persecution, suicidal impulses, etc.

Complications.—Hemiplegic attacks may occur in the course of the malady, but are episodes not so much related to the disease as to the age of the patient and the condition of his bloodvessels. Hemiplegia arrests the tremor on the side paralyzed. Cases have been reported in which transient hemiplegia without discoverable lesion, and in which epileptiform convulsions, cataleptoid conditions, and cramp in the limbs have complicated paralysis agitans.

Diagnosis.—The diagnosis of paralysis agitans is not difficult in a typical case presenting the characteristic tremor, attitude, facies, and rigidity. In the rare cases where the tremor is absent the peculiar position of the body and limbs, the gait, the rigidity, and mask-like face are in themselves sufficient for diagnosis. But there are cases in which these distinctive symptoms are wanting or do not develop for a long period of time, and the diagnosis must be made by exclusion.

Senile tremor is sometimes not easy to distinguish from that of Parkinson's disease, though there is never difficulty when such tremors are typical. Usually senile tremor is found only in very old people, and is in both hands from the first. Simple tremor occurring at any age, generally hereditary in nature, is not to be overlooked as a possible cause of confusion in an obscure or atypical case. Between the intention tremor of multiple sclerosis and the tremor of paralysis agitans, when typical, there is so marked a contrast that differentiation is easily made. But as there are some rare cases in which the tremor of one may simulate that of the other, paralysis agitans must then be recognized by the mask-like face, attitude, age, and gait; while syllabic speech, nystagmus, and exaggerated tendon reflexes should serve to indicate multiple sclerosis. I have seen a post-hemiplegic tremor very like that of Parkinson's disease, but usually post-hemiplegic tremor is fine in character, and the history of onset, the exaggerated reflexes, etc. are distinguishing features.

Prognosis.—The disease is chronic, incurable, and generally progressive. Sometimes a few months only elapse before all the extremities are implicated, but more often years. Occasionally it may become stationary after one or two limbs only are affected. It is not in itself a fatal disease, and probably has but little influence upon the duration of life. The disease has been known to last for thirty years. As a rule, one of the intercurrent affections common to old age is the cause of death. Any amelioration of the symptoms by treatment is all that can be expected.

Treatment.—The treatment is wholly symptomatic, and the remedies made use of are usually given for the amelioration of the two most

trying symptoms—viz. the tremor and the feeling of general discomfort. For the tremor there is no drug which acts so well as one of the isomeric alkaloids, hyoscyamine, hyoscine, or duboisine. The last named of the three is the best, as it has fewer of the unpleasant effects of these alkaloids. One may begin with from $\frac{1}{200}$ to $\frac{1}{100}$ of a grain night and morning, and increase the dose as needed. There are some cases that cannot take as much as this—indeed, a few who cannot take the drug at all. Others, however, take it for months and years even without bad general effect, and with more or less marked diminution of the tremor. The other indication, relief of the restlessness and discomfort, pains, paræsthesiæ, etc., is best met by the use of opium in some form. Heiman is an enthusiast in the use of morphine in these cases. My own practice is to combine duboisine (gr. $\frac{1}{100}$ or less) with codeine (gr. ss–j). There is less likelihood of a habit being acquired in employing codeine, although an opium or morphine habit in an aged person is not a thing to be especially dreaded.

Other drugs which have been used for the relief of these two symptoms are veratrum, Calabar bean, chloral, atropia, belladonna, gelsemium, curare, conium, eserine, cannabis indica, solanin, and the bromides, occasionally with some success. Some of these may be tried where duboisine and codeine have no effect.

Some general indications in paralysis agitans are to be met by appropriate treatment. Thus, the daily life should be regulated so as to avoid physical exhaustion, worry, and care. Tonics are frequently of service, and among these arsenic, strychnine, and the peptonate of iron and manganese have distinct value.

Electricity in any form is of no service, except occasionally in the way of suggestive therapeutics. On the other hand, gentle massage and passive Swedish exercises are often very beneficial. The same is true of hydrotherapy. The affected limb or limbs may be immersed in hot water for ten or fifteen minutes nightly, or the hot wet pack applied. Vibratory apparatus, such as a "shaking" chair, has been used with some success, and deserves further trial.

TREMORS IN GENERAL.

TREMOR is a common symptom in many nervous conditions, and has distinct diagnostic value. Superficially examined, all tremors have a close resemblance, but careful investigation reveals decided differences. There are several standpoints from which a classification may be made. Thus a useful division is into coarse and fine. To the former belong the tremors of paralysis agitans, senility, multiple sclerosis, delirium tremens, and post-hemiplegic polymyoclonus. The fine tremors are those of neurasthenia, Graves's disease, hysteria, mild toxic conditions, and general paresis. The former may be assumed to have a rate of about five per second. They are fused groups of two or three impulses in the normal innervation rhythm. The fine tremors average about ten per second, and correspond, therefore, to the normal innervation rhythm.

Another division may be made into intention tremor and that occurring while the parts are at rest. Intention tremor would include, then, nearly all of the tremors, coarse and fine, with the exceptions of two, that of paralysis agitans and post-hemiplegic polymyoclonus, which belong to the latter category.

The writer would suggest the following as a good working classification:

I. Hereditary tremor;
II. Senile tremor;
III. Physiological tremor (cold, fear, emotional excitement), muscular fatigue;
IV. Pathological tremor.

Organic . . .	Multiple sclerosis, Post-hemiplegic polymyoclonus, Spinal progressive muscular atrophy, General paralysis.
Functional . .	Toxic (alcohol, mercury, copper, lead, arsenic, tea, tobacco, coffee, opium, chloral), Exophthalmic goitre, Paralysis agitans, Hysteria, Neurasthenia.

Hereditary Tremor.—This is a simple tremor, without concomitant muscular weakness or rigidity, under control of the will to a great degree, occurring in young or middle-aged people in whom there is a neuropathic heredity. The tremor may be directly hereditary, even for two or three generations. It is usually fine in character, but sometimes quite marked, irregular in amount of excursion. It ceases during rest, becomes apparent on effort, and is augmented by emotional conditions. It is commonly noted in the hands, but other parts may be affected, such as the neck muscles, the face, and the tongue. It offers no particular interference with one's vocation, and unless very inconvenient needs no treatment. Its presence may give rise at times to difficulty in diagnosis from toxic tremor, general paralysis, or multiple sclerosis if other suspicious symptoms are present.

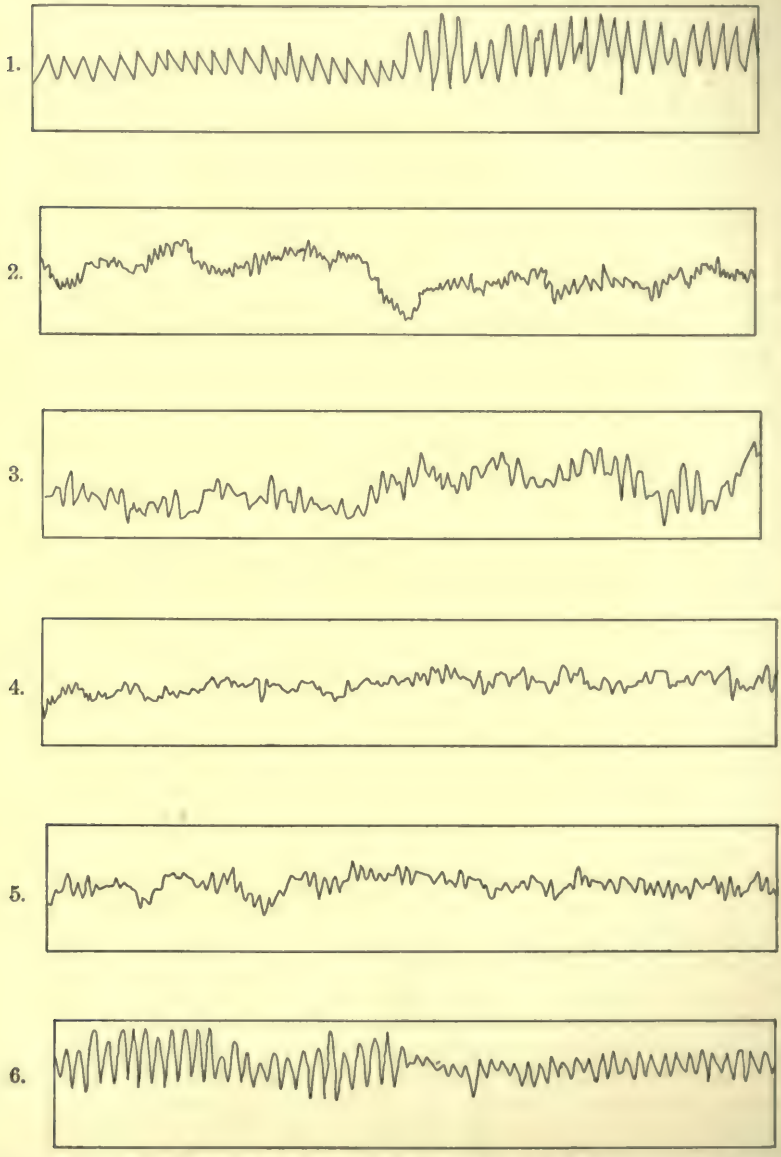
Senile Tremor.—A tremor of fine or coarse character, unequal and irregular, quite frequently presents itself in old age. It ceases during sleep or when the hands are at rest, and is chiefly apparent when an attempt is made to do anything, as, for instance, to write. It affects the hands, arms, and neck. There is none of the rigidity of paralysis agitans and no mask-like face, peculiar gait, nor festinating speech. But occasionally cases are met with in which the differential diagnosis between this condition and Parkinson's disease is very difficult.

Physiological Tremor.—This species of tremor may be of physical origin, as in shivering from cold or from muscular fatigue; or it may arise from a psychic cause, such as fear, anger, or grief.

Tremor of Multiple Sclerosis.—This is a coarse, irregular, jerky sort of tremor, occurring only on attempted movement. It is a typical "intention" tremor. The more the individual tries to control the parts

the wider become the excursions. It is augmented by emotion, attention, and effort. The patient cannot draw a straight line on a black-

FIG. 74.



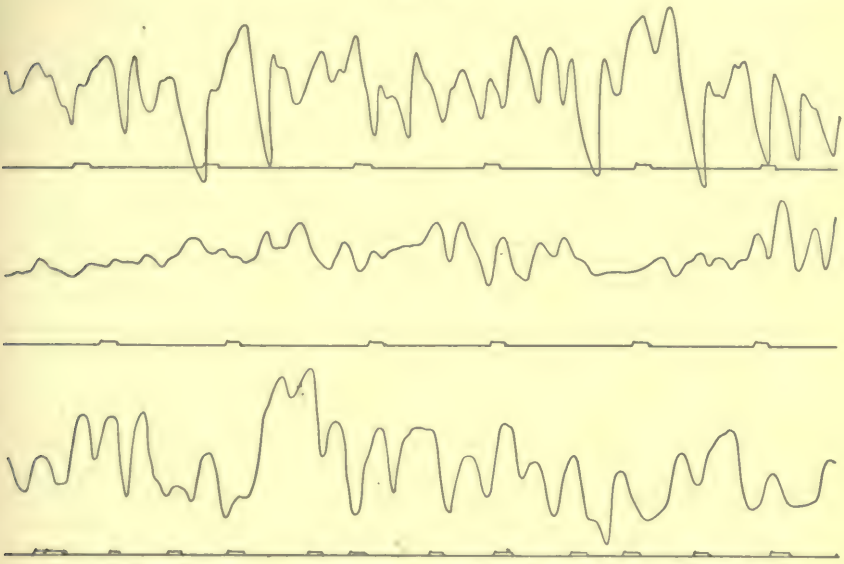
Comparative series of myograms of various tremors, taken with the sphygmograph, and therefore not perfectly accurate as to rate per second: 1. Paralysis agitans, 4.7 per second; 2. Graves's disease, 11.7; 3. Multiple sclerosis, 5.4; 4. Hysterical tremor, 7.7; 5. Neurasthenic tremor, 7.4; 6. Delirium tremens, 5.6.

board, whereas the patient with shaking palsy is able to do so perfectly. The tremor is usually marked in the hands, often implicates the neck

and tongue, more rarely the legs. The face is usually unaffected. (See Fig. 74.)

Post-hemiplegic Polymyoclonus.—This is a name given by myself to a variety of post-hemiplegic movements observed in only a few instances out of several hundred cases of infantile spastic hemiplegia. It consists of a rapid and not synchronous clonic spasm in the muscles of the paralyzed limb. The movements of the separate muscles have about the same rapidity as those of paralysis agitans, and the rate is the same—five per second (Fig. 75). If the muscles moved synchronously

FIG. 75.



Tremor of post-hemiplegic polymyoclonus (taken from thumb only with kymograph).

the condition would be exactly analogous to shaking palsy. The tremor is constant and only ceases during sleep. It is neither a choreiform nor an athetoid movement, but a special kind of tremor. Fig. 75 shows this tremor taken with the kymograph, the whole hand clasping a bulb connected with Marey's tambours and the pen. The movement in the thumb alone is indicated in the illustration.

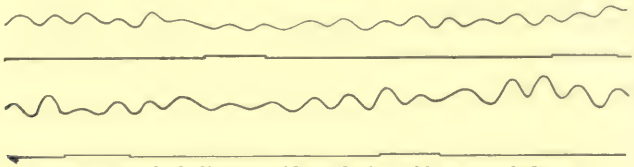
Tremor in Progressive Muscular Atrophy.—This is a *fibrillary* tremor, a fine rippling tremor that travels over the surface of muscles without moving the parts to which they are attached. It goes from one bundle of muscle fibres to another, and is most marked in the wasted muscles. There is no tremor in the fingers or hands. It is present in the spinal type of muscular atrophy, and absent in the primary muscular dystrophies. This minute fibrillary tremor is common also in the tongue in general paralysis, and is occasionally met with in other nervous diseases.

Tremor in General Paresis.—In addition to the fibrillary tremor just mentioned, frequently observed in the tongue in these cases, there is often marked tremor of the fingers, hands, eyelids, face, lips, cheeks,

and lower extremities. At first probably fine in the fingers, it gradually assumes a jerky and coarse character, and may be called then an ataxic tremor, a jerky inco-ordination. It manifests itself on any attempt to use the muscles. As the disease progresses the amplitude of the uncontrollable jerky movements becomes greater and greater. Facio-lingual tremor of this nature is particularly significant, as it is only in very rare instances that it is met with in other disorders than general paralysis.

Toxic Tremors.—There are a number of poisons which give rise to tremor, usually after long-continued action, but sometimes after acute intoxication. These poisons are alcohol (Fig. 76), mercury, copper, lead,

FIG. 76.



Tremor of alcoholism: rapid revolution of kymograph drum.

arsenic, tea, coffee, tobacco, opium, and chloral. Perhaps the tremor of Graves's disease should be included here among the toxic tremors. There is no particular diagnostic difference between the tremors induced by any of these poisons. They all belong to the category of fine, rapid tremors, not perceptible when the parts are at rest, but brought out on intended movement. They are in reality, therefore, a species of intention tremor. They become especially noticeable on having the patient extend and separate his fingers, and then extend his wrists strongly, and also on light closing of the eyelids. A mild degree of poisoning leads to merely a fine vibration of the parts, but continued exposure to the toxic influence causes constant increase in the amplitude of the vibration, until the tremor may become quite as coarse as in multiple sclerosis or paralysis agitans. This is notably true in alcoholism when it has reached the stage of delirium tremens. It is also true of the "metallic tremors," especially of mercury and lead. Where the tremor reaches such a degree most of the muscles of the extremities, neck, face, and tongue may be involved. In alcoholism and mercurialism the facio-lingual tremor may closely simulate that of general paresis.

The **tremor of exophthalmic goitre** is similar in all respects to the toxic tremors, except that it rarely goes beyond the stage of fine vibration on muscular effort. (See Fig. 74, p. 686.)

Hysterical Tremor.—This is ordinarily of the fine variety, but occasionally it approaches the coarser forms. (See Fig. 74, p. 686.)

Neurasthenic Tremor.—The chief objective symptom of neurasthenia of any variety is a fine vibratory tremor, only present on effort, never noticeable during rest. First perceptible in the fingers when the hand is extended and the fingers separated, it generally spreads to the eyelids, and may implicate all of the muscles of the body, so that the patient complains of a constant feeling of general tremulousness. In the face and tongue it only rarely approaches any degree of similarity to the tremor of general paralysis.

HYSTERIA; DISORDERS OF SLEEP.

By WHARTON SINKLER, M. D.

HYSTERIA.

Definition.—By "hysteria" is understood a functional disease of the nervous system dependent upon a disorder of the higher cerebral centres. The functions of the lower centres—namely, those of the spinal cord—are also involved, as well as those of the sympathetic nervous system. The term hysteria, which is derived from the Greek *ustera*, the womb, indicates the original idea that the affection depended upon uterine disease. The ancients seemed to have held most extraordinary views as to the properties of this organ. Hippocrates and other later writers believed in the idea of the "wandering womb," and the globus hystericus was supposed to be caused by the ascent of the womb against the liver and diaphragm. The practice of administering medicines having strong and disagreeable odors to patients suffering from hysterical seizures probably originated in the absurd belief of the migration of the womb to other parts of the body. Aretæus held that it was possible to drive the womb down by presenting fetid odors to the nose, and that it was driven upward by the same odors applied to the vulva. Many of the views as to the influence of the sexual organs in the causation of hysteria appeared from time to time until the nineteenth century. Galen believed that hysteria was caused by suppression of the menses or the retention of semen. Other writers recommended marriage as a remedy for hysteria. Even so late as 1816 a French writer, Villeimay, asserted in a treatise on this subject his belief in the existence of a sperm in the female, and also in the peregrinations of the uterus, and makes prominent his opinion that hysteria is a disease of lubricity. Space does not permit of going into the teachings of ancient writers in regard to hysteria. The different views presented are more a matter of interest from their eccentricity than by reason of any scientific value. Those who are interested in the subject will find a full reference to all of the older views of the subject in the work of Gilles de la Tourette.[1]

The theories as to the part played by the uterus in hysteria received full credence until the Middle Ages; indeed, this influence is seen even to the present day. The popular belief still exists that in every case of hysteria in women there must be some uterine or ovarian disorder, and even among medical men we see evidences of this fact in that their treatment of hysteria is influenced by their belief in the effect of uterine disease upon the affection. The theory of the origin of hysteria in

[1] *Traité de l'Hystérie*, vol. i., Paris, 1891.

the sexual organs was overturned about the end of the eighteenth century by several writers, among whom were Sydenham, Willis, and Whyte. Rush recognized hysteria as a disease of the nervous system, and also its relation to mental disease. Finally, Briquet, and soon after him Charcot, established on a firm scientific basis the true explanation of hysteria as a neurosis. There has been a period in the history of hysteria when the materialistic view of its nature was abandoned; when the pendulum swung too far in the other direction, and an hysterical person came to be regarded as a simulator and impostor, or as one suffering from an imaginary complaint. The result of this teaching has been that even now there are many physicians who regard hysteria as the vaporings of silly girls or weak-minded youths, and the victim of the disease is regarded with contempt rather than compassion. It is of importance, therefore, to bear in mind, and also to urge the fact, that hysteria is a disease as distinct and well defined as epilepsy or any other malady, and that it depends upon definite and constant changes in the nerve centres which as yet we cannot describe, but which undoubtedly exist. We must not fall into the common error of considering hysteria a "protean" affection, a term so often used by writers, simulating, through the conceit or knavery of the patient, various diseases. The fact that anæsthesia is often discovered in an hysterical patient, who has been unaware of its existence prior to the examination of a physician, proves that hysteria is not a condition the result of mendacity or folly. The psychic element in hysteria is distinct and constant, and has great bearing upon many of the symptoms.

Etiology.—The causes of hysteria are numerous, but there is probably none so potent or universal as the influence of heredity. Race, climate, and civilization have more or less effect. In France hysteria is more common than in England, and there the disease is quite different in many of its characteristics. Hebrews are more liable to the graver forms of the disease than other people. Civilization seems to tend to its development, and yet persons living in rural districts are also subject to it, and in barbarous nations conditions exist which resemble hysteria. For example, the excitement which occurs in the religious dances of Indians and other uncivilized people is allied to some of the epidemic outbreaks of hysteria which are seen in educated communities. It occurs quite often in the negroes of this country. The disease is more commonly met with among the poor and the wealthy than in the middle classes.

Heredity.—Heredity is not always direct—that is, an hysterical patient does not always have a history of hysteria in the parents, but some other neurosis has generally existed in an immediate ancestor. Frequently several cases of hysteria will be found in one family, and one generally finds on inquiry that the brothers or sisters of the patient have been the subjects of epilepsy, insanity, or some other form of nervous disease. Faulty bringing up of a child has much to do in creating a predisposition to hysteria and allied affections. Hysteria is most frequently transmitted from the mother. Some authors, however, think that the bad influence of an hysterical mother in the bringing up of her children explains the apparent transmission of the disease. It is often the case that the mother of an hysterical girl is lacking in judgment

and discretion. Gowers truly says:[1] "The near relatives of the hysterical are often conspicuously deficient in judgment, and the little common sense they may possess is often rendered useless by their affection for the sufferers." Heredity plays an especially important part in the hysteria of children. Briquet in an examination of a large number of cases found that hysterical women had a history of hysteria in the parents twelve times as often as non-hysterical women.

Age.—Hysteria is a disease of early adult life, the greatest number of cases occurring between the ages of fifteen and twenty-five years. In women the disease begins at an earlier age than in men. Children may be the subjects of hysteria, but, as a rule, it occurs after the age of seven years, the greatest number being between the age of eleven and fourteen. Clopatt, quoted by Lloyd,[2] gives the statistics of 272 cases under the age of sixteen years; only 9 of these were under six years. It has only been in recent years that hysteria has been recognized in children, and cases have been reported in children as young as three years. The writer has seen a child of three years, born of elderly parents, who was unable to walk and made no attempt to stand. If an attempt were made to place him upon his feet, he seemed much alarmed and cried loudly. A little firmness and training made the child walk within a few days. This was undoubtedly a case of infantile hysteria. Briquet considers that one fifth of the cases of women occur before puberty, and that the disease is most common in women at the age of twenty years. Hysteria may occur in persons advanced in years, but the initial symptoms very rarely begin after the age of fifty. I have recently seen a woman of ninety-one who had developed hysterical phenomena after the age of ninety.

Sex.—Hysteria is far more common in women than in men. The proportion varies according to the country in which the statistics have been made. In Germany it is said that the proportion of hysteria in men to women is 1 to 10. In France, Marie found that the graver form of hysteria was relatively more frequent in men than in women. This observation, however, applies to the lower classes, among whom hysteria in the male is more prevalent than in the higher classes. In relative proportion the cases in males and females vary also according to the age. In the table compiled by Clopatt,[3] embracing 272 cases, the number of cases in girls and boys is most equal between the ages of ten and thirteen years.

Trauma.—Injuries are frequently the cause of hysteria. Some writers, especially the French, believe that trauma is one of the commonest causes of hysteria. Various local palsies and anæsthesias are the result of some injury. The importance of traumatic hysteria is especially great in connection with litigation cases. Many cases of hysteria have been the result of railroad and other accidents, and even slight injuries in persons predisposed to hysteria have been sufficient to develop the disease. Acute diseases are occasionally the forerunner of an attack of hysteria, and it is sometimes difficult to make a diagnosis between hysterical paralysis and multiple neuritis after an infectious disease, such as typhoid fever, scarlet fever, and diphtheria.

[1] *Diseases of the Nervous System*, vol. ii. p. 986.
[2] *Nervous Diseases by American Authors*, p. 96. [3] Gilles de la Tourette: *Op. cit.*

Mental and moral influences are often potent in the production of hysteria. Children who are over-indulged are liable to become self-indulgent later in life, and readily develop hysterical affections. Imperfect education and lack of moral training at home predispose very distinctly to the disease. Fright, grief, and other moral shocks are frequent causes of hysteria. Disappointment in love is not an uncommon cause, and religious excitement or emotion has been, for all ages, recognized as a common origin of hysteria. The epidemics of hysteria which occurred in the Middle Ages originated in religious excitement and assumed great proportions. Even at the present day epidemics of hysteria occur in schools and in institutions without the influence of religious excitement.

Toxæmia.—The influence of poisons in the causation of hysteria has only recently been studied. The prolonged and excessive use of alcohol is undoubtedly a cause of hysteria, and so are certain other poisons. Briquet in 1859 reported several cases of hysteria in males whose work was in lead, and since that time other cases of a similar character have been reported. The opium habit is often a cause of hysteria; indeed, it is seldom that one meets with a case of opium habit in which symptoms of hysteria are not present. Other poisons, such as mercury, lead, tobacco, and absinthe, have the property of inducing hysteria.

Diseases of the generative organs have been for years considered as exerting marked influence in creating hysteria, but it is probable that the influence of ovarian and uterine disease is no greater than that of other organs. Tenderness in the ovarian region is undoubtedly a frequent symptom, but it is not confined to the subjects of hysteria, and it is also true that in an hysterical man tenderness may be elicited in the same region. Severe cases of hysteria have been said to be cured by the removal of both ovaries, but many cases of hysteria have been unrelieved by this operation; in fact, it is much more common to meet with cases which have been unrelieved or aggravated by operation than those which have been benefited. There are certain diseases with which symptoms of hysteria are frequently associated; among these are notably diseases of the uterus and appendages, movable kidneys, and mucous enteritis. The truth is, as Mills says,[1] "Frequent or severe local irritation in any part of the body in an individual of the hysterical diathesis may act as the exciting cause of an hysterical paroxysm or of special hysterical manifestations."

Sexual excesses and self-abuse are probably occasional causes of hysteria, but it is not so common as one would be led to suppose from the older writers.

Defective general health often, but not invariably, precedes hysteria. Hysterical patients are frequently anæmic and poorly nourished, and have various functional disorders, but as often the subjects of hysteria are plump and ruddy. Neurasthenia and hysteria are diseases which are often confounded. It is because the two conditions are so often associated, and we must be careful not to mistake one disease for the other, as they are such distinctly different disorders. Hysterical symptoms are often manifested in organic diseases of the nervous system. This is particularly the case in tumors of the brain. It often occurs that the

[1] *System of Medicine*, Pepper, vol. v. p. 221.

symptoms of brain tumor are masked by evidences of hysteria which exist, and mistakes in diagnosis are frequently made through this source. In organic cord disease hysterical symptoms may also be present. In epilepsy it is by no means infrequent to find that the patient is also hysterical, and two distinct forms of attacks may occur, true epileptic convulsions and hysterical seizures alternating in the same patient.

PATHOLOGICAL ANATOMY.—No gross changes are found in the nervous system in hysteria. In some cases certain alterations have been found in the brain and spinal cord after death, but these are no doubt accidental complications. That hysteria is a purely functional disease is admitted by all authorities. The various symptoms which occur depend upon a disordered state of the cerebral centres. The lower centres control a certain set of symptoms, while other phenomena depend upon derangement of the higher centres. There is no doubt that hemianæsthesia is due to a functional disorder of the sensory centres on one side of the brain. Similar functional disturbances are seen in some cases of migraine, in which, preceding the attack of head-pain, there is transient hemianæsthesia with aphasia. There is evidently some nutritional and functional change in the nerve element of the cortex, which is the underlying cause of the general disorder of function of the centres which occurs. The causes of the derangement of the nerve element may be various, but the derangement is often psychical. Since the discovery that the essential tissue of the nervous system is made up of a number of distinct and independent neurological units which have been called neurons, our conception of the pathological alterations which may occur in the nerve centres without gross lesions has undergone a vast change. The theory of the movement of the neurons, which was first advanced by Rabl-Ruckard,[1] and later by Lepine, has afforded a large field for speculation as to the various phenomena which are met with in the functions of the cerebrum. Dercum[2] suggests a plausible and attractive theory as to the cause of the paralyses and anæsthesiæ of hysteria. He says: "As a result, we will say, of an emotional or other psychical shock, or possibly of a physical shock, the neurons of the arm centres of the cortex retract their processes in such a way that their under-tufts in the spinal cord no longer bear their normal relation to the spinal neurons. In other words, the connection between the cells is broken." He then assumes that as a result of suggestion, either with or without hypnotism, the paralysis disappears. The removal of the paralysis, he then assumes, is due to the extension or protraction of the processes previously retracted, and the resumption of the normal relations with the spinal neurons then occurs. Of course, before this theory can be accepted it must be proved that the neurons possess the power of movement, but the view is one worthy of thought for consideration and investigation.

SYMPTOMS.—Hysteria has been divided into hysteria major and hysteria minor, but for practical purposes this division is unnecessary, as the symptoms are essentially the same, only differing in degree.

The symptoms of hysteria are divided into two classes, those of the paroxysm and those between the paroxysms. The symptoms of the paroxysms are extremely prominent when they occur, but they are not

[1] Dercum: *Philadelphia Polyclinic*, April, 1897. [2] *Op. cit.*

always present in all cases of hysteria. Many cases of hysteria, at least in this country, never have the so-called attacks of hystero-epilepsy. This term is an unfortunate one, although it has the authority of Charcot, but it implies the idea that the hysterical paroxysm is a combination of two diseases—hysteria and epilepsy—whereas this is not the case. The paroxysm is purely and distinctly hysterical without any of the essential features of epilepsy, except that the convulsions are imitated to some extent. It is to be borne in mind that a patient who has attacks of hysterical convulsions may also have true epilepsy. Various names have been suggested for the paroxysm, but none are especially appropriate.

The most common symptoms of the crisis are simply attacks of laughter and crying alternating, and this may become more uncontrollable, and finally terminate in convulsive seizures of various types. In addition to this, there may be seizures of violent pain, nausea, and vomiting, or attacks of coughing, sneezing, or irregular breathing. The paroxysm may also take the form of attacks of trance, catalepsy, and various mental phenomena. In the attacks which are mainly emotional the patient begins to laugh without any apparent reason; she laughs immoderately and continuously, and becomes quite unable to control herself. Attacks of crying, passion, or excitement may occur in the same way. Frequently associated with these seizures is a peculiar sensation in the throat which is described as being like a ball in the throat rising upward. This is the so-called "globus hystericus."

The hysterical paroxysm is always preceded by prodromes or auræ. These are usually mental in character, and the patient for a few days before the attack is restless, depressed, and disturbed. As the time for seizure draws near the patient's emotional nature becomes more disturbed. Delusional ideas and disturbed dreams may be present at this time. Frequently the attack is preceded by disorders of digestion and perversions of appetite and taste. Various other sensory and motor symptoms become conspicuous at this time, such as anæsthesia, hyperæsthesia, and contractures. The convulsions are usually preceded by an aura, and the most common is the globus hystericus and ovarian tenderness. A severe pain in the vertex, which is likened to the driving of a nail into the part—hence the term "clavus"—is also a common aura. There may be other auræ, such as noises in the ears and tender points in various regions.

The convulsion occurs in two recognized types. One of them is that which is described as "hysteria minor," or the less severe form of hysterical convulsion. The other, which is much more violent, is what is termed "hystero-epilepsy" or "hysteroid." In the first form the patient falls down rather suddenly, but not so as to hurt herself, and various irregular movements of the body are executed. The hands and arms are flexed, the legs and feet are extended, and the patient rolls over on the floor. The eyelids are usually closed, but when separated the eyeballs roll about and the pupils are usually dilated. As a rule, the separation of the lids is resisted by the patient. Screams are occasionally uttered, and the patient may talk at random. The tongue is not bitten, and the patient seldom hurts herself in the various contortions which occur. In some cases the attack is nothing more than a general rigidity

of the body, with shaking and shivering, as though the patient had a chill; sometimes the attacks are associated with peculiar noises and movements in imitation of an animal, such as the barking of a dog or the mewing of a cat. This condition is called therio-mimiery. The milder attacks seldom last more than half an hour, and may be cut short by pressure over the ovaries and epigastrium; and dashing cold water over the patient is usually prompt in ending the seizure. In many patients the attack ends here, but in others, after a short intermission, the movements begin again. This has been called the "second period of the grand attack," and has also been named by Charcot "clownism." In this period the movements of violent and extensive form occur. They are either in the nature of disordered contortion or may be apparently volitional in their character. Movements of this kind are what have occurred in the epidemics which occurred in the Middle Ages, and which, indeed, have taken place in our own time. The most common movement is that when the patient, lying on her back, arches herself up so that the body rests upon the neck and heels; and, again, the body may be arched to one side or the other. Other movements consist in alternate flexing and extending of the limbs and swaying of the body to and fro. The patient appears to be in a state of extreme excitement, and the movements are kept up for some time. There is apparently no loss of consciousness in this period.

In the third period, which has been described by Charcot as a period of "passionate attitudes," the patient becomes distinctly emotional and seems to be possessed of hallucinations. The movements are all dramatic, the patient placing herself in various attitudes and assuming expressions of resentment, anger, remorse, fear, scorn, and lamentation. Sometimes the attitude representing the cross is seen. During the third period the patient seems to be oblivious of painful impressions. Needles may be thrust into the skin and strong faradic currents applied without producing any apparent impression. The conjunctiva is said to be insensible, and the patient cannot be roused by loud noises. It is said, however, that after the attack the patient remembers what has occurred during it.

The fourth period of the attack has been called "the period of delirium," and during this the patient is noisy, weeping, or talking wildly. The delirium may end, however, in obstinate mutism.

The grand attack may last for from half an hour to several hours. It must be said, however, that the symptoms of the grand attack are comparatively seldom seen in this country. In France they are much more common, and the description given by Richer is most complete and startling. Gowers declares that these attacks are seldom, if ever, seen in England. They do occur in this country, but not to any great extent. It is probable that if an elaborate study were made of the hysterical paroxysm in a large hospital in which there were many cases of hysteria, the grand attack would be developed in a number of cases as a result of imitation. Quite frequently patients are seen in whom there is an abortive attack in which the prodromes only are a conspicuous feature. The patient, after some days of mental irritability, depression, and some digestive disturbance, has an emotional outburst of laughter, weeping, etc., and this is followed by a slight convulsive attack.

Chorea major is a form of "grand hysteria." There are extensive irregular movements, which are exaggerated by excitement or emotional disturbance of any kind. Either ecstasy, somnambulism, catalepsy, trance, or lethargy may terminate the paroxysm of hysteria major. Hysterical sleep is a condition which occasionally occurs, and many of the cases of prolonged sleep which are reported in the popular press are probably hysterical.

Interparoxysmal Symptoms.—The symptoms which are present between the paroxysms of hysteria major are the most important, because they are present in all hysterical patients. They differ in degree and extent according to the peculiarities of the individual. These symptoms have been called "stigmata," or the marks of the disease. Some of the stigmata are transient and others are permanent. These stigmata have been divided into four classes—sensory, motor, visceral, and psychical.

Patrick[1] truly remarks that "not only may concomitant symptoms and hysterical stigmata be absent, but the so-called hysterical disposition or hysterical tendency may be entirely wanting."

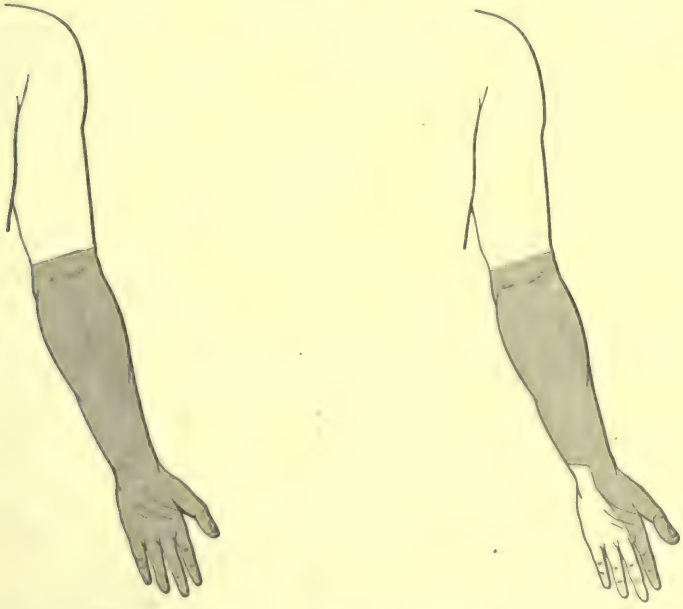
Sensory Symptoms.—Sensory disturbances are always of importance in hysteria, as they are present in some form in most hysterics, although this is not always the case. They should always be carefully looked for, however, for frequently the patient is not aware of changes in sensation which may be present. Anæsthesia is one of the commonest forms of sensory disturbance in hysteria. This may be partial or complete, and it may extend over the entire surface or only in limited areas. Incomplete loss of pain sense is most frequently met with. This was formerly thought to be feigned by hysterical patients, but the analgesia is often discovered by the physician before the patient is aware of its existence, and the distribution of it follows certain rules and laws. Tactile anæsthesia without analgesia is met with almost as often as analgesia alone. Hemianæsthesia is the form which one generally sees in this affection. It is often absolute, the patient not perceiving impressions of any kind. Pins and needles may be thrust deeply in the flesh without causing pain, and the skin may even be burned without the patient being aware of it. In many cases the patient is conscious of a sense of pressure, but nothing more, and is often surprised on looking to see that a pin has been inserted into the flesh almost to the head. Frequently there are vasomotor changes, so that the blood does not flow from the pricks of the needle. The absence of blood, however, is not a necessary symptom. There is loss of thermal sense as well as pain sense. The analgesia is not confined to the skin, but the deeper tissues are also insensitive. The nerve trunks may even be wounded without giving rise to a painful sensation. There may also be anæsthesia of the mucous membranes on the hemianæsthetic side. The mucous membrane of the nose, mouth, eyes, vagina, and rectum may be devoid of sensation.

Hemianæsthesia may come on after the grand attack, but it may occur spontaneously. It, like many others of the sensory disturbances, frequently occurs after traumatism. An interesting fact is, that the ovarian and other deep-seated tenderness may remain unchanged on the anæsthetic side. Another point of diagnostic interest is, that the patient

[1] *New York Med. Journ.*, Feb. 15, 1896.

is still able to use his hands and fingers; as, for instance, in writing without the guidance of the eye or raising a tumbler of water above the head and still holding it. The reflexes, both of the tendons and irides, are preserved. There may also be amblyopia or complete loss of vision, as well as deafness on the affected side. In hysterical hemianæsthesia there is often preservation of touch and heat sense when the pain sense is lost, but there is seldom loss of sensation to the faradic brush, although occasionally this is not felt. A curious and characteristic feature in hysterical hemianæsthesia is the transference of the anæsthesia from one side to the other. The phenomenon of transfer occurs sometimes without any known cause, but it is usually the result of emotion or suggestion, and the application of certain agents, such as blisters, faradism, magnets, and some metals, effects the transfer from one side to the other. The influence of the metals was believed by some

FIG. 77.



A *B*

A, segmental anæsthesia of left forearm and hand (personal observation); *B*, the same case four weeks later. The anæsthetic area is shaded.

French authors to be the result of some occult action of the metal themselves, and these were called æsthesiogenic agents, and much has been written and said on the subject of metallo-therapy. There is no doubt that the action of these metals is only due to suggestion. The action of magnets is undoubtedly the same, for it is impossible to conceive that holding a magnet, no matter how powerful, an inch or two from the surface could influence the nutrition of the nerves. When transfer has occurred, it is usually only temporary, and the anæsthesia soon returns to its original site. As a rule, hemianæsthesia affects one side completely, extending exactly to the median line of the body and reaching from the top of the head to the sole of the foot. In some cases, however, it may

not extend entirely to the median line posteriorly, or it may extend a little over the median line anteriorly. For some unexplainable reason the left side is more often affected than the right, and it is to be noted that frequently ovarian tenderness remains unaffected on the anæsthetic side. Anæsthetic patches of greater or less size may be found in different parts of the body. They may be round, oval, or irregular in form, and constitute the geometrical form of anæsthesia. Sometimes one limb alone is affected—the so-called segmental form (Fig. 77)—and the region affected may correspond to the part which would be covered by a glove or stocking. In rare cases the anæsthesia corresponds to the distribution of the nerves. A pathognomonic feature of the anæsthesia of hysteria is that the border-line of the anæsthetic area is sharply defined, while in organic diseases the anæsthesia shades off gradually.[1] Occasionally the anæsthesia is general, the entire surface of the body being involved. An unusual form is one in which the anæsthesia is complete with the exception of small areas, as seen in Lloyd's case.[2] The special senses undergo marked alteration, vision is lessened upon the affected side, and there may be crossed amblyopia, as seen in some organic diseases. The form fields are lessened upon the anæsthetic or both sides, and the color fields are diminished or reversed. There may be achromatopsia or dyschromatopsia. When there is contraction of the color fields, the fields which are normally largest become smallest; that is, violet is first lost, then green, then red, yellow, and finally blue. In reversal of the color fields the red field is larger than the blue—exactly the opposite of the normal condition. Complete loss of vision may occur in one or both eyes. This usually occurs suddenly after a "grand attack" or an accident; it usually lasts for a short time, but may persist for years, with improvement and relapses. In hysterical amblyopia the pupils continue to react normally to light and accommodation, and no changes are seen in the fundus of the eye. The phenomenon known as hippus is occasionally seen in hysteria. In this condition the pupils contract and dilate without the stimulus of light or accommodation.

The blindness in hysteria is psychic; that is, notwithstanding the inability of the patient to see, it can be demonstrated that she does see, as shown by an experiment of Pitres.[3] For example, if the patient be blind in the left eye and a screen is placed vertically between the eyes, when the right eye is closed the patient can see nothing on a printed page held before the eyes. If the left eye is closed, she sees the letters only on the right side of the screen, but if both eyes are open she sees the letters on both sides of the screen. Prince[4] has reported two interesting cases of hysterical monocular amblyopia coexisting with normal binocular vision; both males. Hysterical deafness is also psychic, as well as hysterical anæsthesia, for it can be demonstrated that the patient does feel certain sensations, and uses the muscles of the anæsthetic side without the aid of vision. Loss of smell (anosmia) may occur in hysteria. I demonstrated this in a case in which there was widespread anæsthesia of the skin and mucous membranes. Hyperæs-

[1] In some cases of syringomyelia, segmental anæsthesia, with sharply differentiated lines, is met with.

[2] *System of Nervous Diseases by American Authors*, p. 108.

[3] *Leçons clin. sur l'Hystérie*, vol. i. p. 192. [4] *Am. Journ. Med. Sci.*, Feb., 1897.

thesia may take the place of anæsthesia in some cases, and may take the same distribution as the latter. It is more commonly confined to certain localities, like the spine and the ovarian and mammary regions, the so-called hyperæsthetic zones. In the hyperæsthesia of hysteria a slight touch is more painful than a deep pressure. The hyperæsthesia may develop spontaneously or as a sequence of traumatism. Pain in certain regions is common; for example, the violent pain in the vertex (clavus) is very characteristic of hysteria. There may also be pain in other parts of the head, either frontal or occipital. Coccygodynia is often present in hysterical subjects. Bremer[1] considers the affection almost always hysterical, and protests against operations in these cases. Hyperæsthesia may be confined to one half of the body or it may extend over the entire surface. In some cases the entire abdomen is tender, simulating peritonitis. The special senses are hyperæsthetic in some cases. It is not uncommon to find that the eyes are extremely sensitive to light, and this hypersensitiveness is fostered by the habit of keeping the patient in a darkened room. Sounds may be very painful.

Paræsthesia.—Disordered sensibility may be met with in various parts of the body. There may be a sense of prickling or formication over the limbs, or a feeling as though water were being poured down the spine, with a sense of shivering and general coldness. Hysterical joint affections were first described by Brodie.[2] They are most frequently of traumatic origin, but they may occur spontaneously. The affected joint becomes exquisitely sensitive, and cannot be moved without giving rise to great pain. The skin over the joint and surrounding it is often hyperæsthetic. The most characteristic symptoms of an hysterical joint are the fear of motion and cutaneous hyperæsthesia. There is often, probably, a small amount of real lesion about the joint, and a considerable amount of swelling may be present. The joint usually affected is the knee. Hysterical joints are very troublesome. They are difficult of diagnosis and hard to cure, and relapses often take place after improvement has occurred. Frequently actual ankylosis results from the long disuse of an hysterical joint.

Motor Symptoms.—Paralysis is common, and is met with in various forms and degrees. The neuro-mimeses of paralysis by hysteria is remarkable. Organic paralysis is closely simulated, and often deceives careful observers. There may be monoplegia, hemiplegia, or paraplegia, and occasionally there is total motor paralysis. The paralysis may or may not be associated with contractures, and contractures may exist without paralysis.

Hemiplegia is probably the most common form of hysterical paralysis. Briquet asserts that he met with 74 cases of hemiplegia in 430 cases of hysteria. In hysterical hemiplegia the face is never paralyzed, although contraction of the muscles may give it the appearance of palsy. The walk is characteristic of the hysterical nature of the affection. The leg, instead of being swung around as in organic hemiplegias, is dragged stiffly up to the other. Hysterical paralysis is never limited to the distribution of a single nerve, but it may be segmental, like hysterical anæsthesia, or it may affect only certain movements. The paralysis is often associated with anæsthesia and occasionally

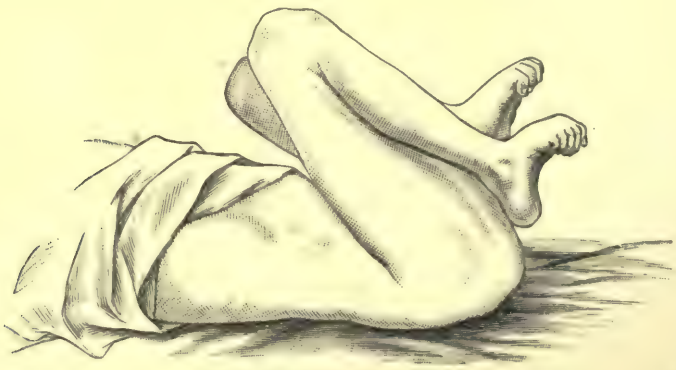
[1] *Med. Record*, Aug. 1, 1896. [2] *Lectures Illustrative of Certain Local Affections.*

hyperæsthesia of the affected part. The paralyzed limb may be œdematous, and the skin may be discolored and mottled from sluggish circulation. Loss of electro-contractility and the reaction of degeneration are never met with in hysterical paralysis, but, for that matter, in the organic cerebral palsies which are commonly counterfeited electro-sensibility is preserved. There is no true atrophy, but there is often wasting of the muscles from disuse. I have seen a case in which there were many symptoms of hysteria, including aphonia of sudden development and hyperæsthesia, but superadded was a neuritis of one arm, with pain, tenderness, and atrophy of the thenar and hypothenar eminences. Hysterical paralysis may develop gradually, involving one part and then another, but in all cases the symptoms are those of a paralysis of central, and not of peripheral, origin; and it is important to bear this fact in mind in connection with the pathology of the disease.

The duration of hysterical paralysis is variable. The patient may recover speedily or the paralysis may continue for months or even years. Complete loss of power may continue so long as to make one doubtful of the diagnosis. I have seen cases which have lasted for many years. It has been suggested by Charcot that in cases of long standing of hysterical paralysis sclerotic changes take place in the cord, but there is no positive evidence to prove this, and it is probable that in cases in which sclerosis has been found post-mortem this has been a complication rather than a result of hysteria.

Hysterical paraplegia differs from paraplegia of organic origin in many important particulars. In the former there is no girdle pain, the

FIG. 78.

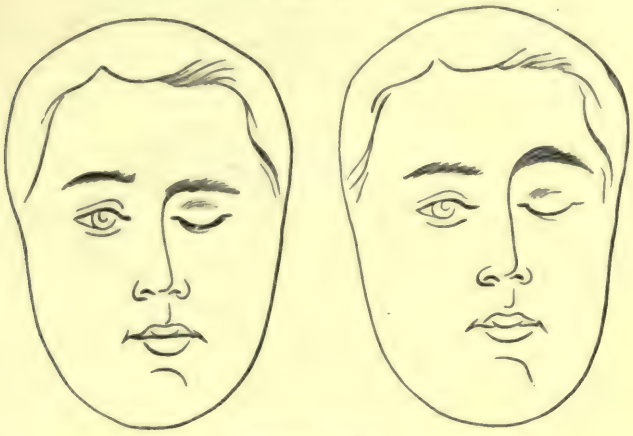


Hysterical contracture of legs.

bladder and rectum are not paralyzed, and there are no bedsores. The reflexes are generally exaggerated, but clonus is rarely met with. Occasionally there is paralysis of the bladder. In a patient under my care the urine dribbled for months, and this was not the overflow of distention, because when the catheter was used the bladder was always found empty. There is frequently contracture in hysterical paraplegia. The limbs are usually rigid and in extreme extension. Gowers says that in hysterical rigidity the legs are almost never in a state of flexion,

but such cases do occur, as was seen in the case of a child of thirteen years whom I saw some years ago. The legs were strongly flexed, as shown in Fig. 78, from a sketch made by the late J. M. Keating, and remained in this condition for many months. Mitchell[1] has reported a case of extreme hysterical contraction of the legs. The eye muscles may be paralyzed; ptosis is not an uncommon symptom (see Fig. 79), and occasionally the muscles of the ball are also paralyzed.

FIG. 79.



Hysterical ptosis.

Some of the facial and neck muscles may also be paralyzed. Contractures exist, either in connection with paralysis or without it. The contractures of hysterical origin usually occur suddenly, after emotion, shock, or traumatism. They are very difficult to overcome, because the antagonistic muscles are rigid, as well as those which are in a state of contraction. Sensation is often disordered in the contracted limb. The contractures may be made to disappear by suggestion. Ritchie asserts that contractures are sometimes exceedingly painful, but, as a rule, they are not. Unless of long standing, they invariably relax under ether or chloroform, and they may or may not relax during sleep. Mitchell[2] considers that there are two forms of hysterical contracture—one apt to be local and limited, and not followed by organic muscular changes; the other apt to affect two or more limbs and almost every muscle of the trunk, and prone to result in muscular and areolar-tissue changes. After hysterical contractures have lasted for several months, especially if they are multiple, they do not relax under ether or chloroform, because then shortening of the contracted muscle has taken place. Contractures may exist in the muscles of the face, eyes, and neck, and hysterical torticollis closely simulates that of organic type and may last for several months (see Fig. 80). Some of the muscles of the tongue may undergo contraction, so that the organ, when protruded, is deviated to one side or the other. Verhoogen[3] reports the case of a boy of twelve who after a slap on the cheek was unable to open his

[1] *Clinical Lessons on Nervous Diseases*, p. 268. [2] *Op cit.*, p. 248.
[3] *Journ. Nerv. and Ment. Dis.*, vol. xxiv. p. 437.

mouth for a month. The masseters were in constant spasm. The patient had other stigmata of hysteria, and his mother was a confirmed hysteric.

Hysterical aphonia is one of the commonest hysterical stigmata. The patient speaks in a whisper, but the cough is usually audible, and in some cases the patient has been known to scream aloud in her sleep. Sometimes the patient speaks in a whisper voluntarily, because the sound of her own voice is distressing to her on account of the hyperæsthetic condition of her hearing.

In many cases there is true loss of power to phonate, and with the laryngoscope it can be seen that the vocal cords do not approximate in

FIG. 80.

Hysterical torticollis (personal observation).

attempts to phonate. Occasionally a patient who cannot phonate is able to sing, and under the influence of an anæsthetic she will speak aloud, although I have seen patients thoroughly anæsthetized without uttering a sound from the beginning to the end of the process. Occasionally the patient may be surprised into uttering a scream by the application of a strong faradic current over the larynx.

Hysterical aphonia may be brought on suddenly by fright or emotion, but it often begins as a complication of laryngeal catarrh, and persists after the laryngitis is cured. In a patient under my care aphonia came on immediately after cocaine had been applied to the gums by a dentist for the purpose of relieving pain in the extraction of some teeth. Hysterical aphonia is prone to relapses, and may last, with short intervals, for several years. The tongue is sometimes affected, so that the patient seems to have lost control of its movements. Hysterical stammering is sometimes met with. It is readily recognized, and differs

from the ordinary forms of stammering in that the patient is prone to repeat the first syllable of various words, and there is no true inability to pronounce words beginning with certain letters, nor are there facial contortions or explosive utterance when the word is pronounced.

In some cases there is marked unsteadiness in standing and walking, dependent upon inco-ordination. The gait and station are markedly ataxic. Mitchell describes this condition as hysterical motor ataxia, and it has also been called astasia-abasia. The inco-ordination does not always depend upon anæsthesia or loss of muscular sense. In some cases the ataxia is so extreme that the patient cannot stand at all without assistance. Sometimes there is a tendency to run backward, as I have seen in a case of Weir Mitchell's several years ago. In other cases the patient reels from side to side as in an exaggerated form of locomotor ataxia.

Reflexes.—Myotatic irritability may be normal or increased. Gowers thinks that it is normal in about one half of the cases. The knee jerks are often exaggerated, sometimes excessively so. A tap upon the patellar tendon or upon the patella itself causes the leg to fly out violently, and often a light blow upon the patellar tendon causes the patient to make a general start and to complain of pain in the back. Sometimes the knee jerks are apparently lessened, and on examination it may be found that the movements of the leg are restrained by contraction of the flexor muscles, either voluntarily or involuntarily. Ankle clonus is seldom found in hysteria, but it can undoubtedly sometimes be elicited. Sternberg says:[1] "In hysterical persons frequently increase of the tendon reflexes occurs, and in about 20 per cent. foot clonus is present." It is more often met with in traumatic hysteria. A pseudo-clonus is not so rare in hysteria. When the foot is suddenly flexed, as in an attempt to induce clonus, it undergoes a series of movements of the extensors, slower than true clonus and more irregular. While the foot is held in a state of flexion they sometimes cease entirely, and after a few moments begin again. These movements give the impression to the observer of being partly voluntary.

The patellar reflex is never entirely absent in hysteria, unless by chance it be in an individual in whom this reflex is abnormally defective. The tendon reflexes of the upper extremities are seldom exaggerated. The plantar reflexes may be excessive or normal. Spasmodic contractures often occur in hysteria; that is, a joint may become flexed or extended rigidly for a few minutes at a time and relax again. The contractures often come on after the "grand attack," or they may come spontaneously. Contractures sometimes affect the muscles of the jaw, so that the mouth cannot be opened, and it is said that occasionally the mouth is held open, so that it cannot be closed—as after yawning, for instance. The arm is sometimes violently flexed at the elbow, with flexion of the wrist and fingers like the late contractures of hemiplegia. The contractures may disappear after hypnosis. Clonic contractions of the diaphragm sometimes occur, giving rise to rapid breathing or hiccoughs or explosive utterances. Rapid breathing is not uncommon in hysteria, and respirations of 50 or 60 a minute are sometimes kept up for days. Hysterical tetany is sometimes seen.

[1] Quoted by Spiller: *Journ. of Nerv. and Ment. Dis.*, July, 1897.

Tremor is often present in hysteria. It may coexist with paralysis and contracture, or quite as often occurs without connection with either. In cases of hysterical paralysis and contracture the tremor often comes on when the limb is moved or if the patient attempts to make a movement. It is usually rapid, quick, and fine. In some cases there is a general tremor affecting all of the limbs and lasting with wonderful persistency. The tremor of hysteria is not increased by voluntary effort like that of multiple sclerosis. Sometimes the tremor is confined to one arm, and seems to keep up as long as the patient is observed. Occasionally placing the limb in some unusual position arrests the tremor, and often, if the patient's attention is diverted, the tremor is lessened or ceases. The tremor sometimes takes the form of rhythmical movements, and is choreic in character, constituting hysterical chorea or chorea major. The movements are quite different from Sydenham's chorea, being more purposive and more irregular. These movements are sometimes seen in an hysterical patient who has been associated with a case of chorea, and cease when separated from it. In some cases the movements are irregular, shock-like, and erratic, like those of so-called electric chorea. In all cases the movements are increased by attention to them. Tremor is frequently a symptom of traumatic hysteria, and is an important feature in medico-legal cases. In a case examined by me in which the patient was suing for damages for injury there was an excessive tremor of both arms. There was also paresis of one arm, grossly exaggerated knee jerks, and scanning speech. The patient was non-suited, and within a short time after I am informed that he returned to a normal condition. The tremor may be rapid or slow, the rapid tremor being eight or ten vibrations per second, and the slow five and a half per second. An attempt to restrain the tremor by holding the part usually increases the severity and range of the movements. Toxic influences, like alcohol, lead, and mercury, may be the cause of tremor in hysteria. Other hysterical symptoms may be caused by metallic poisoning. Tremor may affect the larynx, giving rise to peculiar rapid vibrations of the organ. I have seen a case, which I believe has been reported by Weir Mitchell, in which the larynx moved up and down with great rapidity, giving rise to a clicking sound like the chattering of teeth in a chill.

Visceral Symptoms.—Hysterical vomiting is occasionally met with, and is a most difficult symptom to control. It may last for years, during which time every particle of food is apparently vomited, and yet the patient keeps fairly well nourished. In other cases, however, the patient becomes extremely emaciated and may die for lack of nutrition. The vomiting of hysteria is easily accomplished and is unaccompanied by nausea. Often it seems to be merely regurgitation of the food soon after it is taken, and sometimes the food does not reach the stomach before it is regurgitated from the gullet. The symptom is then called œsophagismus. Anorexia nervosa, or hysterical repugnance to food, is present sometimes to such an extent that the patient will swallow almost no nourishment, and at times the ability to swallow seems to be lost. A patient came under my care who for a long time had been unable to swallow except after the application of a weak solution of cocaine to the fauces. Hysterical dysphagia may be a dangerous symptom from the

difficulty in taking enough nourishment to sustain life. The cases of "fasting girls" which have been reported from time to time have been instances of hysterical anorexia, but in all of these cases there has no doubt been a greater or less amount of deception practised. Various forms of dyspepsia are present in hysterical patients. The most common of these are associated with flatulence and discomfort after eating, with occasional weight or pain in the epigastrium which may simulate gastric ulcer. Sometimes an enormous amount of gas accumulates in the stomach and intestines, which distends the abdomen to an extraordinary extent. This condition constitutes the so-called "phantom tumors" sometimes seen in hysteria. Constipation is frequently present, and the bowels in some cases may not be opened for weeks. There seems to be distinct loss of muscular power in the intestines, for large doses of purgatives and copious enemata have no effect, and cases are on record where the peristaltic action of the bowels seems to have been reversed. A case is reported by Briquet in which tincture of litmus was vomited twelve minutes after it had been injected into the bowel. It is quite a frequent symptom in hysterical patients for an evacuation of the bowels to be followed by a sense of extreme exhaustion or by even actual fainting. Mitchell[1] describes at length the various forms of hysterical disorders of the rectum which he had observed.

Retention of urine often occurs, and the use of the catheter may be necessary for weeks. It is well, however, to avoid the use of the catheter, because when it is once begun it is difficult to leave it off. Incontinence of urine is rare, but it does sometimes occur, as seen in a patient referred to above. Suppression of urine is an occasional symptom, and cases are on record in which not a drop of urine was passed for twelve days, and even longer, without symptoms of uræmia. This is not incredible, because I have seen a case of calculous anuria in which no urine was passed for eleven days, and no symptoms of uræmic poisoning developed. I have recently seen a case of an hysterical girl who passed no urine for ten days, but this patient was not absolutely free from a suspicion of deception. On the other hand, enormous quantities of limpid urine are sometimes passed by hysterical women.

The action of the heart is often disturbed; there are palpitation and irregularity of action. Attacks simulating angina are sometimes seen, and the pain and other symptoms closely resemble those of true angina pectoris. Giraudeau[2] speaks of the frequent coexistence of hysteria and cardiac affections, especially in those suffering from mitral stenosis. I have frequently observed this myself.

Slight causes, like emotion and indigestion, will make the heart beat with such rapidity as to greatly inconvenience the patient. Various vasomotor symptoms occur. The skin flushes easily and in irregular patches, or flushing may alternate with great pallor. The extremities are usually cold, and the surface of the body is often blue and mottled. In some cases there is such a bloodless condition of the skin that pin-pricks do not bleed.

Rapid respiration has been already alluded to. Lloyd[3] gives a case of a woman who, after a surgical operation, had respirations of 75 per

[1] *Clinical Lectures on Nervous Diseases, especially in Women.*
[2] *La Semaine médicale*, June, 1895.
[3] *Op. cit.*, p. 127.

minute for several weeks. Mitchell,[1] reports a case in which the respirations were 150 per minute. Reisman[2] details two cases of hysterical rapid breathing in children: one was a case of chorea in a boy of nine years in whom the respirations were 64 per minute, and the other a boy of thirteen years with respirations of 84 per minute and other manifestations of hysteria.

Coughing is sometimes an annoying symptom. The patient barks persistently, but seems to suffer little or no ill effects from it. Occasionally a peculiar and long-enduring form of yawning is met with in hysteria.

In some instances there is elevation of temperature, the febrile reaction being constant, or it may be intermittent and may last for several weeks; as a rule, however, the temperature is normal in hysteria. In some cases there have been observed extraordinary rises in temperature —to 115° F. These high temperatures have been explained on the theory of fraud on the part of the patient, who makes use of friction or other means to cause the mercury to rise in the thermometer, but there are cases which have been carefully observed in which high temperatures have been recorded, and the question of fraud has been clearly excluded.

Local sweating of the arm, leg, or one side of the face is sometimes observed. Trophic changes may occur in the skin or joints. Bloody sweating and local hemorrhages under the skin are probably always of artificial origin, and the hæmoptysis which is occasionally seen in hysterical women is no doubt caused by injury to the gums. Some curious forms of skin disease occur in hysteria. Mitchell has reported a case in which a bark-like growth appeared on the leg. A patient seen by me, after profuse sweating of the legs lasting for many months, had an extensive eczematous-like eruption covering one leg from the knee to the ankle. In other cases different forms of skin disease of apparently hysterical origin have been reported.

We have attempted to record most of the prominent stigmata of hysteria. No doubt many have been omitted, for their name is legion, and it would be impossible to record all that may be met with in various cases. We must remember that many symptoms met with in a case may be intentionally simulated, but this simulation is only part of the disease and due to the peculiar mental condition of the patient.

Cerebral Symptoms.—The cerebrum is undoubtedly the seat from which arise the disordered bodily functions in hysteria, such as paralysis, hemianæsthesia, and contractures, and it is therefore to be expected that mental disturbances are to be found in different degrees and forms in all cases. The psychical symptoms of hysteria are various in their manifestations. When the functions of the higher cerebral centres are deranged conditions of somnambulism, trance, catalepsy, and like disorders may be manifested. Hysterical patients are easy subjects for hypnotism, and in some cases the patient appears to be in a continuous hypnoid condition. Such persons are easily impressed and readily influenced by suggestion. It is a question whether any person who is readily influenced by hypnotism is in a sound mental state. The practice of deep hypnotization of hysterical subjects is not without danger, for the patient may be left in a hypnoid state for some time after the experiment. I

[1] *Amer. Journ. Med. Sci.*, March, 1893. [2] *Philada. Polyclinic*, Feb., 1897.

would therefore advise against the use of hypnotism in hysterical patients, except with the greatest caution, and then only by an experienced person. Lloyd[1] describes the hysterical patient as living in a sort of a state of double consciousness, forming two groups of phenomena—one constituting his own personality, and the other an abnormal personality, different from the first and ignoring it. Cases of double consciousness and double personality have been recorded in which the person loses his identity and his own original character for periods of months or years. The cases in which the individuality and mental characteristics of the patient vary from time to time, like "Dr. Jekyll and Mr. Hyde," are probably cases of hysteria. In hysteria the patient may have distinct mental aberration. There are delusions, hallucinations, and fixed ideas. The fourth stage of the "grand attack" is usually characterized by delusions, delirium, and sometimes maniacal violence. After the motor symptoms of this stage are over the mental symptoms may continue for a long time in a modified degree. The patient may attempt suicide or be possessed of a mania to set fire to the house in which he lives.

Lloyd[2] speaks of the importance of distinguishing cases of paranoia from hysterical insanity, but undoubtedly the impulse to arson and other destructive ideas do exist in hysterical insanity, being often prompted by the idea of creating a sensational impression. Hysterical symptoms are sometimes present in insanity, but this should not entitle the condition to be called hysterical insanity. The mental disorder in hysteria sometimes takes the form of erotic actions and speech, and symptoms of sexual excitement may be present.

The question of restraint in a hospital or asylum is always a difficult one in cases of hysterical insanity, but there is no doubt that many of the more extreme cases of hysterical insanity require restraint in a hospital, although, as a rule, these cases can be better managed by removal from home and the careful, judicious attendance of nurses in a private house.

Some interesting confessions of hysterical patients have been recorded by Mitchell in which the patient, after recovery, reviews her conduct during the hysterical period. One patient said: "Looking back over the year with the light of the present, it seems as if there was nothing the matter with me, and because of these sensations I carried on a sort of starvation process, physically and mentally."

Diagnosis.—The ordinary forms of hysteria are easily recognized, especially if mental symptoms be present. The stigmata of hysteria are so constant and consistent in most cases that careful search for these will usually reveal the presence of one or more, such as anæsthesia, contracture, perversions of various special senses, and the like. Do not begin the investigation of a case with the idea of fraud and imposture; and although many of the manifestations in hysterical patients are downright frauds, these are, as a rule, merely the outgrowth of the disease. In many cases the diagnosis is difficult, especially when there is also present some organic disease. Many cases of brain tumor have been recorded in which the symptoms of the organic disease were masked by the conspicuous hysterical symptoms. Hughes-Bennett,[3] quoted by Mills, relates

[1] *Op. cit.*, p. 129.

[2] *Op. cit.*

[3] *System of Medicine by American Authors*, vol. v. p. 258.

the case of a wayward and hysterical girl of a neurotic family which had been pronounced hysteria by some of the highest medical authorities in Europe, and after death a tumor as large as a hen's egg was found in the brain. Many other cases of brain tumor have been recorded in which the existence of the growth was never suspected during life. I have seen a case myself in which the symptoms during life were those of hysteria. The patient had many mental peculiarities which had existed during the greater part of her life, and later developed greater mental aberrations with convulsions which were hysterical in type. The patient died after a violent series of convulsions, and the autopsy showed a large tumor in the occipital lobe. Careful and frequent examination of the eye-ground and inquiry as to pain in the head, vomiting, and local palsies will usually prevent the mistake being made of overlooking a brain tumor. When the question is between hysteria and an organic brain lesion, the history of the case will usually aid in the diagnosis. The existence of anæsthesia and its distribution are also characteristic of hysteria. In organic paralysis the reflexes are greatly exaggerated, and there may also be clonus. In hysteria the knee jerks may also be exaggerated, but clonus is rare, and when it is present it is slow and halting. In cerebral syphilis the diagnosis is more difficult, as the paralysis may come and go, and there are usually some psychical peculiarities resembling those of hysteria; but the history of the primary lesion and the presence of skin or bone lesion will usually point to the nature of the disease. Hysterical paralysis is seldom as complete at any stage as that of organic origin. Hysterical paralysis is more liable to occur on the left side, while hemiplegia from hemorrhage or embolism is more frequent on the right. In paraplegia of hysterical origin there is likely to be anæsthesia and rigidity of the legs in extension. In organic diseases of the cord there are bedsores, muscular atrophy, and incontinence of urine. In poliomyelitis there is rapid wasting of the limbs, and loss of contractility to the faradic current is invariably present. When in doubt as to the true nature of the disease, the question can almost always be settled by anæsthetizing the patient. The anæsthetic should be pushed rapidly, so as to produce the stage of excitement and resistance. If the case is one of hysteria, the patient while in a state of ether intoxication will often snatch the towel from the face with the hand which had been before helpless, or will throw about with activity the legs which had been apparently perfectly powerless. In a case of hysterical paraplegia following removal of the ovaries, the patient's legs were anæsthetic from the hips down, and she was unable to move them in the smallest degree. Irritation of the sole of the foot or an attempt to flex the leg would bring on a violent tremor of the limbs, which lasted for several minutes. There was grave doubt as to whether there was not myelitis, for there was incontinence of urine and a distinct bedsore formed over the sacrum. The presence of several hysterical stigmata aroused my suspicions as to the true nature of the case, and ether was administered to the patient. When partially anæsthetized she became quite active in the use of her legs, which were thrown about in every direction with great energy. One should study all cases carefully, and should not be hasty in making the diagnosis. Mistakes are frequently made, and one should always remember what has before been said in this paper, that organic disease

often exists coincidently with symptoms of hysteria. The sex and age of the patient are always to be taken into consideration in making the diagnosis, but it is important to remember that hysteria does exist in men with the typical anæsthesias and other stigmata of hysteria which are seen in women.

Hysteria and neurasthenia are often confounded. It is true that often both conditions may exist in the same patient, but as often they do not. They are thoroughly different diseases, and each has a distinct syndrome of symptoms. Neurasthenia is essentially a disease of malnutrition. The patient is usually thin and anæmic; there are feebleness of body, bad circulation, and lack of energy. Hysteria is essentially a psychosis. In it there are present globus hystericus, anæsthesia, ovarian tenderness, and paralysis. Neurasthenia occurs as often in men as in women; hysteria is most common in women. Neurasthenia never begins or recovers suddenly; hysteria often does so. Hypochondriasis differs from hysteria in that it occurs mostly in men and is a condition of introspective speculation.

Epileptic convulsions may be mistaken for the "grand attack" of hysteria, but a careful study of the seizure will generally prevent a mistake. In epilepsy the attack is sudden; there are biting of the tongue and complete unconsciousness. The attack seldom lasts more than a minute or two, and the patient usually sleeps profoundly as soon as the attack is over. In hysteria the attack usually comes on gradually; the movements are more violent, the patient throwing herself about, and assuming the various positions of opisthotonos and the like, which have been described above. The patient does not bite the tongue, and it is usually found that the conjunctivæ are sensitive, so that when the balls of the eye are touched the lids contract. An hysterical attack is of longer duration than an epileptic attack. The changeableness of the symptoms of hysteria are characteristic: there may be anæsthesia one day and paralysis the next. Above all, the mental attitude of the patient is to be taken into consideration.

Prognosis.—Hysteria is a disease of long standing, and is essentially chronic in its nature. The danger to life in hysteria is practically nil, although cases occasionally do arise in which death results from exhaustion after prolonged vomiting or from some intercurrent disease. Hysterics do not starve themselves to death, although they apparently come dangerously near to it in some cases. In the famous case of the Welsh "fasting girl," who, when a watch was put upon her, died for want of nourishment, it was then found that her parents had been in the habit of supplying her with food surreptitiously. Gowers[1] speaks of death having occurred from laryngeal spasm in an hysterical attack.

The question of complete recovery in hysteria depends largely upon the treatment employed and upon the opportunities for properly managing the case. Many cases recover fully and completely, and remain well; other cases never regain their normal health, but fall into a state of chronic invalidism, in which they remain for the rest of their lives. The contractures become greater and the other stigmata more pronounced. Contractures and local spasms are symptoms which usually last for a long time. Local pains, and especially the tender and irrita-

[1] *Op. cit.*

ble spine, may endure for years. Paralysis and aphonia—and the convulsive attacks as a rule—yield more promptly to treatment. In all cases the prognosis depends upon the surroundings and the individual peculiarities of the case.

Should a definite cause be found which is removable, then the treatment of the case is plain and usually satisfactory. Should there be a bad condition of the general health, with anæmia and loss of flesh, improvement in these conditions is usually accompanied by relief of the hysterical symptoms. This, however, is not always the case, for, should the environment of the patient continue unwholesome, she may gain in flesh and color, but have as pronounced hysterical symptoms as before. Strümpell[1] asserts that in hysteria, as a rule, treatment either produces the most brilliant results or else it has no effects at all. This is, to a great extent, true, but failures in the treatment of hysteria are often the fault of the physician, who undertakes the management of a case when he knows he is handicapped by the unfavorable surroundings of his patient.

Treatment.—The moral and psychical management of hysteria is of greater importance than any other method of treatment, and this must be considered in every plan which is pursued. By far the most successful scheme in the treatment of hysteria which has been devised is that which is known as the "Weir Mitchell rest cure." By this method we can apply various means which are essential for the cure of the disease. The rest treatment enables the physician to isolate his patient and to remove her from the baleful influences which always exist at home. It gives him an opportunity of obtaining a control of the patient without the counter-influences of well-meaning but injudicious friends. It affords the best means for improving the patient's physical health, and removes her from the cares and excitements which are unavoidable in the ordinary home life. Another important influence of the rest treatment is the fact that the prospect of release from the monotony of the treatment and isolation when recovery takes place affords the strongest possible incentive to the patient to make every exertion to help herself. Suggestion, which always plays a most important part in the treatment of hysteria, can be used to the greatest advantage when the patient is under the controlling influence of only the doctor and nurse. It is essential that the physician should gain the ascendancy over the patient's mind, but he must free her from the idea that anything is done in the treatment as a punishment. In order that rest treatment shall be successful every detail should be carried out with the utmost care. The selection of a nurse is of prime consequence. A nurse may be well trained and thoroughly efficient in general illness, but yet be totally unfit for the care of an hysteric. The nurse should be possessed in an unusual degree of tact, and should have resources at her command for diverting and occupying her patient. She should be firm and, at the same time, gentle and kind. There are no cases in which it is so necessary that the "suaviter in modo" should be combined with the "fortiter in re."

Massage is a necessary adjunct to the rest treatment. Not only does it give the much-needed exercise to the muscles, keeping up their nu-

[1] *Practice of Medicine*, p. 813.

trition, as well as promoting the process of digestion, but it makes a break in the monotonous daily life of the patient. It is not desirable to have the massage given by the nurse, but to have a trained masseuse come daily to give the treatment. The masseuse, as well as the nurse, should have special qualifications for her work, and, as Lloyd says,[1] she can often aid in the influence of suggestion upon the patient's mind. Electricity is not so important as massage, but it serves a useful part. I have shown elsewhere[2] that a rise in temperature from one half of a degree to one degree F. follows electrical treatment. Faradism is the form of electricity to be selected, but frictional electricity has its uses in the treatment of anæsthesia and in irritable spine. The length of time for which the patient is to be kept at absolute rest must depend upon the individual case, but it is wise to get the patient up and about as soon as possible. One matter of the greatest importance is not to allow the patient to return to her daily life as soon as she is convalescent. Relapses in hysteria occur readily, and are usually harder to cure than the original attack.

The points of chief importance in the rest treatment are rest, combined with passive exercise, over-feeding, and isolation. While absolute seclusion is not essential in every case, there are but few cases in which we can make an exception. In order to secure proper isolation it is necessary that the patient should be removed from her own home. Few cases are successfully treated at home. The patient should be sent to a quiet boarding-house or properly conducted sanitarium, where the diet can be regulated according to the views of the physician. A good nurse is selected, who is required to remain with her patient all the time, except for a period not exceeding two hours in a day for exercise and fresh air. She should leave her patient at the time when the masseuse comes to give her treatment. The patient is directed to remain all the time in bed. She may be allowed to lie on a cot or lounge which is brought alongside of the bed once or twice a day when the bed is made, but these are the only times when she should get out of bed. The diet at first should be confined to milk. It is not well in any case to ask a patient whether she can drink milk, because so many persons have the idea that they cannot take it. If milk is ordered in small quantities and at short intervals, it is seldom that one finds a patient who cannot, by degrees, be made to take a sufficient quantity. As a rule, it is well to begin with four ounces of milk every two hours, and to increase the amount by one ounce each day until eight or nine ounces are given. At the end of this time, if the patient begins to desire solid food, the intervals between the times for taking milk may be increased and the ordinary meals may be given, beginning at first with very light and easily digested food, such as a lamb chop for breakfast with a cereal, roast or broiled chicken for dinner, with some green vegetables, and supper consisting of stale bread and cooked fruit. By degrees the variety and amount of each meal should be increased, and the patient should be encouraged in every way to eat not only as much as she desires, but a great deal more than she has the inclination for, milk being still given with meals and between meals. When the patient first wakes in the morning a glass of hot milk or cocoa should always be given, and the last thing at night milk in some form should be taken. Malted milk or Nestlé's food

[1] *Op. cit.*

[2] *Mitchell's Nervous Diseases in Women.*

may be given as a substitute for milk in some cases. Massage should be applied once a day in most cases, but a second application is sometimes advisable. Massage should never be given either immediately before or immediately after a meal. There should always be an interval of an hour or two between massage and a meal. Electricity should be applied four or five hours after massage, its object being to give passive exercise to the muscles and to promote circulation. It is not well that one form of passive exercise should follow the other too closely. The following is the usual daily schedule for the average rest treatment: On first awakening the patient should take a cup of hot cocoa or milk, about nine ounces, and immediately after this she should be given a sponge bath, either hot or tepid, followed by friction with a rough towel, and the other parts of the toilet should then be made. The breakfast should now be brought, with which two ounces of liquid malt extract should be taken and a glass of milk. After breakfast the patient should have absolute rest for an hour, not talking or having any reading aloud. About ten o'clock a glass of milk should be taken, and about eleven massage may be administered. As soon as it is finished another glass of milk should be taken, and the patient should try to sleep for an hour, or, at any rate, to remain quiet for this length of time. Dinner should be eaten at about one-thirty, and this meal is to be followed by an hour's quiet. At about 3.30 P. M. another glass of milk should be taken, and shortly after this electricity may be given. At six-thirty the supper should be eaten, and at half-past eight the patient should prepare for the night. After making a slight toilet the nurse may give light massage for fifteen or twenty minutes, then give the patient a glass of milk or hot malted milk, and she should settle to sleep for the night. This routine should be kept up with such modifications as the physician may find necessary, depending upon the peculiarities of the individual, for six or eight weeks. If possible, the patient should be weighed once a week, so that one may judge accurately of the amount of gain in weight. If no scales are in the house where the patient is, one has to judge by the general appearance of the patient, and the amount of food, especially the amount of liquid food, is to be regulated accordingly. At the end of six or eight weeks, if the patient has shown distinct evidence of improvement, she may be allowed to sit up for a few minutes each day, gradually and systematically increasing the time; and, as a rule, when sitting up begins the patient may be allowed to read to herself or to do some needlework or to exercise the mind in different ways. It is not, as a rule, well to admit visitors until the patient is so far well that she is sitting up for a couple of hours twice a day and beginning to walk about her room. The amount of weight gained in such cases is often very surprising, and in patients who are already sufficiently stout the change in the color of the skin, the temperature of the hands and feet, and the firmness of the muscles is most satisfactory. In cases of hysteria with gastric symptoms, such as anorexia and vomiting, the feeding by the tube is of the greatest importance. The stomach should be washed out with tepid water, to which may be added sodium bicarbonate or an antiferment like boracic acid or thymol, and then, before withdrawing the tube, the nourishment which it is intended to give may be poured into the stomach. It is very seldom that this is not retained.

This method of feeding is advantageous, because a large amount of nutriment can be placed in the stomach without the patient's knowledge of the quantity or character of the food; and this is often of great importance. A notable example of the benefit of this method of feeding is that of a young woman who was recently under my charge. She had hysterical anorexia and various other neurotic phenomena. She had become extremely emaciated from lack of sufficient nourishment, for she took only a small quantity of food, and if she felt any discomfort after taking it, she insisted on the use of the stomach-tube to empty the stomach. The patient was twenty-five years of age, five feet five inches in height, and in December, 1897, weighed but sixty-four pounds. She was given eight ounces of milk every two hours during the day, and every morning the stomach was washed out, and there was then poured into it one pint of milk, two raw eggs, and two ounces of beef-juice. The patient had previously declared her inability to digest either eggs or beef-juice, so she was not allowed to know what was given through the tube. In June, six months after treatment was begun, she weighed a hundred and twenty-four pounds, a gain of sixty pounds, or an average gain of a pound and a half a week during her treatment. Her other hysterical symptoms practically vanished.

Travel, change of climate, and sea-bathing are beneficial in cases where the patient will not or cannot take the "rest" treatment. Mills advises against high altitudes in cases of hysteria. Hydrotherapy is of great use in the treatment of hysteria. Not only does it have a marked mental impression, but the douches and sprays have a most beneficial effect on the circulation and general strength of the patient. It is necessary to bear in mind that the baths and douches should be of but short duration. Too long a time in the douche is apt to be followed by a depressing reaction. In the hysteria of children the patient should be at once removed from school, and, if possible, sent away from home to the country or seashore with a nurse.

In the treatment of the "grand attacks" various means have been used with more or less success. Nitrite of amyl by inhalation sometimes cuts short the seizure. Apomorphine, gr. $\frac{1}{10}$, hypodermically, has been recommended in the milder attacks; stopping the respiration by holding the nose and mouth for thirty or forty seconds will often arrest the fit, and dashing cold water over the face and neck will sometimes succeed, but in obstinate cases a cold tub-bath may be necessary. Deep pressure over the ovaries is said to stop a fit, but I have never seen the procedure succeed. Aphonia is often cured by faradism or by drawing sparks from the skin over the larynx with the static machine. Mitchell recommends that a patient with aphonia be instructed to speak only when the lungs are filled after a full inspiration. I have often used this method with striking success. Ovarian tenderness usually disappears after careful and skilful massage. The tenderness in the ovarian region is probably not in the ovary, for hysterical men sometimes complain of pain in the same region. Sometimes a succession of small blisters cures the pain in this locality when all other means have failed. Operations upon the ovaries are seldom justifiable. A number of cures of hysteria have been recorded after removal of the ovaries, but the result was probably due in every case to suggestion. Some of the worst and most intractable

cases of hysteria which I have seen have followed removal of the ovaries. In cases of extreme dysmenorrhœa, associated with severe hysterical attacks at every menstrual period, which are not relieved by a proper course of rest treatment, the operation of oöphorectomy is a perfectly legitimate procedure.

Contractures are often obstinate in yielding to treatment. Sometimes after anæsthesia they are completely relaxed, and the application of a splint to the limb will prevent further return of the contracture. In cases of long standing, however, tenotomy is necessary. Mitchell has recorded a remarkable case in which extensive contracture existed, which was relieved by repeated tenotomies, and the ultimate result was most satisfactory.

Drugs are seldom required in hysteria except to fulfil indications which may arise. It is an incontrovertible fact that there is no drug which is a specific in hysteria. Chloral, bromides, opium, and the coal-tar preparations should be avoided, as they are positively harmful. In cases in which the patient cannot be removed from home some drugs may be used for their moral effects. The valerianates and asafœtida are the most efficient drugs which we have at our disposal, and they certainly seem to be beneficial in some cases. Patients, for example, who come as out-cases to dispensaries improve under the use of remedies. Many cases require iron and arsenic for the anæmia which may exist. Constipation demands serious attention in almost every case, and it is usually most troublesome to manage. Cascara sagrada is a useful drug for this condition, and the well-known combination of aloin, belladonna, and strychnine is sometimes better. In some cases a pill of the following prescription,

℞. Extracti physostigmatæ, gr. $\frac{1}{6}$;
Extracti belladonnæ, gr. $\frac{1}{6}$;
Extracti nucis vomicæ, gr. $\frac{1}{4}$.—M.

given three times a day, affords tone to the intestinal canal, and with the aid of an enema or a glycerin suppository the bowels may be made to move regularly.

Dermographism (Autographic Women).

A number of cases have been recorded under this name in which the skin has the peculiar property of preserving for some time marks which may be made upon it. Any marks or lines drawn upon it rise up, greatly enlarged, as a distinct welt, and remain in that condition for some hours. The experiment has been made of writing with a blunt instrument or the finger-nail upon the skin, and after a few minutes the writing has been shown in marked relief, attaining a thickness of from one and a half to two millimetres. The lesion may last from an hour or two to several days, and the elevated lines are either rose-colored or white. Dermographism has been observed principally in hysterical women, but it has been found also in others in whom there was no suspicion of this taint. This peculiar susceptibility of the skin is regarded by dermatologists as a form of urticaria, for in many particulars it resembles this affection. It is principally interesting because of the

sensational uses hysterical persons have made of the peculiarity when they found that they were possessed of it. The affection has been fully described by Dujardin-Beaumetz and Bartholémy (Fig. 81).

FIG. 81.

Hysterical dermographism (Bartholémy).

HYSTERICAL ŒDEMA.

Œdema is an interesting stigma of hysteria. It is often met with in connection with hysterical joints and contractures. It is generally unilateral, although exceptions to this rule have been noted and a generalized form has been described. The appearance of the skin which is the seat of hysterical œdema is sometimes white, as in ordinary dropsy; sometimes rose-colored or red; but most frequently it is a bluish or violet color. The color usually remains the same in different attacks, even if it be located at different points. The swelling is always hard, and the pressure of the fingers makes little or no impression. Punctures do not give exit to serum. In most cases the œdema affects the entire circumference of the member, but in some cases there is a localized swelling. The temperature of the surface may be normal or lowered in the white and blue varieties of œdema, but in the rose-colored and red forms the temperature is elevated. There are general sensory disturbances in connection with œdema, and Charcot has observed loss of thermal sense. The œdema in these cases decreases and increases under the influence of the patient's general condition, and may vary with the contractures or paralyses which are present in connection with it.

The DURATION of the affection may be as long as two or even five years.

The DIAGNOSIS of hysterical œdema is not difficult when other hysterical symptoms coexist. Of course when the swelling is rose-colored and is about a joint rheumatism is suggested. The absence of high temperature and the comparatively small amount of pain present excludes rheumatism. Hysterical œdema has been mistaken for phlegmasia alba dolens, but this error should not be made if the history and symptoms are carefully observed. In some cases of hysteria certain phenomena are present which are suggestive of Raynaud's disease. The extremities may become white and cold, or may change to a purple color, as in the latter disease.

THE DISORDERS OF SLEEP; ECSTASY AND TRANCE; CATALEPSY.

The Disorders of Sleep.

Introduction.—"Blessed is the man who invented sleep," said Sancho Panza; and in this sentiment agrees every one who has experienced the benefit of sound and refreshing sleep. Nothing is so essential to vigorous and normal mental and bodily health as sleep, and no one can long maintain natural conditions without an adequate amount of sleep. Many persons imagine that they can do without the proper proportion of sleep, but sooner or later outraged nature will show the result of the deprivation of its natural and most efficient restorative. Individuals vary as to the amount of sleep required according to age, sex, occupation, and a variety of other circumstances. Some persons require less sleep than others under similar conditions, and appear to suffer but little inconvenience from the loss of it. Napoleon was said to have taken but four or five hours' sleep nightly, but it is to be borne in mind that he had also the faculty for falling asleep almost at command, and was said to have the capacity for sleeping under almost any conditions and circumstances. It is told of him that, after spending days and nights in preparation for a decisive conflict, he has been known repeatedly to fall asleep in the midst of the uproar of battle and get a few moments' rest in this way. Habit has much to do with the amount of sleep demanded by the individual. A person may accustom himself to take ten or twelve hours of sleep in the twenty-four without any apparent effect on his general condition. Brain-workers, as a rule, need fewer hours of sleep than persons whose life is one of bodily exertion and activity in the open air. The newborn child sleeps most of the time, provided it is healthy, and wakes only long enough to take nourishment. As the individual grows older less sleep is required. According to T. Crichton Brown, the amount of sleep required at different ages is as follows: At four years, twelve hours; at seven years, eleven hours; at nine years, ten and a half hours; at fourteen years, ten hours; at seventeen years, nine and a half hours; at twenty-one years, nine hours; after this, seven or eight hours; and in old age five or six hours are all that are necessary. In cold climates the inhabitants take far more sleep than in temperate or warm countries. The conditions of life in the frigid zone, however, no doubt have much to do with the amount of sleep indulged in, for in temperate climates the hours of sleep taken by an individual in winter do not exceed those taken by him in the summer. Women generally require more sleep than men, although this is not by any means always the case. The length of time for which a person can be entirely deprived of sleep is about the same as that for which he can go without food—namely, about three weeks.

Normal sleep is a state of physiological unconsciousness in which the whole body falls into a condition of functional rest, while nutritive activity is uninterrupted. Nor are the functions of the brain entirely in abeyance during sleep, for we have undoubted evidence that there are certain mental operations which continue during even the deepest

sleep. Dreams are evidence of this, and some writers assert that no natural sleep is ever entirely dreamless. Even in the lower animals there is probably a certain amount of brain activity during sleep, for it is not uncommon to see a hunting dog, while lying before a fire asleep, make short barks and some involuntary muscular movements, as if in his dreams he were in active pursuit of a fleeing rabbit. That there is unconscious cerebration during sleep, as well as in waking hours, is shown by the fact that frequently an individual falls asleep while making an effort to recall some fact, and on waking in the morning he finds that the circumstance has come clearly to him. A remarkable instance of brain activity during sleep is the experience of a professor of mathematics in the University of Pennsylvania who had been working for days to solve an intricate problem, and had almost given up in despair. One night, after hours of hard study over the problem, he dreamed that he had worked it out by a method which had never before occurred to him. On waking the dream was so distinct in his mind that he took pencil and paper and wrote out the problem and its solution with entire satisfaction.

Various theories have been advanced as to the physiology of sleep, but most of them have been abandoned. One of the oldest was that of venous congestion, but this is not tenable, because when congestion of the brain is artificially produced sleep does not occur. At one time the prevailing view as to the cause of sleep was that there was a condition of anæmia of the brain. This idea was the more readily accepted because it was found by experiments on animals that during sleep the blood-supply to the brain was lessened. In 1860 on trephining the skull of dogs Durham found that when the animal was asleep the blood-pressure was much diminished. In man the opportunity has also frequently been presented of examining the cerebral circulation during sleep through openings in the skull which have been caused by trephining for disease or injuries. The degree of anæmia in sleep, however, is only slight, and in cases in which the blood-supply is greatly diminished by hemorrhages or through operations which have cut off the greater part of the cerebral circulation drowsiness has not always been present. We then are forced to the conclusion that the anæmic condition of the brain which is found during sleep is the result, and not the cause, of sleep. The true cause of sleep is no doubt some nutritive change in the cells of the cortex. Some writers believe that these cells become surcharged with effete material during the working hours, and thus the senses are benumbed. It is more likely that the changes in the nerve cells are the direct effect of fatigue. Experiments have shown that long-continued activity, either bodily or mental, causes distinct changes in the nerve cells. Hodge in his interesting observations on birds demonstrated that after great muscular effort the nerve cells showed, under the microscope, distinct nutritive changes, vacuoles appearing in the cells. After rest, and especially after sleep, the cell recovers its normal state, but if the fatigue has been too great and the nutritive changes too pronounced, the cell never recovers. It has been suggested that there is a sleep centre in the brain which when acted upon tends to inhibit consciousness and thus bring about a somnolent condition. The recent theories as to the movements of the neuron would assist largely in the

explanation of the phenomena of sleep were it proven, or possible to prove, that the neuron really possesses the power of contractility. In post-mortem examinations of the nervous system the dendrites of the nerve cells are never found in contact with those of other cells. They may be very near, but do not actually touch. It has been suggested that in a condition of functional activity the cell processes are prolonged until they touch those of other cells, but if through some agency the processes are retracted and no longer communicate with others, then function ceases. In the same manner, when the dendrites of the cortical cells gradually retract a condition of somnolence comes on and sleep occurs.

Hypnotism.

Hypnotism, or *artificial sleep*, is a morbid mental state produced in susceptible persons by certain methods. In hypnosis various phenomena are present. There may be complete suspension of consciousness, the subject being placed in a deep sleep. The most important feature of this state is that the subject loses his own volition and falls into a condition of suggestability, so that his actions are wholly under the control of another person—that is, the operator. While some of the faculties are insensible to external impressions, others seem stimulated to an exalted degree of sensibility, and the mind becomes capable of intense concentration about some idea or feeling. The individual seems to have a dual personality, each personality being quite distinct from the other. In some cases of hysteria the same condition of double consciousness exists. This state has been explained by the theory that there is in the individual a subconscious realm which is capable of reasoning and directing many actions without the knowledge of the principal consciousness. After awakening from the hypnotic state the patient has no recollection of what has occurred while in that condition. About 20 per cent. of ordinary individuals are susceptible to hypnotic influence. Children are easily hypnotized, but after the age of forty-five persons are, as a rule, insusceptible. Sex makes comparatively little difference, although it is considered by some authorities that women, and especially those who are hysterical, are most easily affected. Persons of nervous or imaginative temperament, and more especially those who are accustomed to obey and have been used to discipline, are more readily brought under the influence of hypnotism. In France, Charcot found many patients in La Salpétrière who could be hypnotized. Indeed, the studies made by Charcot of hypnotism in this institution are the most elaborate and complete that we have. The insane are difficult to hypnotize, and many hysterical patients are also insusceptible. After a person has been hypnotized one or more times he is more easily influenced. In some cases three or four attempts must be made before the patient is brought into a state of hypnosis, and many persons can never be hypnotized. In those who have been frequently hypnotized the merest suggestion is often sufficient to put them into this state, and persons may go into this condition by an effort of their own.

Methods.—Various methods of inducing hypnotism have been used. Most of these depend upon impressions made upon the sensory impulses of the body. Among the older methods employed were light pressure

on the eyeballs, making convergence of the eyes upon some near object, sudden or monotonous impulses upon the auditory nerve by sounds, and friction or stroking the skin of different parts of the body. Charcot made use of sounds in causing hypnotism, and even the ticking of a watch was found sufficient to produce the phenomena in some cases. Richet used passes and mesmeric strokes. It is probable that all of these plans acted through suggestion. Many of the lower animals can be readily hypnotized. Some of the marvellous feats performed by the fakirs of India may be explained on the hypothesis that they are done under the influence of hypnotism.

At the present time there are practically but two plans of inducing hypnotism: one is what is called the "fixation method," and is quite old, and is that which was employed by Braid and Heidenhain and also by the Paris school, of which Charcot was the head; and the other is the "suggestion method," which is identified with the Nancy school, represented by Bernheim, Liegeois, and Beaurnis. In the first method the patient is made to gaze fixedly at some bright object, which is held six or eight inches from the eyes and slightly above the line of vision, for several minutes. The convergence of the eyes in the upward position is an important feature in inducing hypnotism, and it is said to be capable of inducing hypnotism in the blind.[1] After the eyes have been fixed steadily on the object for five or six minutes passes may be made with the hand over the face several times, and the subject is asked to open his eyes. If he can do so, the process is again repeated in the same manner as before. An apparatus is sometimes used for the fixation method in which, by means of clockwork, a number of small mirrors are made to revolve before the patient's eyes. This apparatus does not seem to possess any special advantages over the ordinary plan of having the patient look at a bright object. In the suggestion method the operator sits before the patient, and he talks to the latter in a monotonous and confident tone of voice. He tells the subject that he is going to sleep, and talks to him somewhat in this strain: "Think of nothing but that you are going to fall asleep. You are going to fall asleep, and will dream pleasantly. Your eyelids are growing heavy; you cannot keep them open. Sleep is coming over you. You feel tired. Your limbs become heavy. You will soon be unable to open your eyes. Now you cannot open your eyes. You will soon be asleep. You are now asleep." After a few minutes of this talk the patient actually falls into a sound slumber, usually maintaining the sitting position, or he may incline to fall from the chair and have to be supported to a recumbent position. Many operators use a combination of both of the above methods. The patient is made to look fixedly at a bright object, like a new coin or shiny button, and at the same time suggestions of sleep are made by the operator in a quiet but firm tone. Both methods may be aided after a time by gentle passes of the hand over the face, the motions being always in the same direction, and occasionally gentle pressure is made on the eyelids with the fingers. The duration of the hypnotic sleep varies, but if left alone the patient wakens spontaneously after a time, usually after one or two hours, with no remembrance of what has happened. The patient can be aroused by stroking

[1] Mills: *System of Practical Medicine*, vol. v. p. 322.

the head, turning a current of air upon him, or simply by the suggestion of the hypnotizer that he should wake. Self-hypnotism may be induced by individuals fixing the attention on some object. This is usually possible only in persons who have been frequently hypnotized by others.

The hypnotic state has been divided by Charcot into the lethargic, cataleptic, and somnambulistic conditions. In the first the patient remains in the position in which he fell asleep. There is no change in pulse, temperature, or respiration. The pupils react to light and the reflexes are unchanged. There is loss of sensibility to pin-pricks, and in deep hypnotic sleep various surgical operations have been performed without the knowledge of the patient. Teeth have been extracted, and even amputations of the arm have been done, without any recollection of pain during the operation. During hypnotic sleep there is a condition of excessive irritability of the muscular system. Frequently a light touch upon the motor-nerve points will cause active contractions in the muscles supplied by them. While in the hypnotic state the patient can be made to obey various commands of the operator. He does not appear to see or hear any persons who are in the room with him, but if it be suggested by the operator that he sees, feels, or smells anything, he shows by his actions and expression that he does so. A condition of automatism is developed, and not only are the mandates of the operator obeyed, but the patient repeats the words and movements made by him. If ordered by the operator to do so, he will answer another person or do what he is directed to do by him. I have seen a case of hysterical paraplegia in which the patient when hypnotized rose from the chair at the command of the hypnotizer and walked across the room: it was asserted that she had been unable to stand for many months. At the suggestion of the operator the patient may be thrown into the cataleptic or somnambulistic state. These states are, as the names imply, either a condition of catalepsy, or the patient may move about and perform acts under the command of the hypnotizer as if awake, but unconscious of what he is doing.

The degree of hypnosis varies under different conditions, depending, to some extent, upon the subject and also upon the desire of the operator. There may be only slight drowsiness or profound sleep, but in either state the patient responds to suggestions. The patient comes out of the state of catalepsy or somnambulism by falling again into a sleep, and is aroused by the command of the operator. During the hypnotic state the patient often exhibits remarkable acuteness of the special senses. The color sense may be abnormally sharp and hearing phenomenally sensitive.

There is no doubt that frequent repetitions of hypnosis have deleterious effects upon the individual. In hysterical cases the patient is often extremely exhausted after a hypnotic séance. Not only this, but it tends to make the patient imaginative, weak-minded, and nervous.

Pathology.—Many views have been held as to the condition of the nerve centres during hypnosis. It seems probable that in hypnosis there is a condition which is allied to that of hysteria. Charcot believed that there was a disordered condition of the nerve centres in all persons who were susceptible of hypnosis, and it undoubtedly is true that in all cases in which a profound degree of hypnosis has been induced there

must be some abnormal condition of the cortex. There must be a difference between the condition of the brain of a chicken which has been hypnotized by causing convergence of the eyes upon a chalk-line, and that of the cortex of an individual who has been put into a condition of hypnotic somnambulism by suggestion.

The condition of hypnosis is often assumed, particularly by the subjects of travelling mesmerizers who give public exhibitions. The fraud can usually be detected by using strong faradic currents, by suddenly pricking or burning the skin, or by observing the absence of the facial expression which is peculiar to those in the true hypnotic state. The effect of pressure upon the motor points is also absent in impostors. In true hypnosis a limb can be held in an extended position for a long period without tremor, but in fraudulent cases the limb soon becomes tired and trembling will be observed.

Hypnotism is of comparatively little value in therapeutics. Great results have been claimed for it, but in a practical way its use is limited. Many cases of hysteria, in which it would apparently be a useful remedy, cannot be hypnotized. In La Salpétrière many successful results of treatment by hypnotism have been reported, but the class of patients in that institution is unique. Most of them are old hysterics and neurotics, ready to be influenced by suggestion, and are anxious to lend themselves to any plan which would give them notoriety. In this country hypnotism has been exploited by a few writers as a successful means of treatment in insomnia and in morphine and alcohol habits; but it is singular, if it were of such value in these disorders, that it has not come into general use, or at least found favor in the eyes of some of the leading neurologists. It is certainly an unsafe means of treatment, and may act like a boomerang, not only leaving ill effects in the patient, but may also bring discredit upon the operator; for the public is liable to look with some suspicion on an agent of this kind, and it has not infrequently occurred that charges have been brought against the operator of improper conduct toward his patient during the hypnotic state. It is also unwise to encourage the use of any agent by which one person gains such an ascendancy over the mind of another that by a command he can be made to do almost anything. It is to be remembered that suggestion always plays an important part in therapeutics, and that this may be considered a mild form of hypnotism. There is a medico-legal side to hypnotism which must be borne in mind, for the question has been raised as to whether a person may be compelled under the influence of hypnotic suggestion to commit a crime. In a number of instances persons have claimed irresponsibility for crimes which they have committed, on the plea that they had been hypnotized by another who had compelled the deed. Gray[1] refers to a recent Parisian trial in which it was claimed that a man named Eyraud, who had strangled his victim, had hypnotized his accomplice in crime, Gabrielle Bompard.

INSOMNIA.

Insomnia, or ahypnosis, is that condition in which a person suffers from partial or complete inability to sleep. Sleeplessness depends upon

[1] *Nervous Diseases*, p. 503.

a number of causes, and may be either functional or dependent upon some organic brain disease.

Etiology.—Heredity is a common cause of sleeplessness, and no individual peculiarity is oftener transmitted than disorders of sleep. In persons past sixty the amount of sleep required is only one half of that needed by the youth of twenty, but in the elderly there is often a condition of inability to sleep long or consecutively, which then amounts to an abnormal state. In persons using the brain actively in business or professional work there is often great difficulty to secure enough sleep. Indeed, it is exceptional rather than the rule for such workers to sleep enough to repair the effects of fatigue upon the nerve cells. The causes of functional insomnia may be external or internal. Those from without may be noises, bright lights, strong odors, etc. A person may be accustomed to sleep in a considerable amount of noise, provided it be regular, but if the noise stops he awakes. A fair example of this is sleeping in a railway train. If the train stops and there comes that unearthly stillness which is so striking when a train comes to a standstill at night, the sleeper is sure to awake. Internal causes of sleeplessness may be digestive, toxic, circulatory, and nervous. Various diseases induce insomnia by the discomfort produced by them. Neuralgia, for example, may keep the patient awake, and diseases of the skin which cause intense itching will drive away sleep.

Digestive Causes.—It is well known that eating heavily at night will cause wakefulness or disturbed sleep, and so will various forms of indigestion, especially those associated with flatulence. It is also true that too long abstinence from food will interfere with a person's going to sleep, and often, if a meal has not been taken for five or six hours before retiring and the patient is unable to fall asleep, a little food will enable him to go to sleep.

Toxic Causes.—Insomnia may be caused by auto-intoxication, such as gout, lithæmia, uræmia, and allied conditions, and various drugs also produce wakefulness. Tea, coffee, and tobacco are among the articles of daily use which, when taken immoderately or by those unaccustomed to them, cause sleeplessness. In lead-poisoning there is often wakefulness. Cocaine, caffeine, and quinine are also productive of wakefulness. Alcohol, when taken in large quantities, produces somnolence, but when taken to such excess as to cause mania-a-potu it brings about the most violent form of insomnia. Many acute diseases, like influenza, pneumonia, typhoid fever, and also other infectious fevers, are ushered in by painful wakefulness. In malarial diseases the patient is often unable to sleep. Indeed, there are but few infectious or diathetic diseases in which there is not more or less sleeplessness, either at the onset or during the course of the disease. In secondary syphilis there is often persistent insomnia, which, however, is always relieved by proper medication.

Circulatory Causes.—In heart diseases and aneurysm the sleep is usually bad, and in arterial fibrosis, in which the blood supply to the brain is lessened, sleep is poor and disturbed. In anæmia and chlorosis there is often a distressing condition of somnolence by day and wakefulness by night.

Nervous Influences.—The most frequent causes of insomnia are to be

found in the nervous system, either through disorders of the central nervous system or as a result of psychic disturbances. In insanity, especially in melancholia, wakefulness is marked. In neurasthenia it is one of the most troublesome symptoms with which we have to deal. Mental care, anxiety, and grief will drive away sleep effectually, and will bring about a condition in which sleep is unattainable even by drugs.

Reflex Causes.—Insomnia may be the result of intestinal worms and teething in children. Adhesions of the prepuce or clitoris may also cause disturbances of sleep, either directly or indirectly. Other reflex irritations in the intestinal canal or in the pelvic organs will also cause insomnia.

SYMPTOMS.—In insomnia the patient may fall asleep as soon as he retires, and after from one to three hours wake, and for the balance of the night he will be unable to close his eyes until near morning, when he may sleep for an hour or two more. Some patients may sleep until quite late in the morning after a wakeful night, but they usually awake without feeling any sense of rest. In other cases the patient finds it impossible to go to sleep on first retiring, and, after threshing about his bed for hours, may fall asleep after getting up and moving about the room or after taking some nourishment, and sleep for a few hours. In other cases the patient sleeps for an hour or so, and lies awake for the same length of time or longer at intervals throughout the entire night. In young children insomnia not infrequently occurs, contrary to the statement of some writers. Sometimes the insomnia depends upon irritation of the teeth or some digestive disturbance, but in many cases the cause is undiscoverable. The child seems to be wide awake and to be perfectly well, but is simply filled with a desire for play and amusement. Hood's "Lullaby" gives a vivid picture of the wakeful infant. In the sleeplessness of neurasthenics there is much mental activity, and the patient does not sleep because he is continually thinking over business and other affairs. If he does sleep, he dreams continually, and he wakes in the morning unrefreshed. In melancholia the patient usually wakens at three or four in the morning, and is unable to again go to sleep because of the harassing thoughts which come into his mind. He usually lies quietly in bed until it is time to rise. In mania and paresis the insomnia is accompanied by intense restlessness: the patient is in and out of bed or is walking about his room the greater part of the night. In all cases of insomnia there is more or less mental irritability, and the patient looks careworn and is in a bad state of bodily nutrition. He is unequal to prolonged mental activity, and after the insomnia has lasted for some time there is almost always mental deterioration of some kind, although some persons get along for many years with what seems like an incredibly small amount of sleep. It is probable that in all cases the patient sleeps more than he is aware of, and, at any rate, the hours of long rest in bed and a condition of partial somnolence afford to some extent a substitute for sleep.

TREATMENT.—In almost every case of insomnia we encounter the difficulty that the patient wishes merely some remedy which will enable him to sleep and pursue the same habits which he has been following. In all cases there is some underlying cause, either constitutional or the

result of the mode of life, which must be corrected before the insomnia can be relieved. Therefore, in every instance treatment must first be directed to restoring or improving the general health by suitable regimen, diet, and exercise. Of course any diseased condition must be treated specially. In order to be cured of insomnia every patient must make up his mind to a life of self-denial and self-discipline, and he must pursue a regulated course of treatment for some time before he can expect permanent results. He must retire at a reasonably early hour, and should rise at a sufficiently early hour. A person who gets into the habit of sleeping until nine or ten o'clock in the morning is almost sure to have trouble in getting to sleep early, and, while we cannot say that the hours of the early part of the night are more advantageous for sleep than the morning hours, the conditions for sleeping in that period of the night are usually much more favorable than in the morning. The patient should not eat late suppers nor should he go to bed with the stomach empty. A glass of hot malted milk at bedtime or a couple of ounces of liquid malt extract, with a piece of bread, can be conveniently obtained and will often promote sleep. The bedroom should be cool and well ventilated, and the bed itself not too soft; the feather bed —which has, fortunately, almost gone out of fashion—should never be used. The coverings of the bed should be light, but at the same time the patient should be sufficiently warmly covered, especially the feet. Often it occurs that a person awakens when the legs have been accidentally uncovered, because the chilling of the surface causes contraction of the capillaries and sends more blood to the head. It is of the greatest importance that the patient should not read or study in the evening. Writing is particularly apt to arouse the mental faculties, and the best way of passing the evening is either in conversation or in some game which requires exercise, like billiards. A very hot bath before bedtime conduces to sleep, but hydrotherapy is not always successful in such cases. A warm bath frequently awakens the patient more thoroughly, but a cold bath or spinal douche is better, as the reaction after this is greater. The douche should be of short duration, two or three minutes being long enough. The drip sheet is sometimes useful in procuring sleep, but this requires the help of an assistant, who must give a brief rub-down after it. Massage at bedtime is one of the best means of obtaining a long sleep, and general faradization is sometimes equally successful. After the preparation for the night has been made the patient must divest himself of all business thoughts and try not to think too deeply of subjects which are interesting. Occasionally reading a dull book at bedtime or having a book read aloud after retiring is useful, but it often makes a person more wakeful. Repeating poetry or counting or thinking of some monotonous subjects, like the well-known expedient of thinking of sheep jumping over a stile, will often bring about a condition conducive to sleep. It is most important for persons who are sleepless not to use tobacco in the evening. The patient should always take a sufficient amount of exercise in the open air to induce moderate fatigue. In other words, he should endeavor to substitute muscular fatigue for brain tire. Horseback or bicycle riding, golf, and tennis are particularly suitable forms of exercise in insomnia. The diet should receive strict attention. An abundance of nourishing,

easily digested food is indicated in all cases, but rich, highly seasoned, or stimulating food is to be avoided. The heaviest meal of the day should be taken not later than 2 P. M. Milk or other liquid nourishment may be taken between meals if the patient is under weight. Chopped or ground meat, made into cakes and broiled, served with butter and lemon-juice, is palatable and easily digested. Mutton, fowl, and eggs may also be taken. Fruits of all kinds may be eaten with advantage, but in the evening fruit should be cooked before it is eaten. Among vegetables the most desirable are baked potatoes, green peas, beans, lentils, onions, celery, spinach, and greens of all kinds. Lettuce has on many persons a decidedly soporific effect. Whole wheat bread eaten with plenty of butter is advisable. As much fatty food as can be digested should be taken. The articles of diet to be avoided are condiments, pickles and spices, all indigestible food, such as pastry and fried things, tea, coffee, cocoa nibs.

Drugs.—The first thing to be said about drugs is not to prescribe them if possible. Many drugs are considered harmless and unobjectionable, but no drug can be sufficiently potent to induce sleep without leaving some ill effects behind, and there is no way in which a drug habit is more easily formed than in the use of such drugs as relieve sleeplessness. Many persons will secure sleep after taking a stiff drink of whiskey or beer at bedtime, but soon the quantity of these stimulants has to be increased, so that before long an injuriously large amount is required to produce any result. One man whom I knew had been taking whiskey to procure sleep for some years. At the time he came under my care he was consuming just one gallon of whiskey a week, taking it only at night. Should the use of a drug for procuring sleep become unavoidable, the best medication I know is one of the bromides administered during the day at stated intervals. The most eligible way of giving the drug is to prescribe fifteen grains of the sodium or strontium bromide in a bitter infusion after each meal. After taking this two or three days the patient begins to sleep better, and will usually get from six to eight hours of sleep a night without taking any additional drug at bedtime. As a rule, this amount of bromide may be taken for several weeks without any ill effects, but generally it is best not to continue it longer than two weeks. As a hypnotic the bromides given at bedtime are not sufficiently strong and their action is too slow. Chloral hydrate is one of the most effective of all the drugs which we have as a hypnotic. Fifteen to twenty grains of this may be given at bedtime, and one half of the amount may be repeated in two hours if sleep is not induced. It is not always a safe remedy, and its effects must always be watched. Moreover, the chloral habit is one which is formed easily and with difficulty broken. A combination of fifteen grains each of sodium bromide and chloral hydrate, with half a drachm of tincture of hyoscyamus, is more effective than chloral alone. Chloralamid is a milder drug than chloral, but it is much safer and is often very useful as a hypnotic. It should be given in doses of from twenty to thirty grains dissolved in an ounce of sherry wine at bedtime. Paraldehyde is one of the most certain narcotics which we have at command, but its taste is exceedingly obnoxious, and it leaves behind a disagreeable odor of the breath which lasts the whole of the next, and sometimes the second, day succeeding a dose. It may be

given in tincture of orange-peel, compound tincture of cardamom, or other aromatic tincture, as this makes it miscible with water. The dose of paraldehyde is from a half drachm to two drachms at bedtime. The paraldehyde habit is not a common one, but I have seen it two or three times. Amylene hydrate is milder and less effective, but it is not disagreeable to take and has no ill after-effects. A drachm of this may be administered at a dose and repeated if necessary. Sulphonal is an excellent narcotic, and usually it is followed by no unpleasant effects. The dose is from ten to thirty grains dissolved in hot water or broth, or it may be given as a finely divided powder upon the tongue and washed down with cold water. It seems to take about two hours before the desired effect is reached, and it should therefore be given early in the evening. Trional is much like sulphonal in its action, but seems to do better in some cases than the latter, and is often effective in a smaller dose, say, ten grains. Both of these drugs are liable to be followed by dulness the next day and with a tendency to sleep on the second night, so that it is usually not necessary to give a dose two nights in succession. Hyoscine hydrobromate in doses of one one-hundredth of a grain is valuable, especially in mental cases. It is a great advantage that it can be given in water without the knowledge of the patient. It sometimes has a cumulative effect, unpleasant dryness of the throat and dilatation of the pupils resulting. Chloralose is a new hypnotic, and I have used it in few patients with the effect of securing prompt sleep, but in nearly every case there was some mental disturbance after it had been taken for two or three successive nights. Valerian, lupulin, compound spirit of lavender, Hoffmann's anodyne, and lactucarium will suffice to secure sleep in some cases. An elixir of the valerianate of ammonia was in great repute in this city some years ago as a safe and harmless narcotic, but it was found that it contained morphine. Opium in no form should be used as a hypnotic. The opium habit is easily formed, and it is an undesirable hypnotic, because it is so often followed by headache and disordered stomach. In certain cases of active, maniacal insomnia morphine is necessary for its generally quieting effect.

In many cases change of climate and travel will effect a cure when all drugs and most careful regimen have failed.

Somnambulism.

Synonyms.—Noctambulation ; Sleep-walking.

Definition.—Somnambulism is a state of abnormal cerebration which takes place during sleep, and is analogous to the hypnotic state of double consciousness.

Symptoms.—In somnambulism a person when sound asleep performs a series of complicated actions which require the assistance of all the senses. Such persons will get up while asleep and walk about the house, sometimes going on steep roofs and other dangerous places. They may walk through the streets, lanes, and fields, and return to bed without knowing anything of what they have done. During the somnambulistic state the eyes are sometimes open, sometimes half open, or they may be shut. The pupils are dilated, contracted, or normal, but are almost always insensible to light. The actions of persons in the state

of somnambulism are sometimes most remarkable. They perform complicated acts, write, make mathematical calculations, and do many things which to a looker-on could only be done by a person wide awake and in full possession of his faculties. Everything that is done, however, is automatic, and nothing is originated in what is done. A remarkable case is related [1] of a person who was found by Dr. Sloane translating Italian and French and looking up words in a dictionary. His candle was extinguished, whereupon he immediately began groping about, although other candles were lighted in the room, and immediately lighted his own candle at the fire. He was insensible of the effect of any candle except the one on which his attention was fixed. Gray [2] quotes a case related by Mesnet of a suicidal attempt made in his presence by a certain Madame —— while in a somnambulistic state. She made a noose of her apron, and fastened one end to a chair and the other to the top of a window. She then kneeled down as if in prayer, made the sign of the cross, mounted the stool, put her head in the noose, and tried to hang herself. Innumerable instances of remarkable feats performed during somnambulism are on record through popular and scientific literature, and some of the performances of sleep-walkers are almost incredible. There is a medico-legal side to the subject of somnambulism which is of much importance. Murders have been committed by persons in a state of somnambulism, and the question as to the responsibility of these persons is a very serious one. Gray [3] refers to a case of a man named Frazer who killed his child in Glasgow. At the trial some remarkable facts were elicited in regard to previous somnambulistic feats. Other cases of homicide by a somnambulist are also on record. Yellowlees records a case.

Etiology.—Somnambulism may be hereditary. Cases are on record in which a father and sons have been sleep-walkers. Over-eating at bedtime, sleeping in an uncomfortable position, especially with the head low, worry, distress, or mental disturbances, are liable to induce sleep-walking. It usually begins in children before or about the age of puberty, but occasionally it develops later in life. It is often seen in connection with hysteria, and I have seen one case in a girl of ten who was suffering from chorea. The disorder occurs sometimes in connection with epilepsy. It seems to be equally common in both sexes, but in adult life is rather more frequent in women. It is undoubtedly fostered and encouraged by the comment and talk made about the condition by the family and friends of the patient. In school-children there is often much talk and excitement about the occurrence of sleep-walking, and the amount of notoriety which it occasions certainly does not lessen the frequency of its occurrence in the subject. I have recently seen a neurotic girl, a Jewess, who dreamed of a skeleton which was threatening to cut off her hair. While still in a sound sleep she secured a pair of nail-scissors, and, with much difficulty with such an implement, cut off the entire plait of her hair.

Treatment.—The surroundings and mode of life of the patient should be carefully inquired into and all unhealthful habits should be corrected. If the disorder occurs in a child at school, he should be at

[1] *Anomalies and Curiosities of Medicine.* Gould and Pyle, p. 865. [2] *Op. cit.*, p. 965.
[3] *Nerv. and Ment. Dis.*, p. 494.

once taken away and put in the hands of a sensible person who can watch and regulate his mode of life. Change of surroundings and of the whole habit of life are more likely to break up the occurrence of somnambulism than anything else. A change to the country or life in camp in the woods for some time is most desirable in such cases. A person should never be wakened in the somnambulistic state, but he should be conducted back to his bed without being disturbed more than possible, and placed in a recumbent position without being allowed to wake. The prognosis in children is generally good, because the habit arises from some reflex cause which can usually be corrected, but if it is persisted in to adult age it is often irremediable. A patient of mine at the age of forty-five had been a sleep-walker from her youth, and her husband was always in the habit of securing the doors and windows of her room so that she could not get out in her sleep.

Dreams and Nightmares.

Etiology.—Dreams occur in natural sleep to a great extent, but generally are so inconsequent as to make no impression and the individual does not remember the next morning what he has dreamed. In abnormal conditions the dreams are so active that they are distressing and wearying. Confused ideas follow each other in rapid succession, and give rise to such distinct impressions upon the brain that horrid pictures are presented before the patient, who soon awakens in fright. External influences of different kinds will give rise to dreams suggestive of the impression which has been made. For instance, the odor of smoke will give rise to dreams of fire, or tickling of the sole of the foot to dreams of torture of some kind. A person often dreams of the same occurrence night after night without regard to the fact that the matter has been one of thought or consideration during the day. In the superstitious various ideas and forecasts are connected with dreams.

Nightmares usually arise from over-eating, and the sensation of oppression from the consequent indigestion gives rise to dreams of a horse standing on the chest or of being crushed by a weight or some other horrible thing. Children often have nightmares and awake crying with fright, but adults are also victims of alarming dreams, and every one knows what it is to awaken out of a bad dream trembling with agitation and with the heart beating like a trip-hammer. Albers, according to Dana,[1] says: "Frightful dreams are signs of cerebral congestion. Dreams about fire, in women, are a sign of impending hemorrhage; dreams about blood are signs of inflammatory conditions. Dreams of distorted forms are frequently a sign of abdominal obstruction or diseases of the liver." Nightmare usually occurs in incomplete sleep, and often an uncomfortable position in which a person is lying induces the attack. Lying on the back is particularly likely to cause nightmares. They come in persons of a nervous temperament, and are liable to be associated with mental anxiety and distress. Some persons have nightmares all their lives. I know a woman of thirty-five, of full habit and nervous temperament, who since childhood has had attacks of nightmare in which she screams loudly and persistently as a result of a bad dream. Heart

[1] *Diseases of the Nervous System*, p. 494.

disease, anæmia, and other morbid conditions which tend to disturbed sleep are accompanied by nightmares.

TREATMENT.—The treatment consists in attention to the digestion and general health, insisting upon light suppers and that no food should be taken immediately before retiring, and the use of expedients which will prevent the patient from sleeping on his back. The common plan of fastening a spool to a strap around the body, so that it is against the back, will prevent a person turning in the dorsal position in sleep.

Pavor nocturnus, or night-terrors, is an exaggerated form of nightmare in children. The child wakes usually one or two hours after going to sleep, screaming with fright and clutching its mother in terror. The eyes are staring, the muscles of the face twitch, and it requires great persuasion and soothing to have much effect in quieting the child. There seems to be no tangible reason for the alarm, but the child is in a state of terror which it cannot explain. The patient will sometimes get up and run from the room in which it has been sleeping. The trouble is usually connected with heredity and poor health; the subjects of it are usually delicate and anæmic children; and the same causes which predispose to nightmares and somnambulism induce attacks of night-terrors. Indigestion, late eating, intestinal worms, over-study, or worry, and especially enlarged tonsils, are all causes of the trouble. The attacks may be associated with or may precede an attack of chorea. The trouble usually yields promptly to judicious treatment. The administration of tonics, and care in diet and in the mode of life, will usually relieve the condition. The administration of small doses of sodium bromide two or three times a day is almost always efficacious in arresting the attacks, and occasionally a tepid bath at bedtime is of value. In some cases circumcision is necessary, but it is not often that attacks can be traced to phimosis. The hour at which the attack occurs —that is, just after the patient has fallen into a deep sleep—shows that the trouble is dependent upon some cause like over-eating or overtaxing of the brain.

Somnolentia, Schlaftrunkenheit, or sleep-drunkenness, is a rare condition in which a person on being awakened from a deep sleep is thrown into a condition of maniacal delirium. He does not recognize his friends or know what he is doing, and may commit acts of violence. The condition has been defined as "an overlapping of profound sleep on the domain of apparent wakefulness, producing an involuntary intoxication on the part of the patient which destroys at the time his moral agency."[1] It seems to be an exaggerated form of pavor nocturnus, and in a lesser degree it occurs not infrequently in many persons. A number of cases have been recorded in which homicidal acts have been committed by a person who has been suddenly roused from sleep. A case is quoted by Wharton and Stillé[2] in which a laborer killed his wife with a wagon-tire, the blow being struck immediately after he had been forcibly awakened from a deep sleep. The defendant declared that on awakening he was seized with a delusion that a woman in white had snatched his wife away from his side and was carrying her off. There was no doubt felt as to the irresponsibility of the patient.

Morbid Drowsiness.—Undue drowsiness may depend upon various

[1] Wharton and Stillé: *Medical Jurisprudence*, vol. i. p. 393. [2] *Op. cit.*

changes in the brain and bloodvessels, and it is also due to certain toxic causes. Uræmia is a very frequent cause of deep somnolence. Drowsiness also accompanies a condition of anæmia and malnutrition. In starvation the victim is inclined to sleep much of the time. In some forms of dyspepsia and in repletion with food it is well known that the individual is dull and drowsy. Exposure to extreme cold also produces drowsiness, and fatal sleep is known to have occurred in persons who have been exposed to very low temperatures. Certain ocular defects which cause eye-strain make a person sleepy when sitting in a bright light. Obese persons are proverbially drowsy, and they fall asleep when sitting in church or at a lecture or under conditions which do not require much mental attention. The close air of a crowded room which is overloaded with carbonic acid makes almost every one heavy and sleepy. In anæmia and heart disease the patient is drowsy by day and wakeful at night. A condition of somnolence often exists in lithæmia and many other diseases. In acromegaly the patient often suffers from unconquerable desire to sleep. Excessively deep sleep occurs in some persons as a normal condition. They cannot be aroused from sleep by ordinary means, and when once awakened fall asleep again immediately. Such persons sleep long, and waken dull and often with headache. Unusual bodily fatigue induces prolonged and heavy sleep. The effect of drugs like opium and alcohol in producing heavy sleep is too familiar to require more than passing notice. Organic diseases of the brain cause abnormal sleep as to length and depth. Brain syphilis in some cases, especially when associated with arterial changes, is accompanied by a tendency to much sleep. The patient may even be in a semicomatose condition. Disease of the thalamus is frequently associated with a tendency to prolonged sleep, the patient sleeping many hours of the day and most of the night. Tumors of the parietal region, of the corpora quadrigemina, the thalamus, and the pituitary body are more commonly accompanied with drowsiness than are growths in other regions of the brain.

Sleeping Sickness; African Lethargy; Nelavan.—This disease is met with on the coast of Africa in the Congo, and in Sierra Leone. It affects negroes exclusively of both sexes and all ages. Corrè[1] says he heard of a European woman who had the disease, and he saw himself a Moorish woman who had it. It is most frequent in males from the age of twelve to twenty. The cause is unknown, but a parasite, the filaria perstans, has been found in the blood of those suffering from the disease. Manson[2] found this parasite in the blood of 5 of 9 cases which he examined, and he thought it might have been present in the others, although he did not find it in the small quantity which had been sent him. The general health of the patient is at first good, but increasing somnolence gradually appears. The patient falls asleep at his work, and when not disturbed will sleep uninterruptedly for hours. The symptoms are sometimes arrested by active purgation and counter-irritation over the spine, but soon the somnolence returns again. In some cases swelling of the cervical glands is an early symptom; as the case progresses the period of sleep becomes longer and deeper; finally, the patient sleeps

[1] Quoted by Manson: *Davidson's Hygiene and Diseases of Warm Climates*, p. 504.
[2] *Op. cit.*

all of the time, and will not even stay awake long enough to take food. Progressive emaciation occurs, and death usually results in four or five months after the beginning of the disease, but the patient may drag on an existence for two or three years. The disease is almost invariably fatal. Forbes[1] has given an interesting and complete account of this affection. He refers to two cases in which the filaria sanguinis hominis was found in the blood. He recommends purgatives and strychnine as the most useful form of treatment, but, as a matter of fact, no treatment seems to be of any avail. Forbes found, post-mortem, hyperæmia of the arachnoid and slight signs of chronic inflammation of the other meninges. The brain substance was paler than normal.

Narcolepsy.—This is a condition in which a person is liable to fall asleep at any time during the day. This term must not be confounded with nocturnal or sleep epilepsy, which is a totally different condition. In narcolepsy the patient has an unconquerable tendency to fall asleep at any time during the day, sometimes for a few minutes, perhaps for an hour or two. It is more common in women than in men, and from the ages of fifteen to forty. It is frequently seen in conjunction with hysteria and other neuroses. It may also be associated with disturbances of nutrition dependent upon obesity, gout, and diabetes. Narcolepsy is frequently associated with dyspepsia. It may be seen in members of neurotic families, and the subjects of it have often exhibited other forms of hysterical disturbances. Two patients reported by Gélineau exhibited multiple tics. A number of cases of narcolepsy reported by Porter alternated with attacks of vertigo or unconsciousness, which is suggestive of epilepsy. I have seen a patient, a young woman, who fell asleep frequently during the day, particularly whenever she attempted to read or sew, and she would not awake for a half hour or longer. When she led an active outdoor life she seldom had attacks of drowsiness or sleep, but if she had indoor work or studies, the attacks were constant and overpowering. An examination of the eyes showed that there was marked refraction error, and this was probably the cause of the condition. Gowers speaks of a similar case in a girl in which the trouble began at sixteen and continued until she was twenty-two. She would suddenly become drowsy during the day, and sleep for several hours, and wake up fresh and bright. Intensely vivid dreams accompanied this sleep, and she would sometimes speak aloud in her sleep.

TREATMENT.—The treatment of narcolepsy must depend to a great extent upon the cause of the trouble. In all cases the general health should be closely inquired into. If anæmia exists, this should be treated, and any other dyscrasia should receive attention. The case to which I have referred shows the importance of examination of the eyes in these cases. Gowers found that caffeine and nitroglycerin were effective in breaking up the attacks in his patient. Change in the manner of living, or even change of climate, may be necessary to effect a cure.

Sleep epilepsy is a form of epilepsy which is characterized by attacks of intense sleep, not accompanied by convulsive movements. The patient falls asleep and cannot be roused. The seizure may be preceded by an aura, and the attack varies in length.

Sleep Palsies.—Under certain disordered systemic conditions per-

[1] *Indian Med. Record*, June 1, 1895.

sons may awake with numbness of the hands and feet. This has been termed "acroparæsthesia," and it may arise from a number of causes. It may be due to anæmia, gout, diabetes, and the excessive use of tobacco, but it is more commonly met with in women at the menopause, as I have shown in a paper on "Numbness of the Extremities."[1] The numbness is most intense on first awakening, and passes off after the parts have been used for a short time. In extreme cases the numbness is so intense and distressing that the patient is awakened from sleep, and has to get out of bed and exercise to get rid of the sensation. It occurs by day as well as by night. The sensation is usually one of prickling and formication, such as occurs when the part is what is popularly called "asleep." At times, however, the sensation becomes one of marked pain, and the numbness is not only confined to the extremities, but extends up the limbs above the elbows and above the knees. In some cases the numbness is monoplegic or hemiplegic, and there is not only the sense of formication and tingling, but there is also loss of touch sense and marked loss of power, lasting for a short time. According to Mitchell, this form is most likely to occur after the excessive use of tobacco. The attacks of sleep palsies may be repeated night after night, and may last for years. Attention to diet and the removal of any toxic causes usually effect a cure in a short time. I have found ergot of signal value in the treatment of these cases.

Sleep Ptosis.—In persons who sleep soundly there is often great difficulty in opening the eyes when they first awaken in the morning. In weak and neurasthenic women the difficulty may amount to true ptosis for fifteen or twenty minutes. The lids cannot be opened voluntarily, but if raised by the fingers they remain open. The affection is usually a transient one, and is almost always recovered from.

Enuresis Nocturna.—Nocturnal incontinence of urine occurs in neurotic children, but it is an accident which occasionally happens to an adult. It is much more common in boys than in girls. In cases of cord disease like myelitis there is often a giving way of the sphincter vesicæ in sleep. In most cases of incontinence the trouble usually occurs a few hours after the patient has retired and when sleep is deepest. The causes of the disorder are obscure. In some cases the kidneys are unduly active, and in others it seems as if the inhibitory centres of the cord are weak, and when the influence of the will is removed the sphincter no longer exerts its automatic control. Sometimes, as the result of a dream in which the person is emptying the bladder, the discharge actually takes place.

The TREATMENT should consist of withholding liquids as far as possible during the latter part of the day, and especially at the evening meal. The patient should be made to empty the bladder at bedtime, and should be made to get up again in two or three hours. In some cases the expedient of raising the foot of the bed three or four inches is successful. This means seems to act mechanically in lessening the pressure at the neck of the bladder, and may have some influence by reducing the amount of blood in the lumbar cord. Belladonna and cantharides are the most valuable drugs in producing a direct influence upon the bladder, but in many of the cases of nocturnal incontinence

[1] *Trans. of the Col. of Phys. of Philadelphia*, 1884.

with which we meet it is necessary to administer iron and other tonics. Spinal galvanism is often useful, and in some cases where other means have failed direct faradization of the bladder by means of an electrode inserted per urethram is successful. Painting the orifice of the urethra with collodion at bedtime has been suggested as a means of arousing the patient before the urine escapes. Corporal punishment, which has been advocated by some writers, is, in my opinion, unjustifiable. It is very rarely the case that a child is not deeply mortified by the occurrence of bed-wetting, and he usually does everything in his power to prevent it.

Disorders of the Prædormitium.—The period of light initial sleep which comes to almost every one before deep slumber has been termed the prædormitium. During this time many persons experience various nervous and muscular disorders, such as fright, twitchings, and shocks. These vary in degree and kind. Often there is a sense of falling and the person wakens with a start. Children are often seen to have muscular twitchings or slight starts as they are falling asleep. Mitchell[1] has written at length on the disturbances of the prædormitium, and has also described certain similar disorders which occur at the end of the night's sleep, "when the drowsied sentinels resume their post"—the interval between sleeping and awakening. This he calls the postdormitium. He describes cases in which the disturbances occurring in the prædormitium are violent and excessive, and he alludes to the relation between these and insanity. He quotes Baillager, who has written fully on this subject, as saying that certain persons, otherwise sound, are liable to have between sleeping and awakening hallucinations which long precede outbreaks of insanity. Some of Mitchell's cases were affected with violent shocks of an explosive nature, and others had sensory disturbances of an aural nature. He classifies these sensory shocks as follows: "I. *In the sphere of general sensation:* The patient feels as if struck or as if he had a shock like that which a sudden arrest of motion causes, or it is a feeling of rending or as of a bolt driven through the head. These are the most common. II. *Auditory:* A loud noise like that of a pistol shot, or a crash of broken glass, or as of a bell or wire sharply twanged. III. *Visual:* A flash of light. IV. *Olfactory:* Sudden sense of an odor. V. I doubtfully add what I call 'emotional discharges.' These are always merely abrupt sensations of fear, sometimes preceding the sense of shock, sometimes following."

Ecstasy and Trance.

In ecstasy the individual is in a peculiar rapt and exalted condition of mind in which impressions are not received in the usual way. The patient is in a visionary state, has a peculiar radiant expression, and is, as a rule, in an immovable position. Various statuesque postures are assumed, the subject sometimes standing with the arms extended like a cross; sometimes in other attitudes expressive of devotion, fear, love, or lubricity. Most of the instances of ecstasy that are met with are in hysterical subjects, and the condition often alternates with catalepsy and hystero-epilepsy. Extreme religious feeling is one of the most common exciting causes, and religious education carried to the extreme predis-

[1] *Clinical Lessons on Nervous Diseases*, p. 58.

poses to the development of ecstasy. Fear or a fright has been known to cause an attack of ecstasy. The fakirs of India are probably as typical examples of ecstasy as exist. The positions of devout worship which they assume, and in which they are said to remain for days apparently totally oblivious of all surroundings, are most remarkable. Many interesting cases of ecstasy have been recorded, especially in France, and the best known of these is that of Louise Lateau of Bois d'Haine, a village of Belgium. In 1868 the patient, who was a peasant-girl of twenty-three years of age, noticed one Friday that blood was flowing from the left side of her chest. This was found to be repeated on every Friday, and on each Thursday morning a spot an inch in diameter on the back of each hand became pink in color, and at the same time a similar spot was observed on the palm of the hand. About noon on the same day a vesicle containing clear serum formed on the pink surfaces. In the night, usually about midnight, the vesicle ruptured and a flow of blood began. The amount of blood lost from these stigmata was estimated by some observers as being between one and three quarter pints; the blood itself was a reddish color, coagulating in the usual way, and the white and red corpuscles were found to be normal in character and proportion. The flow of blood ceased on Saturdays. During the flow of blood the patient was in a rapt and ecstatic condition. She stated that at the beginning of the ecstasy she imagined herself surrounded by a brilliant light, and then figures passed before her exhibiting the successive scenes of the Crucifixion. During the period of ecstasy the action of the heart was regular, although at times a sudden flush or pallor covered the face according to the changes of expression. From midnight on Thursdays, when she took a silent meal, until eight o'clock on Saturday morning, Louise Lateau took no nourishment, not even water, because it was said she did not feel the want of it nor could the stomach retain anything. From the forehead of the patient blood sometimes oozed from minute points. While in the state of ecstasy the patient was apparently unconscious to all external impressions. The mucous membrane of the nose was tickled, ammonia was applied to the nostrils, needles, pins, and even penknives were thrust into the flesh, and strong currents of electricity were used. To none of these did she respond, but after the attack she remembered everything which had occurred. It is, of course, a question as to the genuineness of the phenomena presented in this case. Many similar cases have been studied with great care, and under a rigid surveillance the stigmata were found to have been produced by deceit; as, for example, the case quoted by Mills[1] of a girl of sixteen under the care of Mr. Henry Lee at St. George's Hospital, London. This patient had discolored patches on the leg, from which she asserted that every month for two years there had been a discharge of blood. It was found that the patient produced these hemorrhages by thrusting a needle into the skin. In some cases of ecstasy the patient simply falls into a trance-like condition, and none of the stigmata or phenomena of visions, etc. are exhibited. In some cases the condition of trance is so complete and deep that the patient to all intents and purposes is dead. The respirations become so shallow that they can scarcely be observed, and the pulse is barely perceptible. In

[1] *System of Medicine*, vol. v. p. 350.

this condition a person may remain unconscious to all surroundings for days.

A number of cases have been reported, mostly in the public press, in which it is alleged that persons have been buried in a state of trance. It is highly improbable that any such cases have actually occurred, but there are authentic cases in which the patient has been thought dead, and it has been proved that there was only a condition of trance. Mills[1] records a case of which he had personal knowledge in which attacks of trance occurred three times during the life of the patient. Once, while this person was an infant and was about to be baptized, it was observed that he became rigid, and was thought to be dying; after suitable efforts, however, he was resuscitated. Again, when about sixteen years of age he was accidentally wounded by a pistol shot, and he again passed into a trance-like condition. Many years later, while apparently in good health, he suddenly fell into an unconscious condition and was believed to be dead. So marked were the appearances of death that after several futile efforts to resuscitate him preparations were made for the funeral. Persistent efforts on the part of friends who were aware of the previous attacks were finally successful. The fakirs of India are capable of falling into a trance in which all of the vital functions seem to cease, and it is asserted they even allow themselves to be buried alive for several weeks. In these cases it is probable that as a result of long practice the subject is able, by self-hypnotization, to fall into a state of trance in which he remains for a long period, in a condition which has been aptly described as being like that of a hibernating animal. The case of Colonel Townsend, which is familiar to all, may be recalled. This gentleman was able, by an effort of will, to arrest the action of the heart, so that to all intents and purposes he was dead. Then after a period a flush appeared upon the face, a slight impulse was felt over the præcordia, and by degrees the patient returned to a normal condition. On one occasion, however, the experiment proved fatal.

TREATMENT.—The treatment of ecstasy and trance consists mainly in general hygienic measures, and more particularly in placing the patient in a wholesome moral atmosphere. The use of hypnotic suggestion is likely to be beneficial in these cases, as the subjects are easily influenced by this means.

CATALEPSY.

In catalepsy there is a peculiar form of muscular rigidity, associated with a perverted state of consciousness. The limbs retain any position in which they may be placed, and have a wax-like rigidity to which the name "flexibilitas cerea" has been given. There is loss of sensation and complete suspension of normal mental action during the attack. The disorder occurs in both sexes and at all ages, but more frequently in women than in men, and in early adult life. The patients are usually hysterics or have presented at some time other symptoms of hysteria. The attacks are liable to occur after excitement of any kind and after injuries to the head and back. The disorder is quite frequently met with in the insane, but chiefly in those suffering from melancholia. Katatonia is a term applied to stuporous melancholia with cataleptic spasms. In

[1] *Op. cit.*, p. 344.

a case of melancholia under my care a condition of catalepsy lasted for several weeks. The cataleptic rigidity did not relax under full ether anæsthesia. The attack of catalepsy may be preceded by some symptoms of general nervous disturbance. The rigidity comes on suddenly and with loss of consciousness. The limbs retain the position which they occupied at the onset of the attack, as though they were frozen. At first the muscular rigidity is great, and difficulty is experienced in moving or bending the limbs, but soon they can be readily moved and placed in any position. The limbs may remain in one position for an hour or more. If the arm is placed in an extended position, after a time it will gradually yield to gravity. The features are expressionless during an attack; the respirations are slow and the heart's action is weak. The patient swallows if food is placed far back on the tongue. In profound catalepsy there is complete analgesia. Sensation may be lost to the electric current, but this is seldom the case, and the conjunctivæ do not respond to irritation. The temperature of the body is low during the attack. The attack may last from a few minutes to hours, and may continue at intervals of days or even weeks. In the interval the patient may seem like herself or she may have vertigo or be in a condition of general nervousness.

Catalepsy is to be regarded as a form of hysteria, and the TREATMENT is practically the same as should be adopted in an hysterical attack (see p. 710). The application of cold water may arrest the attack, and sometimes strong ammonia applied to the nostrils will abort the seizure. Apomorphine administered hypodermically is an effective remedy.

VASOMOTOR AND TROPHIC DISORDERS.

VASOMOTOR AND TROPHIC DISORDERS.

ACROMEGALY; RAYNAUD'S DISEASE; ANGIO-NEUROTIC ŒDEMA; FACIAL HEMIATROPHY; SCLERODERMA; VERTIGO.

By JAMES STEWART, M. D.

ACROMEGALY.

Synonym.—Pachyacria.

The name acromegaly, introduced by Marie in 1886, means large extremities. As the disease, however, presents symptoms much wider in range than are expressed in this term, it is felt to be an inadequate definition. The word pachyacria (compact or hard extremities) is in a sense more appropriate and distinctive of the actual state, but it also is not sufficiently comprehensive to include the essential changes or symptoms of the condition.

Acromegaly is a comparatively rare disease, not more than 150 cases having been reported up to the present time (1896).

Etiology.—It is a disease of middle life, but it has been met with at all ages. It has been described in children under one year, and as late as the sixty-fifth year. The great majority of cases begin between the twenty-fifth and fortieth years. Some three or four cases have been described where it is probable that it was congenital. It has been seen in the different races of mankind, white, black, and Mongolian.

Nothing is known about the exciting causes.

Symptoms.—The onset of acromegaly is very insidious. The victim is rarely able to give even an approximate idea of the probable time of its beginning, the increase in size of the extremities being so gradual that it has reached a very considerable degree before perceived by the patient or his friends. Frequently the physician is consulted about some other trouble or complaint, the acromegaly being found out accidentally.

A well-marked, typical case of acromegaly presents a very striking clinical picture (Fig. 82).

The gait and posture exhibit certain special characters. In standing and walking the neck and chest are thrown forward, and the head is tilted backward. There is a compensatory lumbar lordosis. There is frequently a lateral as well as an antero-posterior curvature. The walk is heavy and clumsy. The arms hang listlessly by the side, appearing by their weight to drag the upper part of the body downward. The facial

aspect is peculiar (Figs. 83, 84). The thickened eyebrows and eyelids, the enlarged ears, together with the thickening of the maxillary bones, give rise to the remarkable change that takes place in the patient's appearance. The face is enlarged in every direction. The lips are greatly thickened; the lower jaw is thickened and projects. The nose is enlarged. The thickening of the soft tissue destroys the finer movements of the muscles of the face, the muscles of expression. The eyes lose their wonted expression. The nostrils are thickened and broad. The teeth become more separated from increase in size of the maxillary bones. The tongue is often enlarged.

FIG. 82.

Photographed in 1891 (Osborne).

The changes in the extremities are usually the most characteristic. The hands (Fig. 85) and feet become enlarged, often enormously so. The increase affects all the tissues—the skin, subcutaneous tissues, the muscles, and the bones. The wrist-joints are usually more or less enlarged, but there is seldom any change in the tissues of the fore- or upper arms. The great thickening and enlargement of the hands give the upper extremities a spade-like appearance. The feet are also greatly increased in size. The great toe is often found to be increased out of proportion to the rest of the foot. The increase in size of the tarsal bones, especially of the os calcis, gives the foot an odd appearance.

Headache, vertigo, lassitude, and somnolence are early symptoms in

FIG. 83.

Photographed in 1887.

FIG. 84.

Photographed in 1891 (Osborne).

The same patient as shown in Fig. 82.

acromegaly. In advanced cases somnolence is a very marked and persistent symptom. Mental disturbance is not infrequent. The patient at first is simply morose and introspective; later great depression is not uncommon, leading at times to suicidal attempts.

Polyuria and glycosuria are met with. Pigmentation of the skin may be set down as one of the unusual nervous symptoms of the disease. The skin is hard and dry. The hair becomes brittle and falls out.

There is very rarely any hypertrophy of the voluntary muscles met with, except those of the hands and feet. The electrical reactions remain unaltered in uncomplicated acromegaly. The enlarged muscles may undergo wasting after the disease has been in existence for some years. This may be due to the development of some other affection, such as peripheral neuritis, syringomyelia, progressive muscular atrophy, or the wasting may occur independently of any gross change in the central or peripheral nervous system. It is rare to meet with any disturbance of sensation.

There may be actual enlargement of the eyeballs or an apparent atrophy of them from the marked increase in size in the brows and lids. The size and shape of the visual field are often considerably altered; sector, hemianopic, and concentric limitation being met with in different cases. Optic neuritis going on to atrophy is present in a con-

FIG. 85.

Right hand, palmar surface (Osborne).

siderable proportion of cases. Nystagmus and ocular paralysis are rarer symptoms.

Tinnitus and deafness in one or both ears, due to middle- or internal-ear changes, are rarer symptoms than those of the eye above described. Both smell and taste may be found disordered. The speech is slow. The voice usually undergoes marked changes in acromegaly, owing to the thickening of the tissues of the larynx. It is low-pitched and has a disagreeable resonant clang.

Polyuria, peptonuria, glycosuria, and albuminuria are frequent complications.

PATHOLOGICAL ANATOMY.—In a considerable number of cases of acromegaly proving fatal post-mortem examinations have been held, and

exhaustive studies of the changes in the tissues made by men competent by training and experience to carry out such work.

The most painstaking study that I know of is that recently conducted by J. Arnold.[1] The case was at one time fully described by Erb clinically. It was a typical one of the disease. The following changes were found by Arnold, and may be taken as typical of the changes met with in this disease: The skin was found thickened over the entire body. The skull showed marked hyperostosis, and here and there exostosis. The ribs, sternum, and the pelvic bones were found thickened. The long bones of the extremities presented their normal form, but were thicker, heavier, and more compact. The different processes for the attachment of tendons were very prominent. The bones of the hands and feet were increased in circumference and in compactness. Osteophytic growths thickly covered the ends of the phalanges and some of the flat bones, as the patellæ. The periosteum was thickened, and under it and between the narrow Haversian canals were found compact masses of bone.

The soft parts showed considerable increase in bulk. The muscles presented various grades of degeneration. Both the large and small branches of the peripheral nerves showed thickening of the sheaths and increase in the interstitial tissue. Similar changes were found in the spinal ganglia, the sympathetic, and the anterior and posterior nerve roots, especially in the posterior roots of the lumbar enlargement.

In the left temporal lobe a patch of softening was present, caused by the degenerative state of the central arteries, the arteries in general being found degenerated.

The pituitary body was turned into a mass of degenerated tissue (lympho-sarcoma?). The new formation infiltrated the underlying bone.

The chief changes met with in cases of acromegaly may be summarized as follows:

1. Very marked hypertrophy of the ends of the extremities, due to an increase in volume of all the tissues—skin, muscle, bone.

2. Degeneration of the muscles, nerves, and bloodvessels.

3. Changes in the pituitary body. In the great majority of cases changes have been described in this gland.

In a few cases the thyroid and thymus glands have been found either absent or diseased.

PATHOLOGY.—Many different hypotheses have been advanced to explain the nature of this remarkable disease. The frequent presence of changes in the pituitary body has naturally led observers to consider that there is a causal connection between such and the dystrophic changes characterizing acromegaly. Arnold is of the opinion, from his studies in his own and all the cases reported up to the present time, that the pituitary changes are a result, and not the cause—that there is some deep underlying cause which in the present state of our knowledge we are unable to explain. Tamburini has collected 22 cases of acromegaly in which a post-mortem examination was made after death. In 19 changes were found in the pituitary body, and in the remaining 3 the diagnosis during life appeared to be a matter of doubt, the symptoms not being sufficiently marked to be characteristic of the disease. He con-

[1] *Virchow's Archiv*, Band 135.

cludes that morbid changes in the pituitary body are the essential cause of acromegaly.

Although much valuable work has recently been done in connection with the ductless glands (pituitary, thyroid, thymus), we are still wanting in a thorough understanding of the office they perform in the economy. At present it would be premature to speak positively of the relation, if any, between the pituitary changes and the disease under consideration.

DIAGNOSIS.—The following affections bear more or less resemblance to acromegaly:

I. Hypertrophic pulmonary osteo-arthropathy;
II. Leontiasis ossea;
III. Gigantism;
IV. Chiromegaly;
V. Myxœdema.

I. *Hypertrophic Pulmonary Osteo-arthropathy.*—This strange condition is characterized by the occurrence in the course of various chronic pulmonary diseases (tuberculosis, bronchitis, syphilis, empyæma, malignant disease) of a slowly progressive hypertrophy of the hands and feet, while the bones and soft tissues of the face and head remain normal. The latter distinction, together with the absence of any changes in the superficial or deep structures of the eyes, or of any mental disturbance, are the chief points to be relied upon in arriving at a diagnosis.

II. *Leontiasis Ossea or Megalocephaly.*—Only a single case of this condition has been described clinically.[1] The descriptions previously given are founded on specimens met with in museums. The condition consists in a hyperostosis of the cranial bones. In one of Starr's cases there was slowly developed but progressive enlargement of the head, face, and neck, affecting the bones, skin, and subcutaneous tissues, the first to the greatest degree. Numbness and other disturbance of tactile sensibility in the upper limbs was made out. The patient was a female, aged fifty-two, and had been troubled with the above symptoms for six years. No improvement followed the use of thyroid powders.

III. *Gigantism.*—The relation between gigantism and acromegaly is a subject about which little is known. The aphoristic saying of Marie contains some truth—viz. "acromegaly is gigantism of the adult; gigantism is acromegaly of the adolescent."

A certain number of acromegalics are giants. Dana reports the case of a man six feet seven inches in height who died, and there were found changes in the pituitary gland, and another case of gigantism (seven feet four inches) in which there was unilateral facial hypertrophy.

Various forms of hypertrophy have been described, either unilateral or confined to particular parts (arms, legs, etc.), or it may involve a particular tissue only (muscle, subcutaneous).

IV. *Chiromegaly.*—The term chiromegaly was introduced by Charcot and Bressand to describe those cases that resemble acromegaly, and which are most frequently met with in the course of syringomyelia. The enlargement of the extremities is more apparent than real. In all such the tissues ultimately atrophy. The characteristic symptoms (peculiar sensory disturbance) will at once enable one to arrive at a correct conclusion.

V. *Myxœdema.*—The clinical features of myxœdema are so striking

[1] M. Allen Starr: *Am. Journ. Med. Sci.*, Dec., 1894.

usually as to enable the physician to at once differentiate it from acromegaly. The pale, waxy, œdematous-looking skin, the full-moon face, the slow speech, and the marked result of thyroid treatment are entirely different features from those of acromegaly. Further, there is no enlargement of the bones in myxœdema.

PROGNOSIS.—The disease is steadily progressive, the patient usually dying after many years from some intercurrent affection.

TREATMENT.—Many different agents have been tried in acromegaly, but without success. Thyroid, thymus, and pituitary-gland powders have had fair trials by different observers, but with no special favorable result. Bramwell reports 2 cases, 1 treated by thyroid powder with some benefit, while another was benefited by an extract of the pituitary gland. No case of cure of an acromegaly has been reported.

RAYNAUD'S DISEASE.

SYNONYMS.—Symmetrical gangrene; Local asphyxia.

DEFINITION.—A disease of vascular origin, characterized by peripheral ischæmia of varying intensity.

ETIOLOGY.—It is met with at all ages, being, however, much more frequent in middle life than either in advanced life or during childhood. Mendel describes a case that started at nine months. Up to the present time the cases described have been more frequent in females than in males in the proportion of about two to one. In the great majority of cases there is a clear neuropathic history, especially of insanity, hysteria, epilepsy, etc. It is also frequently met in people who are at the time suffering from one of the above neurotic disorders. All diseases tending to lower the constitutional vitality predispose to it, as syphilis, rheumatism, rickets, and anæmia. Among the recognized exciting causes of the disease are the poison of the acute infectious diseases, as typhoid, influenza; traumatism in general and of the central and peripheral nervous system in particular; excessive fatigue, fright, etc.

SYMPTOMS.—It will lead to a clearer understanding of the condition to describe separately the various types of intensity of this disease in the manner followed by Osler in his work on the Practice of Medicine. He describes the following forms:

(*A*) Local syncope;
(*B*) Local asphyxia;
(*C*) Local gangrene.

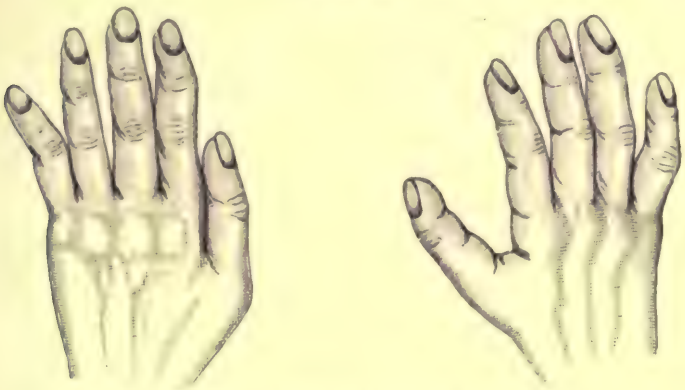
The above forms, although clinically convenient, are only to be understood as types of intensity, and not as essentially different conditions.

In the first form, local syncope, the peripheral parts, as the fingers and toes, after great fatigue or exposure show signs of diminished blood supply. They become white and have a glossy appearance. The parts feel cold to the touch and subjectively also. There is diminution of sensation (tactile, thermic, algesic sensations being usually all more or less interfered with). The local syncope may involve one or two fingers and toes or all. Sometimes the nose and tips of the ears may be the chief or only seat of the circulatory changes.

The condition may persist for a variable length of time—minutes to days, rarely longer than from one to two hours.

As the pallor diminishes and passes away it is succeeded by hyperæmia of the parts. When the reaction is intense, we have an actual local asphyxia, constituting the second or a severer type of the disease. This condition may or may not be preceded by the stage of pallor (local syncope). The tips of the fingers, toes, ears, or nose present a blue-black appearance. It is rare to meet with cases where all the extreme peripheral parts mentioned are simultaneously affected, usually not more than one or two fingers on each hand during the same attack. Asphyx-

FIG. 86.



Local asphyxia of fingers in Raynaud's disease (Henry).

iated patches of skin on the face and on the upper or lower limbs, or even on the trunk, are now and then seen. Swelling and pain of the affected parts attend the discoloration. Sensation is blunted. After lasting a variable time the asphyxia may gradually disappear, the parts assuming their normal appearance.

Occasionally the disease passes on to a deeper stage—the third stage, that of gangrene. Blebs form on the asphyxiated patches. The parts become black, cold, and insensible (Fig. 87). Sloughing takes place; it may be only of a minute point on the pulp of one of the fingers or of a finger, or it may be an entire limb. Rapid and extensive gangrenous destruction of one or more of the extremities is still a further severer type of this disease. Usually, in the severe forms, the disease proves rapidly fatal, although instances are recorded where recovery has followed the loss of two, and even three, extremities.

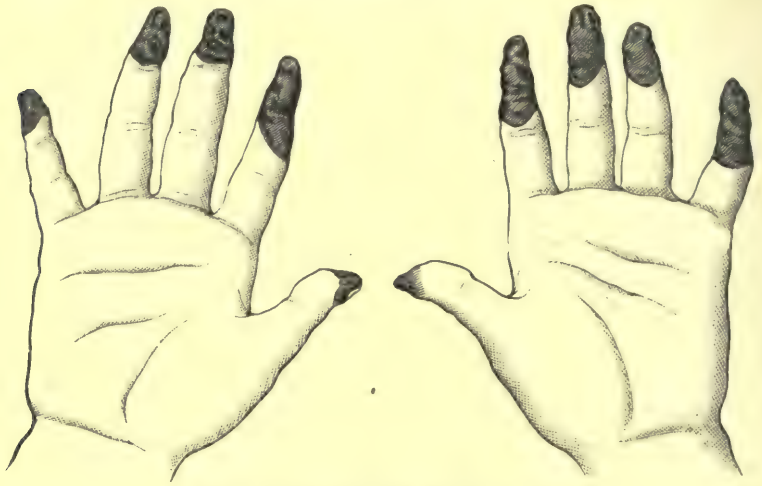
The disease is not attended by any elevation of temperature or other pyrexial signs. There is a feeling of lassitude, and usually considerable mental depression. Occasionally the mental depression may reach a very marked degree. Cases are recorded where maniacal symptoms were present during the attack. Mania may be present with and without delusions. Transient and recurring attacks of hemiplegia, sometimes with aphasia, are also described. Polyarthritis is one of the rarer symptoms met with. Hæmoglobinuria has been frequently noticed during an attack of Raynaud's disease. A remarkable case has been reported by H. M. Thomas from Osler's clinic: Raynaud's disease

occurred for three successive winters, and in association with hæmoglobinuria and epilepsy. Exposure on a cold day would bring on an epileptic attack, with the local asphyxia and bloody urine. I have met with a case associated with Graves' disease, diabetes, and epilepsy in a young female.

Féré and Batigne describe a case of epilepsy in which paroxysmal attacks of local syncope had existed from birth.

Symptoms pointing to disturbance of the special nerve centres have been noticed in a number of cases. Ringing in the ears, deafness, dimness of vision, alterations in the sense of taste and of smell have been

FIG. 87.



Gangrene of fingers in Raynaud's disease (Dehio).

described. Such symptoms are usually ascribed to temporary spasm of the vessels supplying the special nerve centres. Papillary and other forms of disturbance of the sympathetic are rarely met with.

An hysterical form of Raynaud's disease has recently been described by Lévi.[1] He arrives at the following conclusions from his study of the disease:

1. That there is a purely hysterical form of Raynaud's disease.
2. That acute rheumatism often precedes the first attack of the hysterical form of Raynaud's disease.
3. The onset is very sudden, and usually ushered in by some distinct emotional influence.
4. It is commonly attended by either a marked increase or diminution in the quantity of urine.
5. Hypnotism he considers the best therapeutic means. He claims that in this way the psychical factor at the bottom of the trouble can be discovered and by the same means removed.

PATHOLOGICAL ANATOMY AND PATHOLOGY.—Pitres and Vaillard have found neuritic changes in the nerves of amputated parts. It is probable, however, that such changes are of a secondary nature, the

[1] *Archiv. de Neurol.*, Jan.–Mar., 1895.

result of the vascular disturbance in the affected part. Dehio[1] in making an examination of the tissues in an amputated part in Raynaud's disease was unable to find any changes in the epidermis and the rete Malpighii, but he found inflammatory infiltration into the subcutaneous tissues. He also found the arteries and veins of the parts changed; they were the seat of a fibrous endarteritis and endophlebitis. Panas has had the opportunity of making a histological examination of a leg which had been amputated on account of extensive gangrene from Raynaud's disease. He found marked inflammatory changes in the veins and arteries, and degenerative changes in the peripheral nerves, especially in the anterior tibial.

From the course and termination of the disease, and from the fact that it is in many cases a disease of early life, it is clear that the neurotic and vascular changes noted above do not stand in the relation of cause, but rather of effect. It may be said that there is what may be called a physiological syncope of the extremities. All grades of intensity of this condition are met with, from the slightest momentary pallor up to gangrenous destruction. The lighter attacks may be due to simple emotional disturbance or other influence that reflexly acts on the vasomotor circulation. It can be readily understood how repeated attacks will eventually give rise to ever-increasing vascular and neuritic changes.

The local syncope is attributed to a spasm of both arteries and veins, and the local asphyxia to a cessation of the latter and a continuance of the former. Nothing, however, is definitely known as to the ultimate cause and nature of the disease.

PROGNOSIS.—The prognosis is nearly always favorable. It is rare to meet with a fatal issue, and then only in children when the gangrenous stage has been reached.

Slight attacks in the adult may recur at intervals for many years. The rule is that under appropriate treatment they cease to return. If the disease develops secondarily to some profound structural disease of the central nervous system, as tabes dorsalis or syringomyelia, or in the course of chronic phthisis, the prognosis is more unfavorable, due rather to the underlying trouble than to Raynaud's disease itself.

TREATMENT.—The chief indication in the treatment is the removal of the cause or causes which are in operation. Often it is impossible to do this, but it is always proper to use such measures as tend to strengthen the system in general and the nervous system in particular, as a course of hydrotherapeutics and a residence in a mountainous region. During an attack the patient should keep quiet, and the parts affected should be elevated and covered with cotton-wool. The severity of the pain may call for opium in some form. It should not be given unless the attack is a severe one. It should not be used locally. No irritating local applications should on any account be employed, as they serve to increase the tendency to gangrene.

Nitrite of amyl and nitro-glycerin are recommended by some on account of their influence in dilating the peripheral arterioles.

Galvanization of the sympathetic and spinal cord has also been recommended.

In the very mild attacks of local syncope treatment is seldom called for.

[1] *Schmidt's Jahrbucher*, 1894.

ANGIO-NEUROTIC ŒDEMA.

Synonyms.—Acute non-inflammatory œdema; Giant urticaria or swelling; Acute circumscribed œdema (Quincke).

Definition.—Angio-neurotic œdema is a periodically recurring trouble characterized by the sudden appearance of local swellings of limited extent and duration.

Etiology.—It occurs chiefly in subjects who are either suffering from disease of the central nervous system or in whom there is a strong neuropathic tendency. It is especially frequent in Graves's disease, neurasthenia, and hysteria. Direct heredity is a marked feature in many instances. Osler records the history of a family extending through five generations, and where twenty-two members were subject to such attacks. Many other remarkable examples of heredity in this disease have been recorded.

Among the exciting causes the most important are physical and mental exhaustion, the poisonous action of tobacco and alcohol, malarial poisoning, exposure to cold, etc.

Symptoms.—The swelling makes its appearance suddenly, commonly on the eyelids or cheeks. Usually a sense of itching precedes the swelling. The part swollen is usually small in extent and distinctly circumscribed. In color it varies somewhat from a dull white to a distinct rose hue. Although it is most common to meet with the swellings on the eyelids and cheeks, they are also at times seen on the hands, trunk, etc. In one attack one or several circumscribed patches are often met with. The duration of an attack varies from a few minutes to several hours. There is rarely pitting on pressure, and then only when œdema is considerable and has lasted some hours. Swelling of certain of the mucous membranes is frequently met with, and often constitutes the most distressing symptom of an attack.

When the œdema involves the gastro-intestinal mucous membrane, severe colic, nausea, vomiting, and diarrhœa are present. The occurrence of œdema of the larynx may prove fatal. Osler records two instances of this. Transient pulmonary œdema has been described. Among rare symptoms are hæmoptysis, hæmatemesis, hæmoglobinuria, polyuria, and albuminuria.

The time of recurrence varies much in different cases. It may recur every hour, or the intervals may be days or months. In certain cases there is marked periodicity in the time of recurrence.

Pathology.—The disease is looked upon by Quincke, who first described it, as a vasomotor neurosis due to stimulation of the vasodilator nerves. How the different causes mentioned predispose to the condition is not known.

Treatment.—The treatment of angio-neurotic œdema is unsatisfactory. There is rarely any success following the measures recommended for the prevention of the attacks.

The first endeavor should be to discover the presence of any likely predisposing or exciting cause, and, if possible, use measures to counteract or lessen them. Quinine and atropine are recommended as of value in preventing attacks. The general measures mentioned as of value in Raynaud's disease will be found appropriate in angio-neurotic œdema

(p. 747). In the treatment of the œdema during an attack soothing measures are called for. The œdema of the skin rarely calls for anything beyond this, but for the acute pain induced by the œdema of the gastro-intestinal tract morphine should be given.

Œdema of the larnyx may demand scarification or even tracheotomy.

FACIAL HEMIATROPHY.

Synonyms.—Hemiatrophia facialis progressiva; Neurotic atrophy of the face.

Definition.—Facial hemiatrophy is a trophic disorder characterized by wasting of all the tissues on one side of the face.

The disease was first described by Parry in 1825. It is very rare, and, as it does not threaten life or health and cannot be remedied, it is of slight practical importance.

Etiology.—Facial hemiatrophy may be a congenital anomaly. It is usually a disease of late childhood, being rarely met with after the twentieth year. In several reported cases the atrophy has set in some time after a blow on the same side of the face. The writer has described a case[1] where atrophy followed a frost-bite of the same side of the face. It has followed an abscess of the face. A few cases have set in shortly after an attack of one of the infectious diseases. It is more frequently met with in females than in males. It has been met in connection with scleroderma, hysteria, insanity, multiple sclerosis, syringomyelia, tabes, etc.

In several of the reported cases no special predisposing or exciting cause could be ascertained.

Symptoms.—The atrophy may begin in and involve the entire side of the face, or it may start in one part and gradually spread, and finally include the whole of one side of the face. In advanced cases it is usually more marked over the cheeks and lower part of the face than in the upper parts. All the tissues are more or less involved—the bones, muscles, subcutaneous tissues, and the skin. The muscles suffer less than any of the other tissues. In some cases they appear to be in a normal state, as far as can be judged from their size, movements, and electrical reactions. The bones of the upper and lower jaws waste in all directions. The nasal cartilages also share in the wasting. The tongue, especially in its anterior two thirds, is usually found atrophied. A few cases of wasting of the palate have been described. The jaws on the atrophied side usually undergo wasting.

The muscles of mastication are sometimes the seat of spasm, either of a tonic or clonic character. The skin over the affected side is found to be remarkably thin, and often changed in color, either in patches or throughout its whole extent. There may be an absence or an increase of pigment. The hair is much finer and scantier on the wasted side.

When the atrophy has reached an advanced degree the difference between the two sides of the face is very marked and striking. The appearance resembles that of a half face from each of two different persons. A

[1] *Keating's Cyclopædia of Diseases of Children.*

sharply defined vertical furrow marks the line between the healthy and the wasted side. A case of bilateral atrophy of the face has been recorded, and in one or two instances atrophy has been noticed in the arm of the same side. The functions of the special-sense organs on the atrophied side are retained. Sensation is, as a rule, seldom interfered with, a few cases only of disturbance being recorded.

PATHOLOGY.—Many theories have been advanced to explain the nature of the hemiatrophy of the face, some contending that it is due to lesions of the sympathetic, while others consider it to be due to disease of the fifth nerve.

In only one typical case of this disease has there been a complete examination after death. The examination was made by Mendel.[1] The patient, a female, was fifty-one years old at the time of her death. The atrophy set in a short time after an attack of facial erysipelas at the twenty-fifth year. The wasting involved the entire left half of the face, the anterior part of the left half of the tongue, and the muscles innervated by the left musculo-spiral nerve.

The following were the changes found after death, which was caused by phthisis: Proliferating interstitial neuritis affected the entire left fifth nerve from its origin to its terminations. The changes were more advanced in the second division than in the other branches of the nerve. A very marked difference was found between the right and left descending roots of the fifth nerve, that on the left side having undergone almost complete degeneration. A similar change was found in the substantia ferruginea, the nucleus of the so-called trophic root of the fifth nerve.

In Homén's case the onset and course of the atrophy differed from the ordinary hemiatrophy of the face. The course of the wasting was rapid, and after death it was found that this was caused by the pressure of dural growth on the Gasserian ganglion and branches of the third and fifth nerves. The result of the atrophy in these cases shows that pressure on the fifth is sufficient of itself to induce all the conditions considered to be characteristic of facial hemiatrophy. It remains, however, to be determined whether changes in the sympathetic are or are not capable of bringing about a similar state. Seeligmüller has described a case of facial hemiatrophy following an injury of the cervical sympathetic on the same side.

It must be borne in mind that frequently lesions both of the fifth nerve and of the sympathetic are unattended by any trophic changes in the face. At the present time we are unable to say what particular nerve or part of it requires to be invaded before the trophic changes set in. In all probability it is due to changes in the fifth, but it is not necessary to assume that such changes are of a gross organic character. It is probable that the trophic functions of the nerve may be inhibited, while it continues to perform its other functions in a normal manner.

COURSE.—Facial hemiatrophy progresses steadily until it has attained a considerable degree, and then remains stationary for the remainder of the patient's lifetime. It does not interfere with the general health. The term "progressive" which is sometimes used to designate its clinical course is, therefore, not strictly correct.

TREATMENT.—No treatment can be of avail except in cases where

[1] *Neurolog. Centralblatt*, 1889, No. 14.

the fifth nerve is pressed upon by growths which are removable by medicinal agents or surgical means. It is contended by some that galvanism has an influence in retarding the progress of the atrophy.

SCLERODERMA.

Synonyms.—Chorionitis; Sclerosis; "Hide-bound" disease.

Definition.—Scleroderma is a tropho-neurotic disease characterized by a circumscribed or diffuse induration of the skin and subcutaneous tissues.

Etiology.—It is much more frequently met with in females than in males, in the proportion of about four to one. It is a disease of middle life, the great majority of cases reported up to the present having been seen between the twentieth and fiftieth years, the disease known as infantile scleroma being a different affection. It not infrequently has been found to set in some time after an attack of one of the acute infectious diseases, as scarlet fever, measles, pneumonia, and erysipelas. It has been found in conjunction with certain chronic diseases of the central and peripheral nervous systems, as syringomyelia, subacute and chronic myelitis and neuritis, Raynaud's disease, etc. It is probable that all causes capable of lowering the stability of the nerve centres contribute to it. Uterine hemorrhage appears to be not an infrequent predisposing cause. In a considerable number of the cases marked signs of general and neurotic degeneration were present. In a small proportion no special predisposing or exciting cause could be discovered.

Symptoms.—There are two clinical types: (I.) the circumscribed; (II.) the diffuse.

I. *The Circumscribed Form.*—This is identical with Addison's keloid and morphœa. It is characterized by changes in the color and consistence of the skin. These changes are often present for some time before they are noticed by the patient. When fully developed the affected parts have a waxy or yellowish-white appearance. Sometimes the pigment disappears entirely from the affected portion of the skin, leaving it of a pearly-white color. It may be found surrounded by a pinkish hue. The form and extent of the parts involved vary much; sometimes minute points here and there only are observed. In other cases patches the size of the palm of the hand or even larger are met with.

The functions of the affected area of the skin are usually considerably interfered with. The part rarely sweats, and sensation is to some extent abolished. The sclerosed area may be found in the region of the distribution of a nerve trunk, or the disease may affect parts that have no common nerve supply. There the distribution is unilateral; more rarely the upper limbs and upper part of the trunk are the sole seat of the skin changes. It is more commonly met with in the region of the distribution of the fifth nerve and the intercostal and saphenous nerves.

The onset, although usually very slow, may be rapid, assuming an advanced state in the course of a few days.

The course varies much. It often lasts for months or it may be even years. Occasionally spontaneous disappearance occurs, the skin gradually assuming a normal look.

II. *The Diffuse Form.*—In the diffuse form the skin gradually becomes indurated, and so glued to the deeper tissues that it is no longer possible to pinch it up in folds. It becomes white, dry, and very smooth. The natural lines on the skin become obliterated. Usually the functions of the skin are more disturbed than in the circumscribed variety. The amount of disturbance depends on the severity and age of the sclerotic change. When marked, perspiration does not occur and sensibility is blunted or gone.

When the skin of the face is affected the loss of elasticity results in a loss of expression; the mouth is pinched; the alæ nasi are drawn close together; the eyelids are either partially closed or wide open. If the skin over any of the joints of the extremities is involved, it leads to a practical ankylosis of such joint. The rigidity of the skin may be so great and widespread as to render the patient incapable of any movement. The head may be retracted from involvement of the skin at the back of the neck. The mucous membranes in the immediate neighborhood of sclerosed skin may become involved.

The COURSE and PROGNOSIS in the diffuse variety are different from the circumscribed. In the former the outlook is more serious, the patient not uncommonly succumbing to some intercurrent disease, as phthisis. Rheumatic attacks are very frequent in sclerodermic patients.

Other forms of trophic disorders are commonly met with, as Raynaud's disease and angio-neurotic œdema. The occurrence of such considerably lessens the chances of recovery.

PATHOLOGICAL ANATOMY.—The microscopic examination of the sclerodermic skin shows the following changes: There is considerable increase in the connective-tissue elements. Changes in the vessels are found, consisting in peri- and endarteritis and filling of the veins in the peripheral parts of the patches. Changes in the nerves of the blood-vessels, although suspected and carefully looked for, have not as yet been demonstrated.

Lewin and Heller, who have made a very careful study of this subject based on 459 cases collected from the literature of the subject and their own observation, look upon the disease as in most instances due to a disturbance of function of the vasomotor centres situated in the medulla, cerebral cortex, and spinal cord. They consider also that in certain cases there may be brought about a local peripheral neuritis—such cases as arise, for instance, from a local injury of a nerve, as in whitlow. They suggest as an exciting cause the supposed action of a toxin causing a paralysis of the vessel nerves.

The primary stage is in all cases, according to the above observers, characterized by an over-supply of blood to the part or parts, which later on undergo sclerotic changes, the primary thickening finally giving place to atrophy.

TREATMENT.—Measures that tend to improve the general health are indicated. Massage, hydrotherapeutics, cod-liver oil internally and locally, all do good. Several cases have been recently reported where thyroid powder has been useful to some extent.

VERTIGO.

DEFINITION.—Vertigo is a symptom of many different morbid conditions, and one frequently of such a marked and distressing character that it calls for special consideration. The term, popularly and professionally, is used in a very loose sense, meaning any sensation, either physical or psychical, which has the effect of making the subject's position in relation to external objects uncertain.

ETIOLOGY.—1. The most frequent cause of vertigo is ear disease. It is met with as the result of disease of the external, middle, and internal ear.

2. It occurs from errors of refraction and unbalanced ocular muscularity.

3. It results from nasal and laryngeal disease.

4. It is frequently met with from toxic causes, either poisons introduced from without or auto-intoxication. It is often a symptom of the primary action of the poisons of the acute infectious diseases. The so-called gastric vertigo is, at least in many instances, more properly referred to a toxic than a reflex origin. Alcohol and tobacco in those unaccustomed to their use cause vertigo.

5. Vertigo is a symptom frequently met with in cardio-vascular degeneration, especially in aortic regurgitation and general arteriosclerosis.

6. It is caused by organic diseases of the brain, especially by tumors and insular sclerosis.

7. It is a prominent symptom in many functional troubles of the central nervous system, as in epilepsy, neurasthenia, and hysteria. An attack of vertigo may be the equivalent of a migrainous attack or of an epileptic paroxysm.

8. Vertigo is the chief symptom of that rare and obscure state known as "paralyzing vertigo."

Vertigo may be a constant or paroxysmal state. It may be mainly or entirely subjective or objective. It may be a slight transient feeling, or it may be so sudden and profound as to fell the patient. There is, of necessity, a considerable degree of mental confusion in sudden and severe vertigo. Nausea and vomiting are usual accompaniments of severe vertiginous attacks.

AURAL VERTIGO, INCLUDING MÉNIÈRE'S DISEASE.

Vertigo may be brought about by irritation of any part of the external, middle, or internal ear. Vertigo from disease of the external and middle ear is a more common affection than is generally supposed. It may be due to a foreign body in the ear, to the presence of wax, simple or purulent catarrh of the middle ear, closure of the Eustachian tube. Burnett considers that the vertigo of chronic catarrh of the middle ear is due to the retraction and ankylosis of the ossicles, and the consequent pressure of the stapes on the labyrinth.

Ménière's disease is a form of aural vertigo characterized by a set of distinct symptoms. The term is loosely applied, all forms of aural vertigo being included by some writers, while others limit it to certain

definite clinical symptoms brought about by various internal-ear changes. In this section the latter view will be followed.

Some authorities reject the term altogether, but it is open to objection, no satisfactory equivalent for it has been suggested. The term "auditory vertigo" is now usually employed to cover cases of vertigo arising from external and middle-ear disease.

Symptoms.—Ménière's disease is characterized by recurring attacks of vertigo, there being rarely any continuous sensation of disturbance of equilibrium between the attacks. These vary much in frequency in the same and different cases. Often they come on in series, as in epilepsy, two or more attacks in the twenty-four hours being followed by an interval of freedom lasting one or several weeks.

Usually the patient is compelled to lie down. Sometimes he falls as suddenly as an epileptic. In such severe attacks there may be a momentary loss of consciousness. The sense of giddiness is usually very intense, compelling the patient to lie quiet with the eyes shut. Nausea and vomiting come on, and a cold sweat covers the body. The subject is pallid and has an alarmed look. In severe attacks he is a complete physical and mental wreck for the time. The after-state is much more depressing than what we meet with after an epileptic paroxysm. An attack may come on when the person is in the recumbent position or even when asleep.

The attacks vary much in severity, from a slight sensation of giddiness up to the alarming state already described. In duration they vary from a few minutes up to an hour or even longer.

Tinnitus is usually a permanent complaint in patients the subjects of Ménière's disease.

Nervous deafness on the affected side is generally to be made out. It is, as a rule, progressive, leading eventually to complete deafness.

Diagnosis.—The paroxysmal, epileptiform character of aural vertigo marks a distinct clinical difference between it and other forms of this trouble, so that the clinical picture it presents is usually sufficiently distinctive to enable one at once to recognize it and not mistake it for other forms of vertigo.

Cases of minor epilepsy, where the chief symptom is giddiness, may be mistaken for Ménière's disease. The attacks in the former are shorter in duration, and very rarely have the intense and continued nausea and vomiting which are the rule in Ménière's disease. In the latter there is a death-like pallor of the face in severe cases, which lasts many minutes. In epilepsy pallor is only seen as a passing, momentary state at the beginning of the attacks. Loss of consciousness is the characteristic symptom of all forms of epilepsy. It is an exceptional symptom in Ménière's disease, being only present in the severe cases of sudden onset. The special auditory symptoms of Ménière's disease, tinnitus and nervous deafness, will lead the observer in the right direction. In some cases of Ménière's disease there is more or less giddiness in the intervals between the severe paroxysms. This is not the case in epilepsy.

The so-called "congestive attacks" occurring in the course of certain chronic cerebral cases may be mistaken for Ménière's disease. Other symptoms are present which, as a rule, are distinctive enough to render

the diagnosis comparatively easy. In disseminated cerebro-spinal sclerosis such attacks are frequent, but one or more of the classical symptoms of this disease are rarely wanting, such as the syllabic speech, nystagmus, the optic neuritis, and tremor. The "congestive attacks" of general paralysis of the insane, although frequent, cannot give rise to any prolonged difficulty in the differential diagnosis. We meet with congestive attacks of a cerebral character of an unknown nature where considerable care is required before a final judgment is pronounced.

The Nature of Ménière's Disease.—The clinical syndrome comprising Ménière's disease is brought about by many different states of the internal ear. Injuries, hemorrhage, inflammatory exudation, syphilitic process, direct degenerative lesions in the auditory nerve, degenerative arteritis of the internal ear, inflammatory and other exudative processes in the middle ear, may by their pressure produce this disease. It sometimes follows wounds and blows on the head.

Ménière's disease, although primarily due to irritation of the peripheral terminations (vestibular) of the auditory nerve, is essentially brought about by a disturbance of the cerebral and cerebellar centres presiding over equilibration. It is only in this way that the epileptiform character of the attacks can be satisfactorily explained.

Prognosis.—Occasionally we meet with a case in which there has only been a single attack, but this is a very exceptional occurrence, the rule being for the attacks to recur and continue recurring until such time as the auditory nerve becomes completely degenerated, when they cease.

Exceptionally, cases are met where the vertigo ceases short of the production of deafness. The deafness and tinnitus usually persist long after the vertigo ceases.

Treatment.—We are rarely able to diagnose with certainty the nature of the labyrinthine irritation or to remove it if we can diagnose it.

If the degenerative changes in the auditory nerve are due to arteriosclerosis, either simple or syphilitic, iodide of potassium is called for. Whether it has any influence beyond moderating the blood pressure is doubtful. It is not necessary to give it in more than moderate doses. In arterial changes, whether primarily syphilitic or secondary to other poisons or mechanical causes, there is no advantage, but rather a distinct disadvantage, in giving the iodide in large doses.

Degenerative changes in the auditory nerve terminations are occasionally manifestations of the gouty constitutional state. In such cases an appropriate hygienic regimen and diet with alkalies are indicated. In all cases of aural vertigo limitation of nitrogenous food will be found distinctly beneficial, imperfect gastric and duodenal digestion being often a distinct exciting cause. The symptomatic indications call for agents that lessen the central and peripheral irritability of the auditory nerve. As in epilepsy, bromide of potassium fulfils this indication better than any other agent at our command.

When the tendency to vertigo is but slight it will sometimes be found that this drug will greatly lessen it or perhaps altogether remove it. Doses of half a drachm twice in the twenty-four hours will usually be found sufficient. In severe cases the action of the bromide will be found disappointing, even in much larger doses.

Gowers speaks highly of the application of blisters over the mastoid process in allaying the peripheral irritability of the auditory nerve.

The empirical use of the salicylates, and of quinine in doses sufficient to induce cinchonism, is a common practice since its introduction by Charcot. It is rare, I think, to meet with cases where such treatment is followed by marked benefit. In the vertigo of middle-ear diseases Burnett has had good success by removing the incus and thus relieving the pressure of the stapes on the oval window.

Ocular Vertigo.

Ocular vertigo rarely presents anything like the same intensity or persistency as does auditory vertigo.

It is due to false projection of the field of vision or to diplopia in ocular paralysis. It may be combined with squint and double vision or appear as the only symptom. When both eyes are open the vertigo is dependent on the diplopia. If the unaffected eye is closed, it depends upon an erroneous localization of objects in the field of vision. The treatment is dependent on the cause of the paralysis. If this can be removed, the vertigo disappears.

Vertigo arises also from nystagmus as the result of the contradictory impressions from the oscillatory movements of the eyeballs. It is met with more frequently in nystagmus, but may be due to such movements, no matter how induced, whether by peripheral strain or gross disease of the central nervous system.

Nasal and Laryngeal Vertigo.

Vertigo may result from swelling of the mucous membrane of the nose, and it disappears when the cause is removed. A paroxysmal swelling of the nasal mucous membrane is sometimes met with in neurasthenics, which brings about vertigo. In certain organic laryngeal troubles attacks of an apoplectiform or epileptiform character, with vertigo, burning feeling in the throat, and cough lasting a minute or two, have been described.

Toxic Vertigo.

The vertiginous attacks following the use of tobacco, alcohol, and other toxic agents are too well known to require description; neither does the vertigo of the acute infectious diseases require special reference. Both forms are temporary symptoms from the circulation of toxins acting either on the cortical centre or on the peripheral terminations of the auditory nerves.

Vertigo from the absorption of the products of decomposition in the gastro-intestinal tract is, next to auditory vertigo, the most frequent form of this symptom. Some people when they take anything that "disagrees" with them become giddy. No doubt vertigo may be induced reflexly from gastro-intestinal irritation, but in practice it will be usually found that the great majority of cases arising from intestinal causes are toxic and not reflex. Vertigo results also from uræmic poisoning.

Irritation of the intestinal mucous membrane by worms may be the exciting cause of a troublesome vertigo.

The TREATMENT of toxic vertigo is more successful than of most other forms. A recognition of the cause, especially of the article or articles that bring about poisoning or irritation, will lead to a proper preventive treatment.

Of all purgatives and antiseptics, calomel is the most efficient, combining in itself both actions. Given with sodium bicarbonate and followed by a brisk saline purgative, it quickly destroys or eliminates the products of decomposition.

VERTIGO FROM CARDIO-VASCULAR DISEASE.

The vertigo from cardio-vascular disease is an important and common variety. It is chiefly met in advanced life, and is dependent on the intermittent blood supply to the cerebrum brought about by sclerosis of the cerebral arteries or from the defective force of the heart from valvular insufficiency or myocardial degeneration. Both cardiac and arterial changes are found together, going hand in hand. Cardio-vascular vertigo may be either a paroxysmal or continuous symptom. It is often constant in old people with rigid vessels. The correct diagnosis of such cases is a matter of importance. They may be mistaken for the premonitory symptoms of an apoplectic attack or they may actually be such premonitory symptoms. Sudden paroxysms of vertigo coming on late in life are of more serious import than slight vertiginous feelings of an abiding character: the former may be the precursors of complete arterial blocking or rupture. It is important to diagnose vertigo from cardio-vascular disease from that due to auditory disease, in order to save the patient needless alarm. The former is remotely a symptom of much more grave import than the latter.

The TREATMENT of the vertigo from cardio-vascular changes is dependent on the cause. As this is essentially from organic changes, we can do no more than attend to the symptomatic indications. When speaking of the treatment of Ménière's disease from degenerative arterial changes mention was made of the use of iodide of potassium. In cardiac senile degeneration arsenic is a valuable agent through its action on metabolism and as a hæmatinic.

VERTIGO DUE TO ORGANIC DISEASE OF THE CENTRAL NERVOUS SYSTEM.

Vertigo from disease of the central nervous system is of considerably less importance than the forms already described. It may be met with in tumors, no matter in what part of the brain they may be situated; it is rarely, however, troublesome, except in those situated in the posterior portion of the base of the brain, in the cerebellum, cerebral and cerebellar peduncles, and in the corpora quadrigemina.

In peduncular, cerebellar, and medullary growths it may be the most marked of all the symptoms present. A case recently under Blackader's observation of a middle-aged man illustrates this. For three or four months this patient was so troubled with giddiness that he was unable to walk without assistance. At the autopsy a growth the size of an

almond was found pressing on the right side of the medulla. When looking at an object it appeared to be constantly turning in space.

Cerebellar growths usually induce marked giddiness. When this symptom is prominent it has a slight localizing value.

In insular sclerosis vertigo is frequently a terrible symptom.

In tabes dorsalis it is also met with, and not infrequently in an aggravated form.

Vertigo from Functional Nervous Disease.

In certain functional disturbances of the nervous system vertigo is a frequent symptom. In epilepsy it may form the aura or it may be the equivalent of the entire paroxysm. It often becomes a nice point in diagnosis to decide on the significance of a momentary attack of vertigo, whether it is an attack of petit mal or of simple vertigo. The proper place for the differential diagnosis between these two states is in the section devoted to Epilepsy (page 483). In the protean group of symptoms included in the term neurasthenia, vertigo is a prominent one. It may be due to ocular muscular disturbance or to reflex gastro-intestinal irritation. Frequently it is difficult or impossible to ascertain its cause.

In hysteria, exophthalmic goitre, migraine, chorea, vertigo is sometimes, but rarely, a symptom of much importance.

Mechanical Vertigo.

The vertigo induced by movement is of comparatively slight medical importance. Under this head are included the vertigos of "sea-sickness," "train-sickness," "elevator-sickness," "swing-sickness" (see article Sea-sickness, page 659).

Paralyzing Vertigo, or Gerlier's Disease.

This rare condition is characterized by vertigo, paresis of the extremities, ptosis, and mental depression. It is endemic in certain parts of Switzerland.

DISORDERS OF THE MIND.

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MELANCHOLIA.

By WILLIAM BROADDUS PRITCHARD, M. D.

Definition.—The term melancholia is used in alienistic medicine to describe a form of insanity characterized essentially by a more or less extreme and unreasonable mental depression, reflecting a state of psychical pain, associated with sluggish mental reflexes and a morbid propensity to suicide. Hallucinations and delusions may or may not be present. Somatic disturbances, involving torpor or perversion of visceral functions, usually coexist.

Classification.—Certain variations in the clinical picture, with attendant and consequent modifications in prognosis and treatment, justify, and indeed demand, the recognition of sub-types. The affection assumes at least three symptomatic forms:

Simple melancholia;
Stuporous melancholia;
Agitated melancholia.

Each of these forms may be still further subdivided: melancholia attonita, for example, is a more intense and overwhelming variety of stuporous melancholia; hypochondriacal melancholia is a form of the disease in which delusions and hallucinations referable to the viscera predominate; while melancholia with præcordial fear is a subvariety of the agitated form in which exists a delusion referable to the heart. The fundamental mental state, however, is the same, and too much weight should not be attached to these clinical variations, since they are inconstant and interchangeable, the same patient in the course of an attack sometimes manifesting at different periods all three varieties.

The division of Bevan Lewis into the simple and the delusional forms, the former affecting the affective or emotional sphere, the latter the intellectual sphere, is open to objection for this very reason of inconstancy and interchange of types. It is, however, a division quite attractive in its simplicity and of practical utility in diagnosis, since, as a rule, hallucinations and delusions are absent in simple melancholia, except in the hypochondriacal variety, while it is equally and almost invariably the rule to find one or both present in the stuporous or agitated forms of the disease.

Etiology.—Any factor entering into the life of an individual which exerts a prolonged depressant effect upon the physical or mental being may result in melancholia. Heredity is less conspicuous and more infre-

quent than in any other psychosis. Personal predisposition is far more important. The victims of this affection are usually those who have been of a timid, reserved, over-conscientious, and rather melancholy temperament originally. The nervous, anxious borrower of trouble passes most readily under the stimulus of some apparently trivial exciting cause across the border-line. It is not uncommon, however, to find the reverse true, and I have under observation at the present time a patient affected with a profoundly stuporous form of melancholia, who was formerly a jolly, cheerful, careless, happy fellow, whose natural temperament presented the very antithesis of his present state.

Consistently with the above, and as might be expected, we find a much larger proportion of females than males to be affected with melancholia. Regis gives the ratio as two females to one male. Certain periods of life, those of puberty, climacteric, and beginning senility, representing as they do periods of special nervous and mental instability, are to be looked upon as times of crisis in the predisposed. Among the direct causes physical illness if protracted, and especially if it involve the digestive tract, may induce a melancholia. Grip or influenza among acute diseases is especially noteworthy as a cause. Business or domestic worry and friction, grief, disappointment, overwork mentally, involving loss or curtailment of the usual amount of sleep, with irregular eating and consequent digestive disturbances, may superinduce a true melancholia. Insomnia, by the way, while usually considered a symptom, may, and unquestionably does, appear at times as a cause. Sudden and severe physical shock, as from a surgical operation, an accident, or parturition, may prove the immediate and sometimes the sole determinable origin of an attack. Auto-intoxication from ptomaines, toxins, or other autogenous products of stomachic or intestinal decomposition or putrefaction is a well-authenticated cause in many cases. In this connection the etymology of the word melancholia (*μὴλας*, black, *χόλη*, bile) is of special interest, since the digestive trouble is usually duodenal in location, and dependent, in part at least, upon a deficient or perverted secretion of bile, with loss of its antiseptic properties and consequent putrefaction, with subsequent reabsorption. The association of gall-stones with melancholia is by no means uncommon. The condition of copræmia, so termed by Hewett and described as occurring in young girls, a self-poisoning from retention and absorption of fecal matter in the large intestine, is occasionally the cause of melancholia, acting in the same way, though more often associated with anæmia and hysterical manifestations. The previous personal temperament of the individual probably determines the direction of the explosion, the cause being practically the same. The lithæmic or so-called uric acid diathesis (oxaluria) is not infrequent in the subjects of this affection, and the significance of the association is of importance in connection with the prognosis and treatment in individual cases.

Finally, while expressing some degree of personal skepticism upon the subject, I may mention the relations of various sources of reflex disturbance in disease of the uterus and adnexa especially, and in other organs, causing melancholia, and referred to by many writers upon the subject, which relations should at least be considered and investigated in all suspicious cases. Disease of the uterus or of any organ, inducing

a lowered vitality and involving prolonged suffering and anxiety, with loss of sleep, may of course cause melancholia, but that any more direct causative relationship exists in uterine disease or affections of the generative organs, *per se*, I doubt profoundly.

In this connection I wish to call attention to an etiological factor which, if my personal experience be a criterion, is more frequent than would be indicated by the scant references to the subject in current medical literature. I refer to sexual perversion, which has appeared as the cause in three cases of melancholia seen by me within the past four years. In all three of these cases the sexual perversion antedated by several months or years the melancholia, and in two cases the direct causative relationship was unmistakable.

PATHOLOGICAL ANATOMY.—It is an interesting and significant fact that in most modern textbooks upon mental disease the subject of pathology is discussed from the standpoint of the insanities collectively. There is, it is true, a special pathology in general paresis and in the organic psycho-neuroses, such as epilepsy, chorea, etc., as well as in the toxic insanities of alcohol and lead, but even here our information is sadly deficient, and we know practically nothing positively as to the essential morbid anatomy of mania, melancholia, or dementia existing as a pure psychosis. As a matter of fact, the brain is found absolutely free from any demonstrable structural change in relatively many cases dying during the acute stage. Areas of anæmia, of ischæmia or pallor, or of œdema have been observed, but these may be due entirely to the general anæmia and malnutrition common to the affection. Disease of the viscera is quite common as a post-mortem finding in melancholia, especially of the liver, spleen, stomach, and intestines. Impacted gall-stones, stomachic or duodenal ulceration, pyloric constriction, splenic enlargement or atrophy, are among the lesions which have been noted. From these facts, and the well-known constancy with which alterations in the quality and quantity of the blood exist in melancholia, it seems to me that we have reason to expect more from the studies of the chemist than the microscopist in elucidating the true pathology of melancholia.

CLINICAL HISTORY.—As the clinical picture in melancholia varies considerably with the type present in individual cases, I shall describe separately and through the medium of selected case-histories the symptomatology of the three varieties mentioned. There are certain symptoms, however, which are common to all forms, especially in the early stages; and, because of this somewhat prodromal significance and their diagnostic value when collectively present, I shall give them attention first. These symptoms are as follows:

Insomnia;
Headache;
Mental depression with obtunded cerebral reflexes;
A peculiar facies or expression;
Suicidal tendencies.

To these five may be added certain somatic symptoms which are present with almost equal certainty, as well as a description of the characteristics and peculiarities which more or less distinctively mark the hallucinations and delusions noted in melancholia.

Insomnia, in my experience, is an absolutely constant symptom in beginning melancholia. It sometimes persists throughout an attack, even when prolonged for months or years. The degree of insomnia varies widely, and must be determined by comparison—not with others, but with the patient's own previous habit as to sleep. A man who in health habitually sleeps ten hours in the twenty-four suffers from insomnia if his sleep is cut to eight hours, though the latter amount is considered the standard in health. In melancholia the insomnia is not often absolute, the patient, as a rule, getting three fourths or half or one fourth as much sleep as formerly. Sometimes, however, sleeplessness is complete and prolonged over several days or weeks. Indeed, Savage has recorded the case of a female patient who, although visited hourly for three months, was never found asleep during that time. A certain amount of sleep may be obtained in some cases, but the patient will complain that it was unnatural or unrefreshing. Horrifying, frightful dreams, common enough in melancholia, may so harass and distress the patient as to make waking a boon and sleep a dreadful agony.

Headache is also a very common symptom in melancholia. It is, as a rule, most often observed in the simple cases, though this observation is probably only relatively true, since the mental obtuseness in the stuporous variety and the preoccupation and great intensity of agitation in the other type render the headache less conspicuous. The character and degree of the headache vary greatly. By some it is described as a feeling of weight or pressure or tension, by others as a pain, by others as a tired ache, and by others, still, as a burning or numb sensation. The most noticeable and constant peculiarity with regard to this headache is its location in the occipital or occipito-cervical region—a fact to which L. C. Gray first called attention. In certain stuporous patients whose cerebral reflexes are so dulled as to make voluntary statements impossible or questioning valueless the presence of this headache may be manifested by the attitude with the head leaned back, resting the neck upon a support, or with the hands clasped back of the neck, as if for support or pressure. On emerging from this state of stupor such patients will speak of this headache, recalling their sufferings, physical and mental.

Mental depression, always present of course, is actively or passively manifested in melancholia according to the type. This depression in melancholia represents and reflects an intense subjective alteration of the *ego*. The essential factor in all insanities consists in a departure from the normal in the ego of the individual, quantitative in dementia, and rather qualitative in mania and melancholia. In mania the ego is objectively exaggerated; in dementia it is extinguished or obliterated partially or completely by a process of more or less gradual fading or decay. In melancholia the alteration and exaggeration are subjective, the mental life of the individual becoming intensely introspective. Object consciousness is lost more or less completely; the melancholiac is dominated by a subject consciousness which is absolute and always depressive in its emotional manifestations. The receptivity of the individual to impressions from without through the medium of the various senses is markedly diminished or lost altogether, and the mental reflexes are correspondingly obtunded, so as to be elicited with difficulty if

at all. Occasionally in melancholia, especially in the agitated and stuporous forms, there develops transiently a condition which is in marked contrast with the asthenic-atonic torpor usually observed. This transient state is one of sudden or quickly developing furious motor and mental excitement, a sort of explosion of dammed-up mental and physical forces. The term "melancholic frenzy" has been applied to these paroxysms, and aptly describes them, for, while there is often the flushed face and restless motor and mental excitement and even violence of a maniacal outbreak, the mental state is none the less one of profound melancholy anguish. Such paroxysms are more or less common to agitated melancholia, and occasionally occur in stuporous cases. They are peculiarly associated with delusions of præcordial fear, and the presence of this delusion, while not necessarily premonitive of such attacks, should be looked upon as suggestive.

The facies is an accurate reflex of the mental state in melancholia. It is exceedingly difficult to paint in words, and yet it is quite striking, and to the trained observer it is immediately suggestive of the diagnosis. Anxious despair, though etymologically somewhat paradoxical, describes it with fair accuracy as it exists in the simple cases. In the agitated variety the same term would apply, though the element of anxiety preponderates, while in the stuporous type the picture is that of despair alone, but in profound and hopeless degree. Gray describes this facies in melancholia as reflecting a mixture of gloom and suspicion, a description which perhaps conveys more accurately the picture presented. I have never seen in melancholia a pleasurable emotion reflected in the facial expression, and laughter in melancholia would constitute a refutation of the diagnosis.

A morbid propensity to suicide is quite characteristic of melancholia, and is a most important factor to be carefully considered. It is unwise to assume its absence in any case, even where careful and repeated questioning and observation fail to reveal its presence. It is sometimes the first overt symptom of the disease which is noticed. Some patients from the first and throughout an attack manifest a most energetic and persistent determination toward self-destruction; in others the attempt will follow a sudden transient but recurrent impulse which is totally unexpected. The degree of ingenuity and patient, careful deliberation exhibited by certain cases, especially in simple melancholia, in planning a suicide is quite remarkable, and only too often unfortunately successful. In other cases, notably in certain stuporous forms, such as melancholia attonita, the almost total paralysis of physical and mental energy, associated with this suicidal propensity, results in situations which are ludicrous in the extreme. A patient will place a towel around his neck with suicidal intent, but, lacking sufficient mental concentration or physical energy, or both, will listlessly pull the ends with a force which would not strangle a baby. Suicide is most to be feared in the agitated cases, and especially those with præcordial delusions. In simple melancholia the impulse is less frequently present, but when it does exist in this variety it is never to be under-estimated in importance, since it is in the simple variety that attempts at self-destruction are usually made with an intelligence which almost guarantees success. I have noted the interesting fact that a large number of suicidal attempts in melancholia

occur in the early morning hours, before day—a fact of significance in connection with the sleeplessness which is usually present in the latter part of the night in this affection. Homicidal impulses or actions do not belong consistently to melancholia, and as a matter of fact are rare. Indirectly, however, murder—for the action is premeditated always—may be done. A wife anxious to die may selfishly desire to take her husband with her into death. The father or mother, seeing only ruin and poverty or disgrace for self and all the household, deliberately annihilates the entire family. Others, having religious scruples on the subject of suicide, and yet anxious to die, may attack the passing stranger, hoping either to fall a victim to the stranger's wrath or to the law with its penalty of death. In paroxysms of melancholic frenzy homicide may result practically as an accident.

Hallucinations and delusions may or may not be present in melancholia. In the simple form they may be absent throughout an attack; in agitated and stuporous melancholia both are present essentially, and both are in accord with the mental status in that they are always depressive. Illusions are rare, if indeed they occur at all. Illusions involve an exaggeration of object consciousness absent in melancholia; hallucinations, on the contrary, are essentially consistent with a hyperacuteness of subject consciousness, which is characteristic of melancholia. The relatively extreme frequency with which hallucinations of hearing, a subjective faculty, are present in melancholia is in consistent harmony with the fundamental state of mental life in this affection. Hallucinations of hearing "voices," "the voice of God," the voice of a deceased relative or friend, or of an enemy, are far more common than is perversion of sense appreciation in any or indeed all other special sense impressions. Taste and smell perversions perhaps are next most frequent, while visual hallucinations are less often observed. The organs of the body may be foci of hallucinations or delusions known as visceral. The "stomach is rotten," the "liver is turned to stone," the "bowels are full of holes," the entire "body is dead," etc. etc.

Such hallucinations or delusions not infrequently have a basis of fact in demonstrable disease of the viscera. The foul breath and offensive secretions in the mouth in constipated dyspeptics afford an almost rational basis for the idea that some internal organ is rotten. The horrible stench of decayed and dirty or neglected teeth gives rise to the delusion of poisoned food at times. In a case seen by me in which this delusion of poisoned food was present, causing much trouble and anxiety through the refusal of all food resulting therefrom, I found the teeth in a most horrible state of offensive decay. Removal of those teeth, together with the use of an antiseptic mouth-wash for a few days, caused the complete disappearance of the delusion, although another took its place. In another patient a delusion that the bowels were full of holes was relieved with equal promptness when the large intestine was emptied of an accumulation of hardened fæcal masses, some of which had evidently been present for months, considerable pain being involved in their forcible removal by hand. Such visceral hallucinations and delusions have something of the significance in melancholia of the auræ which are present in epilepsy, and should be investigated with equal care and patience.

The most common of all the visceral delusions is that which is referable to the heart, known as the delusion of præcordial fear. It is especially common in agitated melancholia, and its onset is sometimes significant of an impending paroxysm of melancholic frenzy. As a rule, no organic heart lesion is present, but cardiac action is invariably much disturbed functionally in these cases, and sometimes organically. It is not advisable to pay objectively much attention by repeated examinations to the condition of the heart, since by so doing the delusion is apt to be intensified and more firmly fixed.

Certain delusions occurring in melancholia, as in other insanities, have an apparently purely psychic origin, and do not originate through perversions of sensory impressions through illusions or hallucinations. In melancholia these psychic delusions are always sad and distressing, and also entirely egotistic in a subjective way. One of the most common is the groundless fear or belief in some impending calamity or horrible disaster. The business-man sees financial ruin ahead; the senile melancholiac sees poverty and the poorhouse staring him in the face; the mother looks constantly in imagination upon her children or husband dead or dying from disease or accident; the young man looks into a future filled with the wrecks of blasted hopes and ambition, out of which no possible salvage can be obtained. Delusions of self-degradation are quite common. Introspective and retrospective in intense degree, as are all mental processes in these cases, the inevitable and quite natural mistakes of human life are magnified a thousand-fold. The patient has done a gross and irremediable injustice or wrong to wife or husband or friend. Sometimes the sin has been committed against God or the Holy Ghost, which sin in each case is the very one referred to in Scripture as "the unpardonable sin." This delusion of the unpardonable sin is quite common in melancholia, and has been considered by some alienists as being almost diagnostic. It has been observed, however, in general paresis, and in a few cases of religious paranoia. It is more fixed and consistent, however, in melancholia, and is at least always suggestive of the diagnosis. The more or less prevalent idea that the presence of this delusion indicates a more grave or intractable or incurable type of melancholia has little if any foundation in fact.

The *physical symptoms* which are most conspicuous in melancholia are referable chiefly to the digestive organs or tract, consisting essentially of an atony or torpor of function. As a rule, the physical disturbance precedes the mental. The appetite becomes impaired or lost, the tongue coated, the breath offensive, and the bowels constipated. The patient loses flesh and energy, acquiring an increasing indisposition for muscular effort or exertion. There is a feeling of physical weariness, and fatigue follows quickly any physical exercise. The skin takes on a pale, sallow, or muddy appearance, becomes dry and rough, or bathed in cold, clammy sweat. The peripheral circulation is poor; the hands and feet are nearly always cold and often œdematous, especially the lower extremities. Certain vasomotor disturbances are frequently present in the agitated melancholias and in alcoholic cases, and in women in whom the affection develops at the climacteric. These disturbances consist of alternate pallor and flushings, with or without slight vertigo. There is a peculiar pulse rate often observed in the stuporous and in the agitated cases which is

probably of vasomotor origin, occurring perhaps through the medium of some toxæmic state acting upon the vagus nuclei. The pulse in such cases may run up to 100, 120, or 130, remaining so for several days or weeks unless controlled by medication. Less frequently the pulse rate falls below the normal, as low as 35 or 40 beats per minute. Respiration is not correspondingly increased; on the contrary, the number of respirations per minute is reduced if affected at all. Oxidization is imperfect and anæmia results. The temperature may be below normal one-half or one degree, but a slight elevation is more common in my observation. The temperature of the extremities, according to Regis, is always below normal, in some instances falling as much as three or four degrees Centigrade. The urine is scant and usually high-colored, and of high specific gravity, though in the agitated type it may be occasionally quite free. Sexual desire is usually in abeyance, and amenorrhœa is quite common in women. Common sensibility is, as a rule, more or less impaired, the reaction-time to stimulation of all forms of common and of special sensation being much slower than the normal. The experiments of Bevan Lewis demonstrating this fact are of much interest. The motor reflexes, on the other hand, as might be expected, are unduly active, especially the knee jerks.

Simple Melancholia.—Mrs. L. W——, Jewess, æt. thirty-four, a childless widow in fairly good circumstances, was seen by me at her home April 8, 1895. Family history negative. Personal history had been that of good health up to a period of five or six months previous to her husband's death, which occurred in January, 1895. Her husband died of Bright's disease after a bedridden illness of several months, during which time most of the care and nursing fell upon her. The protracted nature of the husband's illness, with its many crises, involved a prolonged strain upon the wife's nervous system, with a constant state of anxiety and mental tension. Her meals were served irregularly and eaten hurriedly; her sleep was seriously interfered with. Dyspeptic symptoms developed, with constipation and much nervous restlessness, culminating upon the husband's death in complete mental and physical collapse, with symptoms of explosive hysteria. This state was succeeded by a gradually increasing fixed melancholy, almost absolute insomnia, failure of appetite, and loss of flesh. Formerly active in religious work, and connected with two or three charities, she lost all interest in such matters, refused all visitors, and pathetically insisted upon being left alone. She became indifferent to her household and business obligations, began to be negligent of her dress and appearance, and frequently stated that she longed for death. She refused to allow her friends to call in medical advice, stating that she was not ill, and that no earthly physician could do her any good. At the time of my visit, which had to be arranged by strategy, I found the patient sitting partly dressed in her bedroom. She had an expression indicative of great despondency. She was reticent, and decidedly persistent in her reticence, but after much persuasion she was induced finally to talk rather freely of herself. She felt utterly despondent and weary of life, she said. There was nothing to live for, and she wished to be let alone. Almost immediately, however, she began to describe certain physical symptoms, and asked with some evidence of anxiety if these symptoms indicated any serious

illness. She spoke of a feeling indescribable and indefinite, but none the less persistent and distressing, of some impending calamity or misfortune which was to befall her. She knew "something dreadful was going to happen." Her head felt tired all the time, especially in the back of the neck. She could not think, and had not written a letter or read one through in three weeks. She passed her nights in sleepless misery. She was afraid to go to sleep lest something dreadful should occur for which she would be unprepared. At times she did sleep, or at least lose consciousness, the two or three hours thus spent, however, representing a most distressful agony through the frightful dreams which occurred, and from which she would awaken at times in tremulous terror, drenched with perspiration. When asked whether she had ever thought of suicide as a means of relief, she became suspicious and evasive in her replies, finally answering me with a counter-question as to whether I did not think suicide a dreadful sin. Under appropriate treatment, began actively at once, this patient made a rapid uneventful recovery, and was discharged as cured six weeks after my first visit, the total duration of the case having been a little less than three months.

Stuporous Melancholia.—A. B——, German-American, æt. twenty-one, the youngest son among several healthy children of wealthy parents living in handsome style, became suddenly insane on the evening of December 29, 1896. The family had retired to the library after dinner, when the father asked the patient to join him in a game of cards. Remarking that he did not care to play, the son sat quietly for a few moments, and then, abruptly rising, he walked toward the window, and looking out exclaimed with a manner indicative of intense mental agitation and excitement, "The day of judgment has come! Judgment day is at hand!" Immediately he began to weep and wring his hands, his whole manner indicating a mental state of agonized anxiety and despair. No argument or persuasion could influence him, and the night was spent until far on in the morning in restless walking up and down the room, accompanied by his father. About daybreak he fell asleep exhausted, but woke again in tremulous fright some two hours later. His excitement quickly subsided, and in a day or two was succeeded by a state of apathetic mental misery with characteristic facies, the delusion of an impending day of judgment still persisting. For three days and nights he neither slept nor ate, lying in bed quietly, but with wide-open eyes, vacantly looking into space or as one wrapped in painful meditation. Formerly quite stout, he began to lose flesh rapidly; the face became pinched and wan in appearance, the feet and hands cold and bathed in clammy sweat. The pulse rose to 110 or 120, the temperature to 99 by mouth; the breath became quite offensive, the bowels obstinately constipated, urine scant and high-colored and loaded with phosphates and urates in excess. He answered questions only after frequent repetition if at all, and then only in monosyllables. When told to get up and assisted gently, he would obey slowly and more or less automatically, offering an apparently involuntary resistance to forced movements, which resistance was always more or less passive and never sustained.

During the early part of his attack he insisted upon frequent bathing, giving as his reason that he wished to follow the scriptural injunction to cleanse himself and put on fresh linen for his appearance before

the bar of judgment. Gradually he developed a state of increasing stupor, and had to be fed by hand as a child, but throughout the attack there were unmistakable evidences constantly present of a latent intelligence, which, though severely stunned, was yet alive. He remained in this condition, with only slight variations in the degree of stupor, for nearly four months, when he began to improve both physically and mentally. The facies became less fixed ; there was more play of expression, with an occasional flash of intelligent interest ; the appetite improved and he began to pick up flesh. This return period was marked by occasional fits of silent, tearful misery, but the improvement steadily progressed, and finally the stupor disappeared entirely, together with all evidences of delusions. At this writing he has been in a perfectly normal mental condition for nearly ten months. I have been totally unable to secure from members of the family the slightest evidence of any intimation observed by them which might have been construed as a warning or prodromal indication of melancholia in this patient, but he himself within the past few days has told me that his breakdown was preceded by several weeks of partial or complete insomnia, and that he suffered much from headaches during the two or three weeks preceding his explosive collapse.

Agitated Melancholia.—Miss D——, aged twenty-four, native of Ireland, single, and employed as a domestic, was seen by me November 14, 1894. I found her crying and protesting vehemently against my admission, stating that she did not wish to see a physician, but a priest, to whom she wished to make confession of some terrible sin which she had committed. She expected to be punished by the law for her misdeeds, and expressed a belief that I was an agent of the police in disguise sent to secure evidence to be used against her at an approaching trial. She expected to be condemned to death, which she would have looked forward to as a relief but for the fact of eternal damnation which would follow. This sin "could never be forgiven," it was unpardonable, but no amount of argument or persuasion was effective then or afterward in inducing her to state the nature of this sin or the time of its occurrence. There were other varying delusions corollary to this one, which was fixed and dominant. This patient suffered almost continuously for several weeks from hallucinations which were chiefly auditory. She heard the voice of God passing sentence upon her, or a chorus of voices which she said were those of angels crying out in condemnation and reviling her. The voices at other times were strange and unfamiliar, and she could not identify them. At other times the sound of bells tolling disturbed her. She at one time was possessed with the delusion that she was dead, stating that she had been condemned as a punishment to pass through life with a living, hopeless soul in a dead body. She cited as evidence of death her cold hands and feet, and refused to eat, stating that food was not necessary to keep the spirit alive and the body no longer needed food, being dead. Her manner was that of intense emotional agitation of a subdued character. The face was pale and anxious as a rule, but frequently it would become flushed, the lips tremulous, and she would break into tears. At such times she would walk the floor for hours, wringing her hands and clasping them in prayer, a picture of agonized suffering. Except when

under the influence of hypnotics she did not sleep at all. She lost flesh rapidly and became much emaciated, at one time presenting a physical condition which seemed critical in the extreme. Twice during her illness forced feeding became necessary, and throughout her attack my closest attention was required in combating nutritional failure and physical collapse. The temperature in this case, *per rectum*, remained persistently above 99 degrees, occasionally reaching 100 degrees for at least five or six weeks, the pulse during this time never falling below 100. For three months she did not menstruate at all, and it is of interest to note that the first symptoms of disordered health followed a suppressed menstrual period. These first symptoms, I learned from the sister, consisted simply of loss of appetite and strength, poor sleep, great nervousness and irritability of manner and temper, and frequent causeless crying spells.

There was no ancestral taint in the family history, and, except for an extreme homesickness, she having left Ireland only a few months previously, no cause could be found for her attack of melancholia. At the end of five months this patient had so far recovered that I advised a visit to the "old country," which advice was followed by her complete restoration to health.

DIAGNOSIS.—The more typical and well-established forms of melancholia are ordinarily diagnosed correctly and promptly. Such cases are so patent in their nature as to involve scarcely the possibility of a mistake. It is with the simple, the atypical, and the initial stages of all types that the greatest difficulty exists, and yet even here we have data of constant and proven value which properly interpreted render a diagnosis comparatively easy.

Among the affections to be considered in a differential diagnosis are certain states of physical disease, notably typhoid fever, meningitis of the tubercular variety, and syphilis of the brain. In the prodromal stage of typhoid fever, which in some instances is prolonged over a period of two or more weeks, there is a depression of spirits and an indisposition for exertion either physical or mental, with listlessness and apathy of manner, and slightly obtunded intelligence, poor sleep, headache, and loss of appetite, constituting a symptom-complex which is at times confusing. The absence of true affective or delusional melancholy or of special emotional disturbance, the absence of the characteristic facies, and the fact that the patient can be aroused to an interest in surroundings, and even to a normal though transient pleasurable emotional state, impossible in true melancholia, are facts which readily exclude that affection. After the characteristic temperature range of typhoid is established there is of course no further doubt, but a slight evening elevation of from one half to one and a half, or even two, degrees is not very uncommon in melancholia, especially the stuporous variety, and it is therefore of no value alone as a point in differentiation. Meningitis due to tuberculosis simulates, as is well known, the symptom-picture of prodromal typhoid fever and presents the same elements of confusion. The greater degree of mental hebetude and more marked insomnia and headache, with occasional hallucinatory or delusional states in meningitis, however, serve to render the diagnosis rather more difficult at times. The more or less quick onset of focal or general symptoms of

grave cerebral disturbance, with vomiting and perhaps active febrile delirium, promptly eliminates any element of doubt or hesitation which may have been present. Cerebral syphilis may be the cause of a mental condition scarcely distinguishable from true idiopathic melancholia. I have seen even the facies of melancholia in one or two such cases. Headache and insomnia are common to both; in both there may be extreme and unaccountable melancholy, and suicide is by no means unknown in the victims of brain syphilis. The intelligence is at times obtunded, and hallucinations and delusions, especially of nocturnal onset, are quite frequent in the active cases of syphilis. These symptoms are, however, not present together in many such cases, and the two affections are, as a rule, not apt to be confounded. The history of infections; the peculiar periodicity of the headaches, with nocturnal exacerbations; the difference in the insomnia, which affects the syphilitic, as a rule, during the first half or two thirds of the night, the opposite being true in melancholia; and the more or less rapidly developing evidences of focal damage in paralytic symptoms—determine the syphilitic nature of the case.

By far the most common error in diagnosis is that of mistaking melancholia for neurasthenia, so called. There is unquestionably a state of nervous exhaustion or prostration which exists independently, and which for the present we must recognize as an entity, but the term has been woefully abused. The relationship between neurasthenia, especially the hypochondriacal form (and nearly all neurasthenics are essentially hypochondriacal), and melancholia is a close one. The line of demarcation is, in fact, in some instances, almost indistinguishable. This is particularly true of simple melancholia. The psychical phenomena observed in neurasthenia, consisting of partial loss of the power of mental concentration, with intellectual confusion, vacillation, and lack of decision, disturbed emotional equilibrium, with morbidly apprehensive states of introspection, leading to the various phobias, which are close kin to and often almost identical with delusions or hallucinations, are strikingly suggestive of the mental disturbance in melancholia. In neurasthenia there are also insomnia and headache. Here, again, however, the insomnia differs, consisting characteristically of alternations of sleep and wakefulness or of light, restless slumber of the type which occurs in transient indigestion. The headache, too, is different in location, and rarely acute, often associated with vertigo, and easily relieved, temporarily at least, by rest or by a diffusible stimulant. The material points of differentiation are, however, the facies, the fixed superlative degree of melancholy and despair, and the tendency to suicide, which are conspicuously absent in neurasthenia. The neurasthenic patient has an expression suggesting fatigue. The melancholic patient, with muscles fixedly converging toward the central line in monotonous reflection of the troubled and despairing mind, eyes furtively downcast, reticent in manner or speech or stupidly silent, presents an entirely different aspect. The patient with melancholia never laughs; the neurasthenic may, and often does. The neurasthenic patient may be diverted, amused, and made, temporarily at least, to forget himself; the melancholic never. If, after all, diagnosis should remain in doubt, the safer plan is to consider the case one of melancholia—a decision

quite harmless in results, since the same general and even special details of treatment are sometimes equally efficacious in both affections.

Perhaps the most vitally important element entering into the question of diagnosis is the determination of the true significance of the melancholia; that is to say, whether the melancholia is a true idiopathic type or simply symptomatic and representing the initial stage of some other mental disease. Mania and general paresis both begin with comparative frequency with an initial stage of melancholy depression, with sleeplessness, delusions of self-degradation or persecution or disaster, and sometimes with impulses toward self-destruction. This period is usually a short one in ordinary mania, though in the insanities of double form—*folie circulaire* (circular insanity), in which mania and melancholia alternate successively—it may be more prolonged. In general paralysis of the insane the period or stage of melancholy depression may last for months, and unless there be present some of the many physical signs usually to be found even in the earliest stages of this disorder, the diagnosis may become quite difficult. There is more vacillation in the mental state, however; as a rule, in paretic dementia the delusions are not systematized, and cropping through those of the depressive type there will often be found the more familiar and characteristic variety of an expansive or grandiose nature. We are, however, dependent chiefly upon the physical rather than the mental symptoms in distinguishing the two, while in ordinary mania the element of time, involving as it does only a few days, will determine the true nature of the case. In dementia, which is sometimes mistaken for melancholia with stupor, there is an absence of the characteristic facies, insomnia, and headache of melancholia, the appetite is good, nutrition is well sustained, the somatic functions being normal except in cases with associated physical disease. There may be melancholy depression, but it lacks the systematized constancy observed in melancholia; the delusions are more apt to be silly or childish in type, and are not fixed, as a rule. The memory is poor or lost, according as the case may be recent or advanced. Finally, suicide is not a factor to be reckoned with in dementia, since the mental state is one rendering the patient incapable of elaborating any plans involving deliberation.

Katatonia (*κάτα*, down, low, and *τονος*, tension), described by Kahlbaum in a paper published in 1874 as a generic form of insanity, and recognized by Spitzka, Kiernan, and others, closely resembles in certain of its phases melancholia of the stuporous variety. As a matter of fact, the two are regarded by many alienists, among them Seglas, Voisin, Tamburini, and Chase, as being fundamentally identical, though the description of Kahlbaum certainly does not conform in the symptom-picture presented of stupor alternating with mania and dementia, cataleptoid or choreiform states, and convulsions, with intensely theatrical manner, verbigeration and delusions of belittlement, to ordinary stuporous melancholia. We may, indeed, have cataleptic conditions with senseless verbigeration in melancholia attonita, and delusions of belittlement in any form, but never an alternating mania or dementia nor convulsions, except as accidents. Fortunately, katatonia is comparatively rare, and does not therefore often confuse a diagnosis.

Prognosis.—The proportion of recoveries in melancholia is larger

than in any other form of insanity. Clouston states that 54 per cent. of 1000 cases tabulated by him recovered. These, be it remembered, were asylum cases. In private practice the percentage is much higher. Of 46 cases carefully recorded by the writer, 40 were restored to normal mental health, 2 died by suicide, 1 with symptoms of grave cerebral disease, and 3 passed into chronic forms. This series included all varieties.

The prognosis varies somewhat with the age of the patient, the cause, and the type tendency present, but it depends in much greater degree upon the promptness with which the condition is recognized and treated, and upon the circumstances, financial, domestic, and otherwise, which affect the conditions attending treatment.

The age of the patient affords certain data bearing upon the question of prognosis which are not without interest and importance. Occurring at puberty, melancholia in girls is a comparatively tractable affection, and in contrast with the more obstinate condition observed in males developing melancholia at this age. Such male patients, if neglected, exhibit a rather marked tendency to develop a chronic form of the disease or to pass into a state of dementia. It is of importance to note, too, that girls affected in early life with an attack of melancholia, and recovering from the initial seizure, should be watched with special care in after-life, especially at parturition and the climacteric, for symptoms indicating a recurrence, since they exhibit more than the average susceptibility to relapses at such periods. The prognosis in melancholia of any type in male or female occurring in old age or in one manifesting evidences of premature senility is of graver significance. The endarterial changes and retrograde tissue metamorphoses peculiar to old age are factors tending to modify unfavorably and in decided degree the prognosis.

In melancholia dependent upon or associated with grave physical disease the prognosis bears somewhat of a ratio to the associated physical condition. In post-operative melancholia incomplete recovery is the rule. Such patients may recover from the melancholy depression of the initial attack, but an eccentricity, or even a fixed delusion, may, and often does, persist. Melancholia due to or associated with sexual perversion is in my experience a most intractable variety.

Heredity, if the case be one of essential and not of symptomatic melancholia, does not at all necessarily affect the prognosis unfavorably, nor does the sex of the patient enter as a factor *per se* in the consideration of this aspect of the subject, although relapses, or, rather, second and third attacks of melancholia, are more frequently observed in females than in males. On the other hand, secondary degenerative psychoses—such as dementia, for example—are more frequently observed in males. The affection terminates in one of three ways—recovery, permanent mental alienation, usually in the form of secondary or so-called terminal dementia, or death. The case may terminate fatally through suicide, always a source of danger and one never to be disregarded. Inanition, the result of prolonged and persistent abstinence from food, may cause death, though such a cause of death carries with it a grave arraignment of the physician in attendance, since it is comparatively easy to practise any one of several methods of forced feeding in these cases. Death from

intercurrent physical disease of febrile or inflammatory type is less common than in mania or in dementia. I have, as a matter of fact, noted with curious interest an apparent relative immunity in these patients from such diseases, with the single exception of phthisis perhaps. Finally, death may terminate a melancholia with symptoms indicative of grave cerebral disease. I recall the case of a patient, for example, who died in a second attack of melancholia, there having been an interval of five years of normal mental health between the two attacks, with symptoms strongly suggesting a low-grade cerebritis or meningo-encephalitis. Unfortunately, I was unable to secure an autopsy and the nature of the cerebral affection was not determined.

An acute form of melancholia may persist through a period of many months or even years, retaining its original symptomatic character, and thus becoming a case of chronic melancholia. Usually in such cases there is progressive qualitative and quantitative mental deterioration and decay, the termination being a true dementia. In a few cases the patient makes an apparently good or fair recovery, but is noted afterward as being peculiar in some particular—unduly emotional, eccentric simply, or perhaps evidencing some more or less marked degree of moral perversion in contrast with previous character. It is difficult, if indeed it is possible, to classify such cases, though the fact that an obsession exists is usually quite apparent.

The tendency to relapses, or, to be more accurate, to recurrence of attacks, is a fact to be considered in every case of melancholia, for, while a second attack does not necessarily occur, it is by no means uncommon. Clouston in the series of cases already mentioned (one thousand in number) found one-third to be suffering from second attacks. Here, again, the fact that these statistics are from asylum practice must be considered. Relapses occur in private practice in probably less than 20 per cent. of all cases. I know of no facts upon which may be predicated in any given case a promise of immunity in this particular, nor do I know of any characteristics of the initial attack or stigmata by which a recurrence can be foreseen.

The duration of melancholia is dependent very largely upon the promptness and intelligence with which the case is recognized and treated. Properly treated, the simple cases will recover in from six to twelve weeks. The agitated variety of the disease is more resistant and more apt to be complicated with some physical disturbance prolonging the attack, which sometimes extends over a period of six months or a year. Stuporous melancholia of the less intense type ordinarily lasts from three to six months, even under active treatment. In melancholia attonita we find the most obstinate and persistent of all forms of the affection, and this variety more than any other is especially prone to develop secondary degenerative phenomena leading to a dementia of the terminal type.

TREATMENT.—While it is probably true that melancholia in some instances disappears spontaneously—*i. e.* without medical treatment—it is equally true that the *vis medicatrix naturæ*, which is so faithful an ally of the physician in physical, and especially zymotic diseases, is utterly unreliable in affections of the psychical functions. I know of no form of mental disease which more imperatively demands active and intelli-

gent treatment, and it is even more emphatically true that no other variety of insanity offers such encouragement to the therapist in the brilliant results which may be attained by the careful and intelligent application of trustworthy methods and measures of relief in these cases. No other form of insanity is of equal interest or importance to the general practitioner, because of the fact that not only is he the one who has the opportunity to recognize the affection in its incipiency, which is the period of most successful treatment, but equally for the reason that he himself in many cases may easily qualify himself to treat the patient successfully. A very small proportion of melancholiacs require asylum or institution treatment. Indeed, as a matter of experience and fact, the majority of such patients are injured and their recovery is seriously jeopardized by commitment to such institutions. There is much to be said, I readily admit, from a theoretical or an *a priori* standpoint in favor of asylum treatment, especially such as is illustrated in the well-equipped and well-managed sanitaria which have become so numerous in recent years. The systematized life, regulated diet and exercise, appropriate baths, trained attendants, etc. represent an environment which is apparently almost ideal. Very many patients affected with melancholia are, however, fully and appreciatively cognizant of their surroundings, which fact is a sufficient explanation of the harm done by incarceration in an institution with association with other insane inmates. Such patients are deluded no more than you are by the terms "institution," "sanitarium," "retreat." They are not only conscious of the stigma, but in the very nature of their affliction such stigma is exaggerated and magnified a hundred-fold. I am fully satisfied, from a comparison between the statistics gathered from asylum reports and my own experience, and that of others favoring and practising the home treatment in such cases, that the average duration of attack is much shorter than in asylum cases, suicides and accidents less frequent, and relapses or recurrences far less common. After the condition has become chronic or passed into a terminal dementia the asylum or institution treatment is of course demanded, but in acute cases and the early stages of all cases the patient should be given the advantage of the home plan.

Before undertaking the treatment of these cases at home two conditions at least should be demanded positively and imperatively: First, absolute control over the patient's personal and domestic life; and, second, the presence constantly of an attendant or nurse, preferably one with special training. In all cases it is advisable that the patient be isolated from the rest of the family, leaving the personal influence and domination of the physician and nurse free from harmful and meddlesome interference. The factors of marital or blood relationship and affection sway and bias disastrously the judgment and influence of husband, father, or sister, and should therefore be eliminated as far as possible. If treated at home, the patient's room should be changed in location and furnishings, with the double object of interrupting painful associated ideas and the establishment of stimuli to object-consciousness in surroundings. Removal to a neighboring house or town, involving as it does a radical change in environment, is at times of material advantage. The presence of an attendant or nurse day and night in

these cases is made necessary on account of the danger of suicide, which is so characteristically present as to be almost essential to the diagnosis. The attendant should be cautioned most carefully with regard to this danger, and should be constantly mindful, not only as to the patient, but also with regard to himself. In attempting his own death the patient is often utterly indifferent as to the hazard to others involved in the method he employs. Sometimes the suicidal propensity develops as an impulse originating in or suggested by an opportunity. A window unguarded, a match left carelessly exposed, a pocket-knife, a table-fork, have each afforded an opportunity seized on the moment in my own personal experience in the cases of three patients. In the debilitated cases, presenting evidences of malnutrition and anæmia, perhaps from preceding and possibly causative physical disease, and in the agitated cases with delusions of præcordial fear and rapid pulse, enforced rest and a generous diet should be insisted upon. The patient should lie abed until ten or twelve o'clock, and should rest lying down for one or two hours in the afternoon. Nearly all cases of melancholia, by the way, are anæmic.

Gentle massage is at times helpful. Physical exercise in these cases should be very moderate in both character and amount. A low diet is rarely indicated in melancholia, although in certain cases dependent upon or associated with marked or obtrusive digestive disturbance the quantity and quality of the food must be regulated. As a rule, the appetite is poor or lost or the patient is indifferent to the necessity of eating, or, it may be, purposely avoids food. Sometimes this results from a delusion that the food has been poisoned, or that by eating he robs some one else more worthy, or that he is exhausting the world's supply by eating. In other instances the refusal of food is deliberate and with suicidal intent, the patient determining to starve himself to death. In such cases it is usually easy to overcome the objection to food by gentle but firm persuasion. If food is placed in the mouth by an attendant, the patient will masticate and swallow a full meal, perhaps all the while objecting and protesting, but in a passive and purposeless way. At times this resistance is more obstinate, and forced feeding must then be resorted to by rectal enemata or by the stomach-tube, or, better still, by an ordinary soft-rubber catheter introduced through the nose and down the throat, through which liquid food in concentrated form may be administered without harmful struggling or psychical damage to the patient through more or less violent resistance.

The physical inactivity and torpor of circulation present, together with the indifference to surroundings manifested by melancholic patients, make it necessary that the nurse should exercise some special oversight and care with regard to the warmth and clothing of the patient.

The drug treatment resolves itself into three indications: The relief of insomnia, the correction of visceral derangement and disturbed secretions, and the relief of psychic pain.

For the insomnia we have a large and satisfactory supply of agents. I do not believe it is wise or of advantage to trust to nature or the so-called simple remedies for the relief of this insomnia. With very many melancholic patients the difficulty exists, not in getting to sleep, but in remaining asleep for a sufficient length of time to afford rest to a tired

and exhausted brain. Some reliable hypnotic should be used at once. Personally I prefer trional above all others. It has been free from injurious or disagreeable effect or action in my experience, and is more certainly reliable than any other. The dose is from ten to twenty grains in water shortly before retiring. The addition of an equal quantity of sodium or potassium bromide is sometimes of advantage. Trional does not seem to lose its hypnotic action even after prolonged continuance, nor does it tend to establish any drug habit. Occasionally the sleep induced by a full dose is somewhat alarmingly prolonged, but this is a rare idiosyncrasy and of no harmful significance. I am aware, of course, of the published accounts of untoward results which have been reported in a few instances as following the use of this drug, but my personal observation and experience after using the drug freely for four years in a large number of cases, are positively contradictory of any such testimony. Only a short time ago a patient of mine suffering from insomnia from alcoholism recklessly took six powders within two hours, each containing fifteen grains of trional and twenty of potassium bromide. She slept nine hours, woke up much refreshed, and beyond a slight vertigo and some feeling of weakness in the legs for a day or two, she suffered no ill effects. Next to trional I have found chloralamid in doses of twenty to thirty grains in the form of an elixir very satisfactory; a second dose, however, is often necessary, which may be given after an interval of two hours. Sometimes it fails altogether. Sulfonal in ten- or fifteen-grain doses, given just before retiring, in a cup of hot milk or cocoa, is also quite reliable. It occasionally affects the circulation, however, depressing the heart markedly. I have seen two cases of serious cardiac weakness with cyanosis follow (*propter hoc*) a ten-grain dose in melancholia. It is especially contraindicated in those with primary weakness of the circulation and in the agitated type of melancholia with delusions of præcordial fear, and in those patients exhibiting the peculiar rapid pulse rate observed at times. Other hypnotics which may be used, but which are inferior to the three mentioned, are somnal, chloralose, hypnal, paraldehyde, amylene and chloral hydrate, or the alkaloids of hyoscyamine.

The correction of digestive disturbances or abnormalities is of essential importance. Constipation, which is almost invariably present in some degree, should be relieved and kept relieved, but drastic cathartics should be avoided. A reliable preparation of cascara sagrada acts excellently well, especially if supplemented every week or so with decimal doses of calomel or occasional doses of aloin in combination. A mild aperient water is sufficient in some cases. The active saline waters, such as Hunyadi, Rubinat, and others of this class, are exhausting and harmful in my experience. Although digestive disturbances may in some instances be due to hypersecretion of some one of the digestive agents, as a rule in melancholia the opposite is true, and we are dealing with a deficiency. If the effect is stomachic in location, it is often found associated with gastric dilatation or with some structural disease of this organ, and direct treatment should be based upon the findings. By far the most common anomaly is that found in the intestine, and especially the duodenum, and here the large group of intestinal antiseptics serve an invaluable purpose. First in the list I place the subgallate of bis-

muth in doses of five to seven grains after meals. Almost as efficacious is salol in the same dose. I often give them in combination, adding, if anæmia is strikingly present, an equal quantity of ferratine. Betol, β-naphthol, naphthaline, resorcin, and others of this group may be employed. Occasionally, where the physical indications are quite evident, particularly in the lithæmic cases, I have used dilute nitromuriatic acid in ten- to twenty-drop doses, well diluted, *before meals*, with excellent effect. Thyroid extract, lauded by some as affecting favorably the depression in stuporous cases, has not proven of any value, so far, in my hands, though I have not given it any extended trial, and a further experience may modify more favorably my present opinion. Protonuclein has seemed to exert an influence, in some cases decided, upon the debility and anæmia, but I have not observed any modification of the mental state therefrom, except such as was purely incidental to the improved nutrition.

Headache in simple melancholia may be an annoying symptom, requiring some measures for its relief. Electricity in the form of galvanism, one electrode applied over the forehead, the other to the back of the neck, sometimes acts almost magically, not only in relieving the headache, but also in promoting a less despondent mental state. The electrodes should be carefully applied, preferably through a rheostat, and the current strength should not exceed three or four milliampères, and ordinarily it is better not to exceed one and a half or two. If delusions are present, electricity should not be used at all, and its use is contraindicated in stuporous and agitated cases on this account. The same advice applies with regard to the employment of the hypodermic syringe or any other unusual procedure for the same reason.

For the relief of the psychic pains nothing equals opium, used in the form of the aqueous extract in doses varying from one tenth to one fourth or one half grain from one to four times daily. It is almost a specific in its action in relieving the mental suffering and depression. The dose must be determined experimentally by the effect in each case. Beginning with an eighth of a grain twice or three times daily, the dose may be increased or diminished according to the effect upon the depression or stupor. Great care should be taken not to discontinue its use prematurely, and it is safer to prolong it in small doses, even after all symptoms have disappeared, for some days or even longer. In one case under my care the patient kept up the drug in exactly the same dose, gr. one eighth twice daily, for nearly seven months after he had been discharged as cured, with no bad effect and not the slightest indication of any drug habit. As a matter of fact, I have yet to see the first case of opium habit as a result of the use of this drug in melancholia. The physician, however, should dispense the drug himself in these cases, as an added precaution and for the advantage in moral effect. Codeine in somewhat larger doses is sometimes a satisfactory substitute. I often add this drug in doses of half a grain to the nightly dose of chloralamid with very happy effect. I have never used tr. opii or morphine, finding the others mentioned satisfactory and free from constipating tendencies or danger of a drug habit. I have tried cannabis indica, but without much positive evidence of effect, good or evil. I have used the bromides of sodium and potassium occasionally with distinct benefit, especially in

agitated melancholia with motor restlessness and excitement. If continued for any length of time, however, the bromides interfere with digestion and nutrition, and undoubtedly may aggravate and deepen the stupor present. More injurious still, and even dangerous, are the alkaloids of hyoscyamus which are sometimes used to control and quiet the patient in paroxysms of melancholic frenzy. If given at all, the physician should remain with the patient until satisfied that no danger exists. Strychnine, on the other hand, is a drug of very positive value in the treatment of melancholia, particularly in the convalescent period of stuporous cases. Although apparently antagonistic in physiological action, strychnine and opium may be given conjointly, with perceptibly good effect from both. Either the nitrate or sulphate may be employed, in doses varying from gr. one sixtieth to gr. one thirtieth or one twentieth, twice or three times daily.

Hydrotherapy is much lauded by its advocates as a measure of relief, the truth of which I am unable to confirm or deny, never having given it any extended trial. The skin should be kept active of course, and cleanliness preserved by ordinary bathing at sufficiently frequent intervals. It is scarcely necessary to call attention to the desirability of investigating the status of all the viscera, the kidneys, heart, and lungs especially ; and in this connection the hallucinations and delusions referable to the viscera, which are often present, deserve attention. Such hallucinations and delusions often have an important therapeutic significance, analogous in some respects, as I have stated elsewhere, to the localizing significance of the auræ in epilepsy. The delusion of præcordial fear may be, though such is not the case with any frequency, associated with some cardiac disease. I have found in cases exhibiting this delusion with no demonstrable lesion, but with a rapid heart and pulse, with or without agitation, that much symptomatic and perhaps more important relief may be obtained from the use of digitalis in small doses, or of sparteine, which, combined with rest in bed, sometimes lowers the pulse quickly to the normal. The toxins of Bright's disease may cause melancholia which is perhaps purely symptomatic, but none the less actually existent and demanding treatment, which should, of course, have reference to the cause.

MANIA.

By WILLIS E. FORD, M. D.

Definition.—Mania is one of the great subdivisions of insanity, and the term is used to designate those cases which are chiefly characterized by excitement or exaltation, both of body and mind. Every case of mania must, therefore, conform to the general definition of insanity, which is a disease of the brain causing a departure from the natural or ordinary manner of thinking, feeling, and acting.

When a layman uses the terms "lunatic," "insane," "crazy," or "out of his mind," he usually means a mania, for this is the form of insanity which is perhaps most frequently met with by the non-professional observer. While typical cases may be easily recognized by a person not expert in mental disorders, there are certain forms of mania that are hard to define or even to demonstrate to courts of justice. From a medico-legal standpoint this is, therefore, one of the most interesting of all the forms of insanity. To the practising physician it is the most important of all the divisions of mental alienation, because it is the initial disorder which often terminates in other forms of insanity, and because it is more amenable to treatment, or at least requires an earlier discrimination and a more ample treatment of the individual in order to have the disease run its most favorable course.

This state of abnormal, continuous, mental excitement or exaltation, which is the essence of mania, is not generally a painful one, being in this respect entirely different from melancholia. In the main, patients do not seem to be distressed by the disorder, though it is by no means true that all the hallucinations and delusions that accompany mania are pleasurable. Patients having the milder form of the disorder express themselves as feeling well, better than usual, and they even boast of the agreeable sense of strength, ability, and power which they have.

The morbid exaltation usually affects the emotions, the intellect, and also the muscles of the body. In all cases there is a restlessness of body, a lack of self-poise, an inability to sleep, and the absence of power to rest accompanying the mental perturbation. In fact, the entire organism is thrown out of balance; there is a want of co-ordination; the functions of the mind, the muscles, the glandular system, the digestive apparatus, and the emunctories of the body are disturbed. In the height of the attack the patient moves about constantly and aimlessly, and his ideas crowd forward for expression in a deluge of meaningless words.

The violence of the attack, its duration, and the direction of the intellectual perversion are elements which determine the form of mania under consideration. The simplest division of mania is that of acute, sub-

acute, and chronic, and under these terms may be classified all forms of the disease. A distinction, however, is made, and justly too, between the types of the acute mania, so as really to create a fourth division called "simple mania," which, while it is usually acute, is yet devoid of the general characteristics of acute mania as described in the books.

In the State of New York, of the whole number of cases of insane patients in asylums, 273 per 1000 are classified as mania. Of this number, 104 of acute mania, 69 of subacute mania, and 86 of chronic mania, and the rest of recurrent mania make up the list. Many of these cases, indeed, which are now called chronic mania, have formerly been classified as acute mania, and the same cases may in the future pass into a hopeless state of terminal dementia, so that it is impossible to give the relative proportion in numbers of the various divisions of this disorder. Simple mania is, perhaps, rarer than the other forms of mania, and is the one which is most frequently misunderstood by the friends of the patient and by courts of justice. This is for the reason that there is often an absence of well-marked delusion, which, after all, is the only single test to be applied to any form of insanity.

In England the number of cases of mania in all cases of insanity admitted into public asylums is about 35 per cent. In this country, and more especially in this State, the proportion of cases of mania to the whole number of cases admitted to asylums is about 31 per cent. It must be remembered that such statistics are not of the greatest value, for the reason that there is no uniform classification of insanities subscribed to by all alienists.

The older writers divided insanities into mania, melancholia, and dementia. With few exceptions the writers of twenty years ago exhibited quite the reverse of this simplicity in making an infinite variety of insanities, based chiefly upon the clinical features of the disease. Thus monomania, homicidal mania, pyromania, kleptomania, etc. were the terms used to characterize the leading delusions of the maniac. Some changes also have been brought about in our theories of the classification of manias by the elucidation and definition of certain forms of insanity as separate disorders which were formerly classed among manias, such as general paresis, paranoia, and alternating mania.

Some writers have also, and with a good deal of justice, it seems to me, taken epileptic mania or epileptic insanity out of the general class of manias and have described it as a disorder by itself.

Etiology.—The progress made in all departments of medicine during the past few years, and especially in our knowledge of the causation of diseases in general, makes more prominent than ever the fact that the causes of mania are obscure and indefinite. This is the more to be regretted because dependent upon this fact is the practical problem of the prevention of mania. There are many factors which enter into the causation of all forms of mania, and these factors must be studied separately and estimated at their full value in order that the prognosis given may be at all correct and that the treatment may be effectual.

No one, however, will doubt that heredity plays a most important part as the remote or predisposing cause in the production of mania, as of all other forms of insanity. This is true whether we admit the possibility of the direct transmission of the disease, or whether we say that

the physical structure of certain individuals, while perfect of its kind, is endowed with less resisting power to disease, and that especially the nervous system of some persons more than others is susceptible to the influences which produce great exhaustion and disturbance of mind. In either case it amounts to the same thing, and statistics may be quoted to show that about 30 per cent. of all cases of mania have a distinct history of heredity.

It is difficult to state exactly what is meant by the neurotic tendency or neurotic inheritance, and yet it is plain that certain families have a greater tendency to the development of nervous and mental disorders than have their neighbors living under apparently similar conditions. So, also, we know that a man with sound lungs is much more likely to die of phthisis if his ancestors in any considerable number have died of this disorder. It is true, too, that in certain families the lungs are strong or the circulatory apparatus is unusually perfect, thus securing a greater average length of life than in other families. In the same way, the muscular system of certain families is more perfect, and the physical endurance is greater, than the average of their fellows. And so the nervous system of some families seems to maintain its integrity in the face of all sorts of shocks, accidents, and strains, while in other persons lesser causes produce the most disastrous consequences.

This tendency to a weakness or instability of nervous organization is seen very early in life. Irritable, peevish children, who are prone to become delirious whenever slight febrile disturbances of childhood come on, or who have convulsions or outbursts of uncontrollable temper whenever they are in ill health, and who are unmanageable beyond the average child during the little illnesses common to their age, are more likely to develop mental disorders later in life than those who do not show these early symptoms. These indications are often of the most serious moment. If the family physician, keeping in mind these facts, should advise in the education, and even in the choosing of the occupation and the kind of life generally to be led by such persons, the mental failures in life would undoubtedly be fewer than they now are.

The civil condition seems to exert a marked influence upon the causation of insanity ; thus, it is found that a very much larger percentage of unmarried people become insane than of married people. Whether this is due to the conditions of the emotions, or to hardships, or to the repression of instincts which are natural, or to the greater temptations which beset celibates, is merely a matter of speculation. It is undoubtedly true that where the marriage relation is wholesome and mentally agreeable the discipline which it imposes upon those emotions which are most deleterious helps to make a symmetrical character.

Egotism, selfishness, cruelty, want of control of temper or of the appetites are more commonly seen in men who live alone. Sorrow and disappointment are better endured when shared by a faithful companion. I am inclined to think that it is more in the discipline of the emotions than in any physical effect that we must look for the cause of this disparity in number between the married and the unmarried.

In the same category of causes we find it laid down that civilization has a marked influence in producing insanity. This must, of course, be taken with a great deal of allowance, and, as in former cases, statistics

are hardly reliable. Here come in the two factors moral and physical, though the moral factors, it seems, have some offsets in this case.

If in civilized life the temptation to certain vicious habits is greater, and the excitement of political, financial, and religious discussion is fraught with danger, it must be admitted that, as a rule, the emotions and passions are far better controlled by civilized than uncivilized men. This is certainly true as respects anger, unreasoning grief, abject fear, and unmitigated hatred, but probably one factor—namely, anxiety—is more efficient than all other moral or mental causes put together in producing insanity. Then, too, there may be some fault in statistics, for the apparent increase of insanity in civilized life is certainly due, in part, to the fact that it is more commonly recognized and that the individual cases are more generally known. So of the predisposing causes, aside from heredity, very little can be definitely said. While it has often been claimed that the changes of the seasons have their influence, yet this and other causes can probably be more rationally accounted for by the consideration of the environment and occupation as well as the dietary.

Among exciting causes the emotions are chief factors in producing insanity. One must admit, however, that other forms of nervous disease, such as neurasthenia and chorea, are much more frequently seen as a result of disturbed emotions than are the pure insanities.

If, however, the intellect is bright and the will rather weak, so that sensory impressions are profound and the feelings easily disturbed, then it is easy to see how the emotions may ultimately dominate the will and intellectual perversion may result. Mania, however, is not so common an outcome of this condition of dominant emotions, especially in civilized conditions, as is the opposite depressing condition of insanity called melancholia.

As to love and fear, I have never seen any cases of mania that I could ascribe to these factors alone. Continued anxiety, however, of an intense sort—seen more commonly in this country than in any other civilized nation—may, and certainly does, have a most harmful influence. It is easy to see how this may occur. The emotions have so much influence over the digestive apparatus, the circulatory system, and the excretions of the body that general ill health may first come on and the bodily powers become weakened, and, finally, the power of resisting a pervading anxiety may be lost, and insanity result from physical disease, but remotely from the moral cause.

It seems to be continuous mental anxiety, and not intermittent periods of mental strain and worry, that does the most harm. Some years ago I collected the statistics regarding the lives of stockbrokers in a certain city, and was surprised to find that nearly every person who lived a sober life and continuously studied the ups and downs of the money market failed either mentally or physically in a short time—less than a dozen years—ultimately disappearing from active life. On the other hand, the men who were operators of great skill and coolness, and who lived regularly most of the time, but occasionally gave way to the drink habit, and disappeared several days at a time on account of helpless drunkenness, lived longer and had fewer mental disasters. This, of course, can not be construed into an argument in favor of drinking, even occasionally, but was to my mind a very strong indication of the benefit coming

from the occasional complete relaxation from intense mental anxiety. Frequent vacations passed in the woods or at the seaside without social duties, and where, temporarily, men could revert as nearly as possible to primitive life, even for short periods, would, I am convinced, be much better. Protracted anxiety without rest breaks more men than does hard intellectual effort.

Very few young men break down mentally during their college days, nor, if they are well equipped, do they often break down mentally in the practice of their professions. The cases of this sort that I have seen have usually been brought to mental disaster by attempting to accomplish without adequate preparation or without ordinary mental endowments what other men easily accomplish. Nothing can be more certain than that the isolated instances that do occur show a weakness in the individual and the error of attempting to thrust forward such youths toward positions they have not the mental ability to fill with honor.

Among the physical causes, general ill health, from debilitating sickness or want of proper nutrition or the excessive use of alcohol, perhaps ranks first. This is so well known as a clinical fact that any attempt to show the modus operandi is out of place here. The steady drinker, who finds it necessary to increase his drams, is the one who soonest undermines his health and endangers his mental stability, while the man who occasionally "sprees it" is not likely to fail so soon; yet this possibility of being wholly overcome by temptation indicates a weakness of character which in other directions may be as serious in its effects as is actual insanity. Syphilis also is a direct cause in many cases by producing its characteristic brain lesions.

The narcotic drugs do not usually produce any form of mania, though it is possible that the general health may be so undermined that any form of insanity may supervene. The chloral habit, which is not so common as in former years, produces but few cases of mania. The habit of taking cocaine, which of late years has been steadily increasing its number of victims, seems to be a more dangerous habit than that of alcohol or chloral or narcotics generally. That the cocaine habit is very seductive and very dangerous cannot be doubted, but the immediate effects of the drug, though exciting and exhilarating to a great degree, soon pass away, leaving a permanent state of depression. While there are not statistics to bear me out in the assertion, it is the general impression that forms of mental depression are more likely to be produced by cocaine than genuine mania.

Any drug in order to produce a habit must give a pleasurable sensation, either immediate or remote. The yielding to this desire for pleasurable sensations slowly but surely enthralls the will, and makes the victim a slave to his emotions and sensations; and just so far as he ceases to be governed by the dictates of reason and conscience he becomes the prey of accidental circumstances, and therefore likely to succumb to any form of nervous or mental disorder. This is not more true of mania, however, than of other diseases.

The recent acquisition to our knowledge relating to the subject of auto-infection has been one of the most striking advances in internal medicine. It seems to me that many of the obscure forms of nervous diseases which have heretofore eluded our skill will be found to depend

directly upon imperfect oxidation and the excretion of the worn-out detritus of the body, and to the imperfection of that process of secondary digestion which transforms foods into healthy tissue. To a less degree we shall probably find that sudden nerve storms amounting to mania are often due to the actual poisoning of the cerebral masses by the presence in the blood of these toxins. We certainly know that the heart and the arteries are greatly deteriorated by the presence of such toxic elements in the blood ; and it is not at all improbable that changes in the cerebral tissue are produced by the same blood conditions that cause exudation about joints and inflammatory conditions in white fibrous tissue. The benefits of the bromide salts may be due to the presence of the alkali as much as to the action of bromine itself. This most interesting phase of the subject has not been sufficiently worked out, up to this time, by those who have studied internal medicine. Gross injuries to the head which produce local inflammatory softenings, or which produce thickening of the membranes covering the brain, thus shutting off the bloodvessels which come from them to nourish the brain, can readily be appreciated as producing mania.

It is in this connection that brain surgery, or perhaps cranial surgery, promises the most brilliant results in the future. Cerebral hemorrhages, while profoundly disturbing the activity of the brain and even obliterating consciousness for long periods, are rarely followed by maniacal attacks. Other forms of insanity, however, frequently follow.

Pathological Anatomy.—It is probably true that there is no sole, pathological, distinctive lesion of the brain or of its membranes which we can say always produces mania, and which is never present without mania ; and yet it is also true that when the brain is examined after death, there are some general and a few distinctive pathological changes found in the brain. It is probable that the appearance of the brain after death does not wholly correspond with the actual condition during the progress of the disorder, for the tissues are generally anæmic rather than hyperæmic.

The condition of the smaller bloodvessels, especially of the capillaries, is perhaps as distinctive as any of the lesions observed upon dissection. The pia mater sends the arteries into the cortex, and they do not break up into capillaries until they have reached the fifth layer of the cortex close to the centrum ovale, and then the branching of the capillaries takes place upward to the surface layer. Where these arteries divide into capillaries there are often found aneurysms ; and this is quite characteristic where the disease has lasted for a long time. The capillaries may show fatty infiltration, and it is not uncommon to see fatty degeneration also. This infiltration is a characteristic change, making the bloodvessels look as if enclosed in an irregularly folded wrapping.

In melancholia the capillaries of the cortex often show a similar coat, but it is much wider and contains pigment granules. In advanced stages of insanity the change is again different from that of mania in that the bloodvessels show an irregular thickening or callous degeneration. The changes in the membranes themselves are not constant, but when the insanity has been of a violent nature or of long duration, there are usually found evidences of distended sinuses, with inflammatory thickening and consequent adhesions.

The nerve tissue itself in mania is infiltrated, so that there is often the appearance of granule-cell formation approaching yellow softening. Throughout the gray cortex there are found capillary hemorrhages after the severer attacks, and in other cases a simple exudation here and there of a few red corpuscles.

These changes in the vascular system, so frequently found in the brain after mania, are also accompanied by alterations in the ganglion cells of the cortex. These latter are more frequently found in the middle and lower frontal convolutions toward the anterior central convolutions. This is the portion of the brain which is most prominently developed in men, giving the human brain its characteristic shape.

If we distinguish five layers of nerve cells of the gray cortex, the most marked changes are found in the third and fourth of these layers. The contraction or coagulation of some of these cells seems to occur, and this is seen chiefly at the base of the cells around the nucleus and in their multipolar prolongations. Sometimes a fatty infiltration of the cell itself around the nucleus is seen. In the fifth layer, which is provided with multipolar spindle cells, these changes are not seen so frequently, though there is sometimes found exudation of blood-corpuscles to the extent that it marks the division between the gray cortex and the central white very distinctly.

Anatomical lesions, however, are so numerous and differ to such an extent that it is impossible, as yet, accurately to determine, from the examination of the brain, the kind of insanity which has preceded.

The reparative processes which we must assume to be going on when the acute symptoms begin to subside, with even extensive lesions, may be removed or their appearances so modified that the dissections made later fail to reveal the exact condition during life. The changes, for instance, in the vascular system, which leave a detritus along the lines of the capillaries or actually choke the smaller bloodvessels, may disappear during the period of convalescence, and probably do in most cases. The pathological changes in other organs than the brain are not so distinctive as to require special mention.

SYMPTOMS.—The symptoms of mania vary to a great degree, dependent upon the type or stage of the disease. There are, however, certain general symptoms which accompany all forms of mania, and which have been alluded to as conditions of exaltation or excitement both of body and of mind. There is great latitude also to be allowed because of individual peculiarities or accompanying physical disorder. The age of the patient, his social position, his cultivation, as well as his occupation and his general physical state, must be taken into account, since all these elements tend to make each case of mania somewhat different from every other.

All cases of mania, however, may naturally be classified as either (1) simple mania, (2) acute mania, (3) subacute mania, or (4) chronic mania.

(1) SIMPLE MANIA.

Simple mania is not so frequently met with in institutions for the insane nor in courts of justice as are the other forms of mania. It consists of a delusional state in which there is great exaltation of mind and

restlessness of body, without, perhaps, any fixed delusion, though the acts of the person are so strange and unlike his former methods as to mark him as insane; and the person may not be incoherent or be so far beyond his power of self-control but that moral suasion may have a decidedly restraining influence.

Simple mania may come on at any age, though it is more frequently seen just after the period of adolescence, and again between the ages of thirty and forty-five. It is more likely to come on slowly, and, unless it passes into a stage of acute mania, there is rarely an attempt at physical violence. The attack is preceded, sometimes for weeks, by a condition of listlessness, a tendency to inattention to ordinary duties, and by an apparent abstraction of mind that is rather depressing. The patient complains of feeling badly, and sometimes of having pains in the head, and usually of sleeplessness. After a short period the patient becomes restless, impatient of restraint, inclined to be talkative, and perhaps actually does or talks about things which he would formerly have considered improper, lewd, or indecent. When this marked muscular activity, accompanied by a sense of well-being or mental exaltation, comes on suddenly and to such a degree as to be a decided change from the former condition of the patient, and remains for some weeks or months, the diagnosis of simple mania can be made without any more marked symptoms.

This sense of unusual well-being, with expansive notions of importance and elation of spirits, combined with a certain levity which is often paradoxical, chiefly distinguishes this form of mania. During its course the appetite is often markedly increased or perverted, so that the quantity or variety of foods taken may be truly astonishing. The tongue is furred, the secretions of the mouth are perverted, and the breath is very offensive, while the condition of the kidneys and bowels is very sluggish. All this does not seem to be due to indigestion, but rather to that perversion of all the secretions which goes with conditions of profound emotional disturbance. Even with this increased amount of food and apparent digestion, there is a steady loss in bodily weight, which often progresses so far as to constitute a danger: this is not true to the degree that it is in acute mania, and comparatively few patients die in the attack.

During this whole course there may be no actual violence shown, though it is usual that opposition is met by violent outbursts of temper and a determined resistance which may amount to maniacal violence. The tastes of the patient are very often perverted to the extent that he not only eats and drinks things which he formerly did not like, but he dresses differently. He is likely to be slovenly, whereas he may have been very particular in dress, or he suddenly becomes more smart in his appearance, while he formerly had been careless. But the greatest change is seen in his moral perversion, often becoming quite the reverse of his former state. This has led many writers to distinguish a moral insanity which is really simple mania with insanity of conduct, without marked delusions.

It is probably true that the weak points of the normal character of the individual are those which are thrust into prominence in simple mania, thus indicating the loss of mental inhibition, which is really the

essence of the disorder. Most of us have certain traits of character which must be kept in abeyance, and which, if held by a firm will, rarely show themselves in conduct or in speech. The loss of this control allows these ugly traits to step forward and to characterize both speech and action.

I know of one case in which, during the past twenty-four years, there have been five separate and distinct outbreaks of simple mania, such as I have described, characterized mainly by pernicious activity, exalted notions of self-importance, with an exaggerated idea of her attractions, and with a tendency to obtrude herself upon the notice of men, and, finally to cause scandal of the most disagreeable nature as the outcome of the attack. She is a well-bred woman, yet in these attacks solicits the attention of men, and if this is not responded to, she accuses persons of attempting to seduce her, or of having made proposals of marriage to her, or of having grossly insulted her in various ways. Then the sweetness and plausibility which characterized her first efforts changes to the vilest abuse and the grossest misrepresentations, often accompanied by scenes of great violence, and sometimes with homicidal threats. It is doubtful whether during any of these attacks a jury would have convicted her of insanity, and yet upon her own statements during the attack she would be a vile, lewd woman unless her insanity was admitted.

The course of the disease may be modified by any incidental bodily ailment, by the age of the individual, by his environment, and by his medical treatment. As a rule, these cases do not recover under three, and often are prolonged for six, months, even when a favorable termination is ultimately secured; but in many instances, if the attack is prolonged, it passes into a more acute type, and finally into chronic insanity.

An attack of simple mania is often observed to precede by several years another and perhaps a more serious form of disorder; so that, taken as an indication of the mental stability of the individual, such an attack must be looked upon as one of the most grave character. These cases do not often come into criminal courts, but often cause endless business complications and litigations.

It has been observed that upon the death of a wife or a husband, the middle-aged survivor frequently develops this phase of mental disturbance, so that many ill-timed and injudicious marriages are the result really of this mental disorder. These same persons, if they have wealth, often markedly change their habits of thrift and economical living, and spend money freely, lavishly, and foolishly.

Thus a man of sixty, of fair mental capacity, without hereditary taint, succeeded by his own industry and sagacity in accumulating a large property. He was of a serious turn of mind, very attentive to his religious duties, though always reckoned as rather a sharp man at bargains. No one, however, doubted his honesty or his veracity. His wife suddenly died under most distressing circumstances. There was every outward manifestation of great grief and sincere sorrow. Within a few months, not more than four, however, he was noticed to dress better than he had ever done, to exhibit an elaborate politeness to his friends, which was foreign also to his former custom, and to give very largely to

charitable enterprises: from a humble worshipper in the congregation he became talkative, rather pushing, and a demonstrative exhorter.

He bought some fine horses, though he knew nothing about horses, and had never cared for them. But now he was very critical. He sold a valuable team for almost nothing because of some slight defect, buying another pair within a few days, and ordered a rather expensive and showy equipage.

He was much more agreeable to his neighbors in this condition than he had formerly been, though he took certain liberties which perhaps his age excused him in doing. Soon after it was discovered that he was making love to a person whom he had known and cordially disliked all his life, and, notwithstanding the vigorous protests of his family and the scheming of his grown-up children to prevent it, he married the lady and began a social career. He had never known or cared about society before this.

During this time he ate well, and did not show the muscular restlessness common to these cases, and it is not known how well he slept. He was the cause of much gossip, some slander, frequent misrepresentations, and a great deal of ridicule in the community, and a source of unmitigated sorrow and mortification for about a year, when he passed into a state of profound dementia.

I have seen many instances of this disorder in young people with unstable nervous organizations, who have been hard pushed at school or who have been allowed to enter society too young.

One case of a girl of seventeen who had a slight attack of unilateral chorea interested me very much several years ago. She had a most unstable nervous organization, with inheritance from her mother's side strongly predisposing her to insanity. She showed the approach of the disease soon after the chorea had disappeared by an undue familiarity with servants, and a tendency to loquaciousness which was entirely foreign to her nature. From this she became disobedient and irreligious, though her upbringing had been most carefully conducted by pious intelligent parents. She became careless in dress, rather inclined to expose her person, and, though there was no marked obscenity in her conduct, being a full-grown woman this was extremely mortifying. As the disease progressed she exhibited tendencies that were still more intolerable to her family. She asked young men to call upon her and insisted on receiving them; and finally visited stores and offices and talked familiarly with clerks and employés, inviting them to her house.

At this time I was consulted, and told the family that she must be secluded, and told the patient what the tendency of her conduct was. She would promise to do better, and when with her attendant, who always accompanied her out of the house, she was manageable. Sometimes at home, however, she would secrete herself for the purpose of causing great alarm in the household, and would laugh heartily when she witnessed the distress of mind she had caused in her family, who had been searching for her for hours. She wrote letters of the most friendly, and even amorous, character to men whom she knew only by sight, and would signal passers-by if she could do so unobserved from her window.

These main features of her case continued for about six months, when

she recovered. She has remained well for many years, and is now a bright, attractive young woman.

The youngest case I have seen was that of a boy of fourteen, who first became restless, sleepless, excitable, and then unmanageable, loquacious, profane, obscene, and untruthful. His conduct so shocked his well-bred parents, and was such a change from his ordinary manner, that they sought advice. He was anæmic and thin of flesh, though he had a voracious appetite. His father devoted himself to the care of this patient for some months, keeping him in the open air, and securing such exercise as to produce bodily fatigue and consequent sleep.

In this case the prescription of a dog which should constantly accompany the boy was most efficacious in bringing him back to his normal condition. This attack lasted nine months, has been followed by no relapses, and the boy has grown up a well-educated, level-headed young man.

Simple mania does not always terminate in recovery, at least of a permanent nature, as the following case will illustrate: A man of forty who had become a successful business-man, possessed of considerable property, known as a conservative man in business circles, with a well-established character for honesty and business integrity, who was free from the drink habit or any of the grosser vices, became suddenly impressed with the idea that his business could be extended so as to control the trade in his line in the State of New York. His busy season was approaching. He executed all orders with more than usual promptness and ability. He made money, and his domestic relations were very happy. He became more talkative than usual, slept much less than usual, spent much of his time at night in travelling upon the cars—which he had not formerly done—and showed a certain levity, jocoseness, and familiarity of manner which was quite unnatural to him, and yet was not vulgar or disagreeable. Indeed, he seemed brighter, more alert, more companionable, than ever before. Though he slept little, he explained this fact by saying that he required less sleep than most men; that his family were peculiar in that regard; that he could sleep soundly upon the cars or in a carriage, and in that way get all the rest he desired.

Within a month it turned out that he had bought and started a new business in two other cities, and that he was negotiating for a large business in a third city, and had plans for still further enlargement. When his family found this out and remonstrated with him, he was most plausible in his arguments justifying his conduct. He gave instances from the lives of distinguished men, quoted Scripture to justify himself, and asserted that he had "grown and developed," that he had "raised himself above the ordinary rules of business which had heretofore hampered him, and that hereafter he would astonish the community and the world by the brilliancy and the magnitude of his enterprises." He was willing to discuss his private affairs with everybody, and talked about his family relations to people who were comparative strangers to him. He was strong and voluble in the doctrinal points of his religion, and yet began to indulge in smoking, in drinking, and in loose expressions, and to acquiesce in sharp practices.

It would have been difficult during any part of this time to have

established before a jury the fact that this man was insane. Indeed, there was at no time any single delusion which could be pointed out as establishing a case of insanity, and yet for the three months that this thing continued he was insane, and suffering from what we would term "simple mania." On recovery he had very little recollection of the details of his business or of the particulars of his conduct. He has since relapsed, after some years of successful business life, into recurrent mania.

While the causes probably operate in this milder form of mental disorder that produce graver ailments, yet physical deterioration is markedly less, and, indeed, a fair condition of physical health may be maintained throughout the attack. There is a loss of flesh, and, if the excitement subsides rather suddenly, there is a good deal of exhaustion apparent. But owing to the fact that the nerve storm is not so severe, the dangers are less, the recoveries are proportionately greater, and there is less wreckage done than is seen in a more violent disorder.

Statistics, however, cannot be found bearing upon these points, mainly because the cases are not, as a rule, sent to asylums or are not sent until the type is changed to a more pronounced one. I believe these cases of simple mania occur more frequently than is generally believed, and that if a diagnosis could be made early, the attacks might be cut short or the patient placed where he could do no harm.

(2) Acute Mania.

This is the most important division of all the insanities, for the reason that it is more amenable to treatment, is likely, if neglected, to leave the subject in a hopeless condition, and is the most startling to the friends and to the community.

It is an explosive or fulminating type of disease, characterized by great exaltation of all the bodily functions and running a definite course, generally with few complications, and terminates fatally in but 6 or 8 per cent. of cases; in recovery in about 60 per cent. of cases; and in chronic insanity in about 30 per cent. of cases.

It is the disease of youth and early manhood, and it not infrequently seems to the friends of the victim to come like a bolt out of a clear sky. Its victims are commonly of an excitable temperament; women are more frequently attacked than men, while more cases develop in the spring and summer months than at other seasons.

There are certain prodromata which are unfortunately overlooked in many cases until after the attack has asserted itself. These signs are, chiefly, general ill-health, often an obscure dyspeptic condition with depression of spirits and a sense of impending danger, accompanied by sleeplessness which lasts for a few days only or, it may be, for several weeks, and is followed by great excitement of body and mind, which speedily culminates in a delirium and an incoherence and a violence which make it necessary to remove the patient to an asylum at once. A few cases do not seem to have this period of mental depression, and, again, a few cases have a considerable period of simple mania without delusion, which after a few weeks passes into the violence of acute mania.

Acute mania usually reaches in a few days its climax of violence, in which the patient has no appreciation of his condition or surroundings, has no care of his person, is incoherent, violent, shouts, tears his clothing, and attempts to injure himself by throwing himself against the wall or furniture, breaks or destroys anything he can lay his hands on, refuses food, refuses to sit down or remain quiet, walks wildly up and down gesticulating, and requires constant watchfulness to prevent injuries to himself. The mind may be in too confused a state, too chaotic a condition, to have any one delusion expressed, though the fear of poisoning, which is so common a symptom, may be persisted in so as to make the administration of food and medicines very difficult in this state. Unless this delusional fear of poisoning does exist, the patient is apt to eat and drink voraciously, taking three or four times as much food as an ordinary man, and digesting it without difficulty. Though the breath is offensive, the skin dry and harsh, and the secretions foul and sluggish, yet this is not an indication of indigestion, nor should it in any way interfere with the chief object of treatment—namely, the administration of large quantities of suitable food.

The excitation of all the faculties causes such a disorderly tumult that incoherence is natural and definite delusion rare.

Early in the attack there is frequently a tendency to alcoholic or venereal excesses, while later on unspeakable lewdness of conduct and expression characterizes the condition. Hallucinations are not frequent, though sensory illusions are very common, as are mental illusions. The patient acts in the most agitated manner a dream which is most vivid, grotesque, and fantastic. A single word excites a whole train of ideas, which struggle for immediate expression, and the sound of some word in the attempted expression summons up an entirely different train of associated ideas, which in turn crowd tumultuously forward. Thus meaningless words having a somewhat similar sound are repeated over and over again.

The following stenographic report is a fairly good example of the speech of an acutely maniacal man:

(*Pounding his chair with his fist.*) "I don't care where they sit. I will be there. He has been under the United States Government all his life, and never had a day off. Never been out of prison. Wish you never was born; every one. (*Striking chair.*) Yes, goes right through here. Some of them people are just going. I know all of them. No, I don't want to be down all the time. I am right. Yes. Taking a walk around the block."

Another example is given where there is more coherence:

"I want an examination of my nerves and some medicine, and I want to know why I am detained from my business, at your earliest convenience. Because this stud of hell, he says that I came here by force. I was sunstruck, betrayed and brought here. You will grin out of the other corner of your mouth, Fred Smith, now remember that; may God have mercy on your soul! He is a damned rascal if there is one this side of hell. Have you got anything in your pockets for me? He has got my Elgin watch, that damned rascal has. Why don't you feel bad? He is, he is a damned rascal, and the Lord knows it. I will make this building tremble if I don't have my liberty, I am not going

to be detained from my business for ever. I can go out any day and get my living as a medical, botanical doctor. That's my name and my nature. I cannot tell you my true name if you want to know. They call me everything the devil can think of. That damned rascal has got my money. He will never get his just desserts until he has been pounded by a cat-o'-nine-tails. This is the only hell God Almighty ever made. I don't believe that Beelzebub, prince of devils, knows anything about the misery I have been through in this —— —— institution. It is called a hospital. It's a —— —— asspital, that's what it is. It's the damnedest place. When I get out of here I shall prosecute New York State for one straight million of dollars, and perhaps for five millions. For fifteen or twenty years, night and day, I have been pounded and kicked with the same energy that a man would take a hammer and pound a bull with. Have you got a shirt on your back with a pocket in it? If you have, you have no business to wear it. I am the inventor of it, the shirt with a pocket in it. I want my seat changed at the table. I don't want that damned nigger to sit near me. He's the damnedest rascal this side of hell. If he ain't the devil himself, he's the devil's own brother."

The urine and fæces are frequently passed involuntarily, and the sensory illusions impel patients to destroy their clothing, to denude themselves, and often to smear their persons with filth and even to eat their own dejecta.

The fats of the body disappear very rapidly under this process, for, besides the great muscular excitation, there is usually a slight rise of temperature—of from one to two degrees—and without the greatest care exhaustion may speedily ensue. The first fear, therefore, is that of exhaustion and death, to which, however, not more than perhaps 7 or 8 per cent. succumb. After several days, amounting sometimes to two or three weeks of this intense delirious excitement, a favorable change is indicated by a lessening of the muscular restlessness, by increased hours of sleep, and by periods of half-appreciation of his condition. When the physical and mental excitement terminates suddenly, there is little hope of the permanency of the recovery, and a relapse of the same nature is to be apprehended or a period of depression indicating an alternating mania or folie circulaire.

If the case is to recover, the delusional condition passes off without leaving any single fixed delusion. So long as the mind flits from one fancy to another there is more safety than in a persistent delusional idea of even a slight character. No doubt the character of the delusions is influenced by certain bodily sensations from which the patient suffers. Thus, the idea of poisoning is suggested by the perversion of the secretions caused by prolonged talking and shouting, or by the indigestion produced by improper food or the ravenous and brutish manner of eating. Or, again, the notion that he is played upon by electric batteries —a very common form of delusion—undoubtedly is suggested by certain sensations of prickling and tingling in the skin or by certain ringing or explosive sounds in the head. Illusions of sight and of hearing may also be the direct result of intracranial pressure, so that the ringing in the ears is interpreted as being voices—sometimes of friends, sometimes of spirits—directing his actions. Nothing is more common

than the illusions of sight, the patient mistaking the identity of those about him, and talking to strangers as if he had known them, calling them by their first names, often the names of his family or of friends, some of whom may have been dead for some years.

But, whatever the form of delusion, whether based upon the hallucinations or evolved out of the acute delusional state previously described, if it remains as a single delusion, well defined, governing the actions of the patient, not improving as the bodily health begins to improve, then the case is liable to terminate in chronic mania.

If, after a few weeks of excitement as described, a period of comparative tranquillity ensues, and the patient becomes indifferent to his surroundings, and gains in bodily weight and in his power to sleep at night, and the mind shows less activity, and a torpor ensues, then the most unfavorable of all conditions of insanity may follow—namely, terminal dementia, that hopeless form of insanity which dooms the living man to a mental death.

These, then, are the various methods of termination: Recovery, about 60 per cent.; chronic delusional insanity or mania, about 10 per cent.; terminal dementia, about 20 per cent.; and death, about 8 per cent.

Inasmuch as this is the most violent of all forms of insanity, it would be natural to expect that the damage done to the mind and to the body would be greater, and would occur more speedily, than in any other form of mental disorder. The chief thing, therefore, for the general practitioner to remember is that even a short delay in instituting proper means for the prevention of great exhaustion and for proper medicinal treatment must be fraught with the most serious consequences, and that the method of the termination of the disorder will largely be determined by the promptness and vigor with which the treatment of these cases is begun.

Some years ago a man of twenty-two years, a machinist, was married in a small country town, and within the first ten days afterward was noticed by his wife's people, with whom he was temporarily living, to show strange, morbid fancies, especially talking of his unworthiness to marry their daughter and of his inability to support her. Two or three days later he stopped work, appeared at the house in a very talkative, excited state, and it was suggested to the family that he had been drinking. I saw him the same night and advised his removal to an asylum, his condition being that of simple mania without much violence, telling his friends that this probably preceded a most serious state. The advice, however, was not taken, and after several days of loud and incoherent talking in a changed and unusual manner, with loss of sleep, he became suddenly violent, boisterous, noisy, destructive of clothing, incoherent, with extreme muscular restlessness, so that he had to be bound by ropes and in this condition carried to the nearest asylum. With proper care and management this excitement subsided in two weeks and recovery gradually supervened.

I believe, however, that all recoveries from such violent attacks of mania are but comparative, and that no person who has thus suffered—for we will say three months—will return to exactly his former condition. He is queer in some way or in many ways; his disposition is changed to

a greater or less degree; he has lost his ambition; or there is a marked change in some of his former tastes. None of these things alone would indicate insanity, and a patient may be well and sane, yet he has not reached the same state of sanity that he had prior to his attack.

One evening, some years ago, I came into my office to find three gentlemen sitting there, one of whom was writing in rather an excited or hurried manner at my desk. They were evidently well-bred people, the gentleman at the desk being more than an ordinarily intellectual-looking man. He rose as I came in and asked me if I was the doctor, and said in a laughing way that one of the three was insane, and asked me if I could tell which one it was. I replied at once that he was the insane man, to which he took no exceptions, but at once began the history of his life, of his recent trouble with the firm of which he was a member, and made the statement that he thought he had been "out of his head a little," but that he had gotten over it now, and that his belief, which he admitted was a delusion, that a distinguished man of his acquaintance was about to come and assume charge of his business, had entirely left him. He was well-bred, a graduate of Yale, and a very successful man; and, though his statements were somewhat exaggerated, yet his friends in the main coincided with what he said. He had been so violent at home, for a few hours only, that he barricaded the doors of his house, though he afterward explained that he had been threatened with incarceration in some institution, and his books were in such a state that he must complete them before being forcibly taken away from home; he had, therefore, resorted to this means. He made very light of all this, and said he was glad to get away from the excitement of his business, and that a good night's sleep would clear him up. His manner was very nervous; he walked rapidly in the room, was anxious to explain small particulars; he talked of his domestic matters in a way that one gentleman rarely talks to another; and confessed that he had not slept for many nights. I advised him to go at once to an asylum, saying to him that I believed that this one fixed delusion—that his distinguished friend was coming to take charge of his affairs—was still believed by him, though concealed. This he denied, saying that, whereas he had once believed it, he did not believe it now. Without the evidence of this delusion it would have been impossible to sign a certificate committing him, though I was thoroughly convinced that it was the beginning of an attack of acute mania, and told him so.

The next morning I was summoned to the hotel with great urgency. Found him lying in bed, his face to the wall, refusing to speak; the furniture was much disordered, and his friend, who was a large and powerful man, showed every outward sign of having just emerged from a free fight without honors. He wrote on a slip of paper and handed it to me, saying, "—— (naming his distinguished friend) will be here in six minutes." Signed, J. C.

I at once said to him, "Your delusion has returned, and is even worse than it was yesterday, and you now believe yourself to be Jesus Christ, and we shall be obliged to take you to an asylum." He sprung from his bed like a tiger and attacked me, though I was fully prepared for him, and received him with such an impulse from my own body as to stagger him. It was very difficult to overpower him. The police

were called, the porters of the hotel assisted, and he was dressed and taken to an asylum. There he was violent, incoherent, tremulous in his muscles, very restless; at times noisy, profane, and obscene in speech for three weeks. Then he became tranquil, his delusion faded, and he recovered inside of three months. This man took the trouble to call on me afterward and to express his gratification that he had been dealt with in a perfectly frank, sincere manner—that no attempt had been made to deceive or cajole him or to trick him in getting him into the asylum, for he remembered, or at least had a vague remembrance of, many of his former words and acts.

Acute mania rarely runs its course in less than three months, and it is often six months; and it has been said with, I believe, much truthfulness that if a patient does not recover from acute mania inside of six months, he will go a year and a half at least without recovery. I heard this statement made early in my medical experience by a distinguished psychologist, though there was no reason given for it, and I do not now know of any reason why such should be the case, yet in numerous instances has this statement been verified.

Acute mania may attack its victim at any age, but just as melancholia and dementia are more common in advanced life, so the state of morbid exaltation is more commonly seen in early life. Many cases occur just after puberty—in girls from fifteen to eighteen and in boys from sixteen to twenty. Whether it is a want of readjustment of the bodily forces after the establishment of new functions, or whether it is due to the fact that the emotions, which at this age undergo a profound enlivenment, may so disturb the intellectual faculties as to bring on this explosion, I am not prepared to say.

I have seen many cases of acute mania in the young in which erotic tendencies seemed to predominate, and this, too, in properly brought up youths where I am sure no nastiness of mind or filthy practices preceded the attack. The assumption that these erotic expressions and the lascivious conduct are evidences that masturbation caused the disorder is wholly unwarranted. Indeed, I have never known a case of insanity that could be fairly attributed to this cause alone or even chiefly to this practice.

The habitual repression of the emotions, which is an essential part of good breeding and which finally becomes a habit, does not indicate an ablation of the emotions, so that when insanity ensues, removing the inhibitory power, there is often a very great rebound. The particular direction which this emotional excitement may take may be determined by peculiar bodily sensations, and not at all by the previous habits of the patient. As a matter of fact, I have seen the most dissipated and vicious persons in an attack of mania exhibit few lecherous tendencies.

In the severe cases, where the secretions become so vitiated that food is refused or rejected, great bodily weakness comes on with rapid loss of flesh, and, unless nourishment is administered artificially, so as to in some measure sustain the strength of the patient, the gravest apprehension may be felt.

A persistent diarrhœa or dysentery sometimes comes on, while in a few cases passive congestion of the lungs develops into a pneumonia, and death follows. Generally, however, a fatal termination is the result of

extreme exhaustion, the pulse becoming faster and weaker, and at the last a marked rise of temperature may occur. A fatal termination usually results, if at all, in the first or second month. If the course of the disease carries the patient to the fifth or sixth month without any abatement of symptoms, and if the bodily health shows a marked improvement, and the patient begins to regain some of the flesh he had formerly lost, the tendency of the disorder is toward chronicity. The danger is, as the excitement subsides, a certain amount of self-control returns, and the incoherence diminishes, that there will remain either a fixed delusion or a delusional state constituting chronic mania.

If the case progresses favorably, the indications that recovery is to take place may be looked for by the second month, and even much later, in the lessening of the excitement, the subsidence of violence, and the fact that the patient ceases to lose flesh, and begins to improve in physical appearance, while the incoherence may remain and the illusions continue, there is less violence and an observable return of sleep.

The necessary condition seems to be that the mental symptoms keep pace with the improvement in bodily health. As the improvement continues there will be periods of quietness, when the principal thing to be observed is mental dulness and listlessness, which almost approaches dementia, and yet is not accompanied by its physical signs. This feebleness of mind is very characteristic of approaching recovery. In this state delusions fade, illusions disappear, good sleep is obtained, and with it there continues to be a steady improvement in the bodily health. From this condition of feebleness and mental inactivity the patient emerges after several weeks to resume his normal mental state.

If these processes of convalescence are continued without relapses, even though very slow, occupying months, even a year, the most hopeful view may be taken of the case. The instances of sudden restoration from an attack of mania to the comparatively normal mental condition are very rare, and are not looked upon as favorable for ultimate mental stability.

(3) Subacute Mania.

Some authorities do not distinguish this form of mania at all, yet while it embraces a much smaller number of cases proportionately, and is clearly a less important division than either of the others, it has a few distinctive features, and may properly be described by itself. It is more slow in its development, has less mental excitation, and a tendency to chronicity; but there is less violence than in acute mania, and rather more physical excitation or bodily restlessness than is seen in chronic mania. It more nearly resembles chronic mania, however, in the character of the mental symptoms than it does acute mania. The prodromata chiefly relate to the physical condition of the individual. These are symptoms of physical exhaustion or depression, with a disturbed state of the digestion, and often marked dyspeptic symptoms, with loss of sleep. During this time the patient is sluggish in his movements, not so alert either mentally or physically, and complains of feeling pains about the head and in various parts of the body. The tongue is furred, the bowels are sluggish, and the skin is unnaturally dry and harsh. In

a few days, or perhaps weeks, this is followed by an unnatural excitement, an increased restlessness of body, and an elation of feelings.

The mental symptoms come slowly, seeming at first to be mere exaggerations of certain natural traits of character, which, increasing in intensity, develop into a delusional state. The delusions are rarely of such marked degree or of such fixed and lasting nature as in chronic mania. As the departure from the normal mental state is more gradual and is not accompanied by such violence, so the shock of the onslaught is not so startling and is less easily recognized.

The mind is very active, and the memory of facts acquired long before seems to be reawakened. Many persons recite volubly what they had learned in childhood, or recall correctly what they have heard and have apparently forgotten years ago. The morbid restlessness of the mind sometimes enables the person to solve problems that were quite impossible in health and to aid in inventions that are remarkable in ingenuity. The most striking fact, however, is the phenomenal feat of memory so frequently performed.

Hallucinations rarely, if ever, occur in subacute mania, and as the predominant mental conceptions are exaggerations of natural traits of character, such as pride, ambition, or suspiciousness, which, while they may be absurd and fantastical, are expressed with clearness, the ideas are not strictly delusions. There is often an exaggeration of vicious tendencies, so that these patients malign their friends with great loquaciousness, and at times become actually violent. This tendency to make spiteful comments to those about them, and to use their wit—sharpened a hundred-fold by the mania—to lash their attendants, is common. There is, nevertheless, a certain amount of self-control, though sometimes this is lost in what would seem to be paroxysms of temper. Acute mania sometimes replaces this state or is superimposed upon it.

In many cases of so-called alcoholic mania the taking of alcohol is but a symptom of this disorder, though it is often looked upon as a cause, and again as a distinctive type of disease itself. Alcoholic delirium is characterized by hallucinations, and the withdrawal of the alcohol brings about a speedy change; whereas if the condition of subacute mania exists, complicated by alcoholic excess, the withdrawal of the stimulant does not cure the disorder, and alcoholism must be looked upon as a symptom only. Gross exhibitions of drunkenness or of venereal excesses and gambling by persons of previously good habits and of well-established characters must be regarded as indications of maniacal disturbance.

As illustrating certain of these points a case may be recited: An acquaintance, who was a business man, came to his store one morning and asked the clerks about a certain electrical apparatus which he said had been put into the building during the night. He said he was present when the workmen were there, and had seen the crowd of people collected outside and had heard various comments by the passers-by. He did not attend to his business as usual, but walked excitedly about the store and frequently peered out of the windows, as if expecting some one, and asked his clerks strange questions about the electrical apparatus. He pointed out to the clerks the people on the streets who were manifesting an unusual interest in his affairs, and remarked that the

crowds of people were unusually large, though in fact there were few passers-by and no demonstrations that showed any interest in his affairs. His physician tried to persuade him to go home, but he would not, saying, he had business of very great importance that he must attend to at once, though he did not seem able to state what the business was. He restrained himself so as not to show much excitement, though his lips and hands trembled; but he showed no particular depression of spirits. His wife said that for two or three weeks he had been out of health and had been depressed in spirits, and had asked her if she did not think "the neighbors were down on her," and whether she had not better "buy goods" in a neighboring store in order to show "friendliness." He had slept but little, often awoke in the night and walked about the house, saying he thought he heard somebody talking outside. In the morning he vomited and could not eat his breakfast, looked pale, and his hands were cold. His clerks had not noticed anything unusual.

On the evening of the day first mentioned he explained the occurrences, and stated that several times within a few days men had been about his house trying to put up some kind of apparatus—that he could not state what the apparatus was nor connect the object with himself personally. He was coherent, and the only change in his manner was his alertness of mind and body—the perfectly apparent effort to control his emotions and to state his case clearly. He talked much better than usual. He did not resist our efforts to keep him in the house, but was perfectly docile, though he had to be reminded frequently not to talk too much. He was given hypnotics, and slept fairly well during the night. The next day he showed more eagerness of manner, was more indifferent to matters of business, and begged to be protected from certain doctors, whom he named, who were coming to "open his skull" for the purpose of removing pain. When told that it would not be done, he expressed no emotion, and did not refer to the subject again in several hours.

He offered to give over his business into the hands of the neighbors the next day, saying that he thought it would be conducted better by some one else than himself. When the neighbors came in to see him, he talked in a perfectly rational manner, and all they noticed was that he was excited. He talked about electrical apparatus—made absurd propositions about his business to his wife and physician, though, when asked about figures he was able to give a correct account. He laid in bed as directed, though he had to be frequently reminded that he must not get up and that he had promised to stay in bed. He took whatever food was given him, and when told that he was threatened with some mental disturbance, and that he must exercise his self-control, he said that he would do it, though for a long time he had thought he would "bring up in a lunatic asylum."

He was kept secluded, attention was given to his physical condition, sleep was procured by hypnotics, and in a few weeks he recovered and remained well afterward. I charged him with drinking, and he frankly said that he had taken some alcohol for a few days previous to the outbreak, but that he had been depressed in his spirits and had the apprehensions mentioned for several weeks before, and that he was not accustomed to taking stimulants at all.

Another case, which did not terminate so happily, was that of a man

of twenty-seven who was a broker. His parents were notified one day by the managers of the hotel where he boarded that he was acting strangely, and a member of the family went to the town where he lived and induced him without much difficulty to shut up his bucket-shop and go home. He had the appearance of a person who had been drinking too much, though his family said he was not given to this vice. It was afterwards learned, however, that for two or three weeks he had shown some excitement which was attributable to alcohol, and that he had been given to sexual excesses at the same time. He looked sick, his muscles were tremulous, he was thin in flesh, his skin was pale, and it was with a good deal of effort that he restrained himself in the office so as to sit still and answer questions. He was perfectly cheerful, said he felt "first rate—everything was coming his way"—yet he showed no regret that he had been obliged to leave his business nor any anxiety to get back to it, and did not demur to the story which was told by his sister, but seemed rather pleased with the interview. The thumb of the right hand was very much inflamed in its whole length, and the skin was gone for an inch and a half by three quarters of an inch in width. This I was told was caused during the two nights preceding his return home by his standing on chairs and bureaus and rubbing the wall as if he were rubbing out chalk-marks on a blackboard. He made no other disturbance, and said he was doing a large business in the stock exchange. He had not slept well for several weeks.

At the close of the interview he sprang out of the chair as if shot up by a spring, and in a most excited and nervous manner began to adjust his overcoat to go out. During the interview he showed considerable self-control. He was perfectly coherent. There was no marked delusion, though a delusional state of mind was perfectly apparent; for between the answers to questions he would often relapse into a condition of one thoroughly preoccupied in mind. His lips would move, and he was apparently talking to himself, though he made no sound. By the expression of his face and his complacent appearance and frequent nods of satisfaction it was evident that he was in an exalted and delusional state. His hands and feet were cold, his head was hot, his pulse was 120, his tongue was thickly furred, his breath very offensive, and the pupils of the eyes were slightly dilated. I advised his removal to an asylum, which the family refused. I then advised them to take him home and secure the constant attendance of two persons to care for him, and to keep him in bed as much as possible, and indicated a line of treatment. On his way home he was quiet enough until he met some boys whom he did not know, but who were talking and laughing rather loudly, when he at once threw his hat on the sidewalk and executed a jig, waved his hands in the air, laughed immoderately, and was promptly landed in the police station. He was released and taken home, where with judicious care he steadily improved. During the daytime he would often sing and whistle or recite poetry, though in this he was easily checked. He would go through the motions of a baseball game, would call off quotations of stocks, sell pools, and if he saw a picture of machinery that he knew about—such as a locomotive—he would at once imitate as nearly as possible the motions of the machinery, making with his lips the sound of escaping

steam, blowing of whistles, etc. At night he would sing or whistle most of the time unless he was given hypnotics. During all this time he was easily checked in any performance by requests from his mother or father that he should stop. He would answer questions correctly about himself, and if a neighbor stayed but a few moments in the room, he would maintain his gravity and, to all appearance, his sanity. He would lie on the bed for hours with his eyes closed, though not asleep, with an elated and pleased expression upon his face, which he would explain by saying that he was thinking of something funny. He showed no violence, and after three weeks began to sleep without hypnotics, showed great self-control, and, in fact, seemed to be recovering. He ate very well during all this time; indeed, would take a meal at any time, night or day, when it was brought to him, but, in spite of all this, he lost considerable flesh, and was scarcely able to walk at the end of six weeks. He often manifested erotic excitement, but if his father or mother came into the room he at once desisted and manifested shame. For a short time he passed his urine and fæces in the bed. After two months he was able to walk out with his father in the street, and began to gain in flesh, but soon became more sleepless, and developed the delusion of poisoning, which necessitated his removal to an asylum in order that food might be administered.

The fact that in these cases there remains so much self-control has often caused them to be classified as "delirium" or "hysteria," or as cases of moral perversion. The distinction between this and simple mania seems to be that the latter is mainly a condition of loss of inhibitory power, while subacute mania is a true maniacal exaltation.

A large proportion of cases of subacute mania recover, though acute mania may develop out of it and run the usual course, or delusions may develop and chronic mania remain. The usual course, however, is about the same as that of acute mania.

(4) Chronic Mania.

Chronic mania is rarely a primary affection, but is developed out of some more acute mental disturbance. When acute mania has run through its ordinary stages, and the bodily health of the patient has been restored, and the violence of the attack has not been sufficient to disorganize the brain to the extent of producing dementia, a stage of comparative permanency of the disease remains, usually accompanied by a delusion or by a delusional state, and is called chronic mania. The patient, having passed twelve months in the condition of acute mania, has by many authorities been classified afterward as a case of chronic mania, unless the symptoms of dementia are sufficient to obscure the mental activity.

Thus the case which we have spoken of before may in time come to be classified as chronic mania, the division between the two designations being arbitrary. The characteristic feature, therefore, of chronic insanity is a well-defined delusion, or delusions which govern the speech and the actions of the individual, and may be based upon hallucinations of either hearing, or sight, or taste, or of the tactile sense; but, whatever the basis, it rules the life of the individual.

A man with chronic mania may have so far recovered from the state

of mental excitement or exaltation as outwardly to show little signs of mental disorder unless the subject of his delusion is touched upon. Many cases are of gradual development, and have no pronounced initiative of acute mania, but settle into a fixed delusional state from the subacute attack of insanity. Sometimes the original disorder is melancholia, and not mania at all; but the pervading depression of mind having passed away, and a single fixed delusion having taken its place, chronic mania is thus developed without any acute symptoms of exaltation. These are the cases that remain for a long period in asylums; or they may so successfully conceal the delusion, and have such control of themselves in other directions, that they are allowed to remain at liberty in the community. But they are the most dangerous, perhaps, of all classes of insane people; for the delusion, which at one time has apparently a feeble hold upon the individual, may in conditions of slight ill-health or emotional excitement suddenly become so pronounced as to make the person violent, incoherent in speech, and most determined in conduct. When it is known, therefore, that a person is suffering from an insane delusion, unless the character of the delusion be fixed and of a nature that will not interfere with the safety or the rights of other people, it is not safe to allow him to remain at large.

In order to constitute insanity a delusion must be shown to be the result of disease of the brain, and not the result of misinformation or of any theory. Such delusions must therefore mark a departure from health, and must be clearly attributable to sickness. Every one has delusions, and it is only when there is no other reason for the false belief than sickness, especially if this false belief is based upon hallucinations of hearing or of sight, that this of itself proves the insanity of the individual.

Some years ago a lawyer brought a client into my office for the purpose of having me make an affidavit as to his sanity, upon which was to be based an application to the court to have the committee of his person and estate discharged. He said he had always known the man, and while, at one time, he had believed he was insane, he was now certain that he ought to have the management of his own affairs, and he was very anxious to right what he considered to be a gross wrong to his client. I had formerly known this patient, and at once asked him whether he was still annoyed by having hot air blown up his back at night by women who concealed themselves under his bed. He said "Yes," and that when he got the charge of his property he would bring such legal actions against these conspirators as would effectually punish them for his years of suffering. That was the end of the conference, the lawyer at once saying that he would have nothing further to do with the case, and expressing great surprise that he had not discovered the man's palpable insanity. I was perhaps more surprised within a few months to be called to testify in court as to the man's mental condition, another lawyer having taken up the case. The man evaded questions regarding his delusions on this trial, and while it was perfectly apparent to those who knew him best that he still retained his insane notions, his committee was discharged and he was given the charge of his property.

The delusions of chronic insanity are in some cases most intricate and fantastical, while in others they are simple, fixed insane beliefs. Thus

in every asylum there are some who assert that they are kings or princes or great artists, or even that they are the Deity, and insist upon being treated as such, and consider the restraints which are put upon them as indignities of the meanest sort.

Every person with an insane delusion is so thoroughly occupied with it, at least for the greater part of the time, that he is unable to co-operate with others; and this of course is the reason why so small a number of attendants in an asylum can care for violent and even dangerous lunatics.

In chronic mania there is often considerable self-control—a sort of subconsciousness of error on the part of the patients—which is shown by the fact that they can be better cared for in a well-regulated institution without friction and accident than in private life. The wholesome restraint and the air of discipline which pervades a well-organized institution are often sufficient to prevent anything more than a mere assertion of delusions and empty threats and boasts on the part of those who had been most violent and dangerous at home.

This condition of chronic mania must be distinguished from that form of delusional insanity which has its basis in original mental defect and is called paranoia, chiefly by the history of each case, for paranoia in certain stages exhibits most of the characteristics of chronic mania (see p. 842). It must also be distinguished from that kind of false belief which is held by so many people as the result of misinformation, or that want of intelligence which characterizes many persons who are enthusiastic advocates of some *ism*. This is done by taking into account the history of the case, and by comparing the former sane and sensible condition of the person with his present altered condition, due to some illness, and usually having a fixed point of departure from health and sanity.

Chronic mania is, therefore, not incompatible with comparatively good bodily health, though in a vast majority of cases there is evidence of physical deterioration, a history of wakeful or noisy nights, and an obliteration or change in the lines of expression about the face which indicate insanity. So far as the appearance of the eyes is concerned, upon which much stress is put by laymen, little can be said, for, taken alone, the appearance of the eyes of a maniac have not a diagnostic value, and is rarely relied upon by experts. Of course this does not refer to any pupillary changes, which may indicate more or less intracranial pressure or a condition of paresis.

A man with chronic mania may remain for a long time in a state of mental and physical exaltation with varying delusions, with considerable excitement, with sleeplessness, incoherence, violence, filthy habits, and all those symptoms which characterize the more acute form of mania, except that the lapse of a year has stamped his case with the stigma of chronicity. On the other hand, as before intimated, a single insane delusion may persist alone, and the habits of self-control may in a large measure be restored, so that, excepting upon those topics upon which his delusion bears, he may talk rationally and even intelligently.

It is not, indeed, possible for any person to retain his former poise and judgment, even though the delusion may be confined within narrow limits and be held in abeyance by the patient for the purpose of maintaining his liberty.

I have known a man for fifteen years who had not been able to live with his family, and is maintained by them, who has been continuously insane and has delusions of exaltation—sometimes that he is a great journalist and is a contributor to leading periodicals, sometimes that he is a painter or a sculptor. He will work, accordingly, for months with great industry and zeal, and will exhibit the products of his skill with pleasure and pride, though they are most wretched in appearance. He has at times the delusion that he is a fine musician, and then he bangs the piano for hours, to the distraction of everybody within hearing, and yet all this time he is perfectly well dressed, attends places of amusement, goes to church, takes part in athletic games, and a casual acquaintance would not dream that he was suffering from chronic mania. He is entirely out of touch with the world, has no thought of business, keeps up no associations with his former friends, and manifests little impatience with his method of living. He sleeps well, eats well, and, as his delusions are always of a harmless kind he is allowed his liberty. This condition followed an attack of acute mania in a young, well-bred, well-educated, level-headed man. Few are able to control themselves as well as this man.

The subjoined letter from a chronic lunatic who is at liberty is characteristic:

Mr. ———,

Dear Sir,

The Dorty people are Bawling around yet, night and day I must tell you about—I was out at—followed by——the Bird Voice and Old ——or else the voice. (for the Brat Keeps pace Of me All Of the time.) I was sitting outside of a Hotel. Old——and Birdie Old——got upstairs in the Sitting room. The Proprietor of the Hotel across the Road, her name was——old——hollered down Many a time she lifted her Shirt——, Old Birdie, chirped to it. I told her that many a time she got it lifted, a laugh. I believe Old——lifted her own shirt, with the Bald head a little while after that. Old Birdie——came with her frightened Bird Voice and wanted to meet me out two or 3 miles At the Cemetery. I went. she was giving pitiful Shrieks all the way along trying to frighten——to Marry Her. She was there. ——trying to hold her. and several Other D's after that 7 or 8 mo. she came out playing the H. for a bluff. staying Out wet night, for a bluff.—— ——and her sister went up in the deal. I was coming By Gooble house when her sister came to the windows with the B. pretending it was legitimate another old voice telling me that it was——. he heard of it. and came yelling that it was not his. The Biggest O. Dis—— in the world too numerous at present. bad set. Help to erad and catch them!

Signed ——— ———.

Such persons are liable, however, to commit sudden acts of violence, and the history of many homicides goes to show the fatuity of trusting to the apparently harmless nature of insane delusions. Society is so organized and our lives are so moulded by constant association with sane minds that we come to adjust ourselves automatically according to rules

which in the main govern sane minds. Persons with insane delusions so thoroughly put out of joint the relation of the individual to society that only those who know the form of delusions can foretell the acts of chronic lunatics.

If one were to see for the first time a case of chronic mania in its exacerbation of excitement, with its restlessness, incoherence, and violence, and have no history of the case at all, he might be quite unable to determine whether it was a case of acute mania or one of a chronic character. The history alone would clear up this question of diagnosis. And, again, if a case of paranoia is presented where the delusions happen to be of an exalted nature, accompanied by considerable excitement, it might be quite impossible to distinguish it from chronic mania (see p. 842).

The incoherence of chronic mania differs from that of acute mania in that it rarely has a rhythmical tendency, and that it shows an incoherence of ideas rather than of words. That is, there may be certain sane expressions and a general cohesiveness of sentences, with occasional lapses into incoherence, as the following verbatim report indicates:

"I told you that there were a great many men in the State of New York, both male and female, who do not know what they are living for. Trustworthy American patriots are perfectly well aware what they are living for. They obey the national laws and respect the national government. They were the words you told me. You and me don't agree on that point. The crib club and press gang of the city of Utica come here any time in the day or the night to outrage and brutally assault the inmates of this institution, of which you are well aware. Are you aware that Edison was here. He has been here for four years. I have seen him four years ago in the cellar of the Infirmary. August, 1892, I saw him. He was looking at the men putting up the electric light wire. I heard the men say he was Edison. In the City of Schenectady. He paid a thousand dollars to that man to take the claim of the chief magistrate of the United States and insult every member of the national government. I would not go around the block to tell him what I know about him or any other man. What would we say of a man who was found with a bolt in a place where he didn't belong? At the dark of the night with the bolts, tampering and interfering with the bank. There are nothing to conceal here. He is here for his interest. He says he has interests here. How do the cities refuse to pay him for that portion of his interest? The State have not refused to pay him and he has no business here. He has no business here. He was the man that I heard say. I don't care if he is in the cellar listening to me, and I don't care a damn where he is. If he was standing there before me naked, I would tell him. I have heard that hollering in the cellar of the house. And with overalls on, with hands tied behind their back led to the slaughter-pen. I have heard them cry for their lives. I have heard them in the basement, and I heard them there last night, and I hear them every night. I was not close to them to hear what they said. I have heard them cry for help, I have heard them cry for a great many things. I know it ought to be stopped. I read a statement a while ago about the Governor of this State. He issued an order to the sheriff of

Kings County for to prohibit Mr. Corbett and Mr. Fitzsimmons and Mitchell. The sheriff of Kings County acted upon the order received. The prostitution and desolation that is going on here, there was nothing at all said about it. That was according to law, I suppose. But a friendly contest with four-ounce gloves would be likely to bankrupt the State."

There are certain physical characteristics of chronic mania worthy of mention. Many cases are seen in which a whitlow, or furuncle in the ear, acute pleurisy, or a recently broken bone is found accidentally, no complaint of which had been made by the patient. The same indifference to pain is also seen in acute mania, though perhaps less frequently. The gravest physical disorders are often discovered, which under ordinary circumstances must have been accompanied by great suffering, no intimation of which had ever been given by the patient. No satisfactory explanation has been offered for this phenomenon. The lowered tone of the sensory nerves, the exalted condition of the motor apparatus, together with an all-pervading excitement of mind, must be taken into consideration in explaining this condition. As to broken bones, much has been written tending to show an increase in the lime-salts of these structures, making them more friable in the chronic insane, like those in old age. This, I think, however, has never been substantiated, and is rather put forward as an excuse for those accidents which must necessarily occur to insane people, whether in asylums or out of them. A more plausible explanation, to my mind, is the want of co-ordination of muscles, the natural protectors of bones and joints, so that the muscles, acting in an erratic manner, cause fractures from slight outside violence. However this may be, broken bones are very often found of which no true explanation can be given. In those cases of mania in which there is fixed delusion, having paroxysms of excitement and violence at intervals, and then subsiding into a more quiet, delusional state, the patient frequently makes the most unjust charges of abuse and violence against those who have them in immediate care. This is most natural, and accusations of the foulest kind are constantly being made against their custodians by those who are controlled by delusions. It has been my experience that fewer accidents of a serious nature occur in well-regulated institutions than are found in private practice, where the attendants are less skilled, though actuated by the best of motives. It is impossible for any person to meet the violent assaults of chronic maniacs with perfect self-control, being thereby able to exercise restraining force sufficient for safety, and yet so applied as to do no harm to the patient, unless there has been a long training in this particular kind of work. The hand of affection is not so liable to be applied with discretion as is the skilled touch of the trained attendant.

The establishment of training schools for attendants upon the insane is one of the most marked advances that has been made in recent years in the care of lunatics.

Another characteristic of chronic mania is the power to endure excitement for long periods without sleep. No sane person can feign the maniacal excitement of chronic mania without exhaustion for such a long period as we frequently see lunatics endure. The length of time that a patient goes without sleep, or with but two or three hours of

sleep in the twenty-four, is a very important factor in determining the amount of exhaustion and the amount of physical deterioration that will be found to accompany the period of quiescence which follows. It seems incredible that any human being should go for weeks, and sometimes for months, with almost no sleep at all, and yet such is the case in chronic mania where the paroxysms of excitement are very prolonged.

The question of lucid intervals is one which has formerly been debated with great earnestness in medico-legal cases, though of late years the consensus of opinion among alienists is that in chronic mania with fixed delusions there is never a lucid interval so long as the mania lasts. That a delusion may be successfully concealed for a time, and that it may not possess the same controlling force at one time that it does at another are readily conceded; but the only safe doctrine on this point seems to be that so long as insanity exists the patient must be considered as at no time wholly sane. The chronic maniac, therefore, cannot be said to fully appreciate at any time the nature of his acts or their consequences or the moral aspect of his conduct.

The duration of an attack of chronic mania must depend chiefly upon the age of the individual, the amount of vitality which he possesses at the outset, and the nature and violence of the attack. Many persons live for years with but little apparent deterioration in strength, especially if the chronic delusional condition is not accompanied by frequent and serious exacerbations of excitement and violence. The average duration of life, however, is very much shorter than that of a person not mentally affected. Gradually there come on more enfeeblement of mind, a lessening of the physical forces, and a sinking into that terminal dementia which soon ends life.

On the other hand, the accidents which occur as the direct result of a maniacal condition are very numerous. Of first importance among such accidents or intervening conditions must be placed apoplexy and the various forms of meningeal inflammations, and next the advent of some acute disease, so much more likely to come on and destroy life because of the lowered nervous vitality of the patient. Thus, all forms of lung disease, both acute and chronic, are very common, though, strange as it may seem, taking into account the perverted secretions generally seen in all forms of mania, fatal disease of the liver and kidney is apparently infrequent. The vascular system is at fault in a large number of cases, and possibly this is due to the fact that some weakness or disorder of the bloodvessels is most likely to precede and often to cause insanity.

Persons have been known to recover after long periods of chronic mania: though these cases are very rare, it cannot be said that the patient may not recover even after several years of sickness.

These great divisions of mania already described ought really to include all forms of the disorder where the word "mania" is attached to the name. Still, many authors continue to describe special forms of mania, adopting of course a nosology based upon the clinical features of the case. It is easy to see that no two cases of insanity can be exactly alike. No two cases of mania exhibit all the same symptoms, and yet the assemblage of all the symptoms and the objective signs of the disease

will usually establish the type of disorder, so that a simple classification can be made.

Monomania.—The term monomania is not properly a mania at all, because it is used to designate a state of mental depression having a fixed delusion, which is classified as a melancholia. The term in its strictest sense implies a single delusion, and is sometimes used to denote, not insanity at all, but a species of dogged devotion to one idea which may not be a delusion.

Pyromania has been described alone, but is really a subacute or chronic form of disease, with a delusion which gives a constant tendency to set fires. This can in no way be called a separate type of disease, some sensory disturbance or hallucination ordinarily giving direction to the disease.

Kleptomania is the term ordinarily applied to persons who are in good social position and who are found to be inclined to pilfer small and unimportant trifles. I have studied several such cases occurring in young people, and have found them mentally defective in other ways, so that I should suspect a case of so-called kleptomania in a very young person to be one of paranoia. There is nothing about any of these cases that suggests the name mania. In older persons, where the tendency to pilfering is discovered, I have always found it due to a rather loose conception of morality, accompanied by a desire for an unusual excitement: other stimulants to produce a new sensation having been tried, this immorality was enjoyed until after its discovery. I do not think, therefore, that it can possibly come under the general description of mania whenever found, unless the individual is acting under some well-defined delusion, and then the mania can be easily classified.

Homicidal mania was a term that came into use when moral insanity and morbid impulse were discussed as entities, apart from their association with some well-defined insanity. The term would probably never have been brought into use by medical men but for the medico-legal aspects of certain cases that could thus be conveniently designated.

When a man has a delusion that he is persecuted or that he is about to be killed, or when he has an hallucination of hearing, and the voice directs him to slay his neighbor, or when the paroxysms of maniacal excitement and turbulent restlessness impel him to seize and throttle an attendant, in none of these cases can the man be said to suffer from a distinctive form of insanity which is beyond the simple classification mentioned. It is not worth while to multiply terms. The fact is, that in chronic delusional insanity or chronic mania, as it is called, if a name must be given to each separate case acted upon by an extraordinary delusion, we should have almost as many subdivisions of mania as there are patients.

Surgical mania has been described as a special form of disorder, though it in no way differs from an ordinary acute mania, and it usually runs a course like acute mania.

Puerperal mania has also been described by itself, but it is commonly a subacute mania, which has perhaps more frequent hallucinations of sight and hearing than is seen in the ordinary run of this disorder. More rarely it is of an acute type, but in any case it may be classified

under the forms described, and its course and termination are very much like the disorder arising from any other cause.

Folie Circulaire.—There is one form of insanity which is as much a mania as it is a melancholia—that condition of alternation of exaltation with depression for longer or shorter periods which forms a distinctive type. The classification adopted in New York at least, and in this country generally, includes alternating mania as a distinctive form of disease. This has also been described for many years as folie circulaire. While this is not strictly one of the manias, it ought to be mentioned in this connection to distinguish it from the common manias. The onslaught is about the same as that of subacute mania, being preceded, for a few days or even weeks, by a feeling of general indisposition, some slight physical depression, such as would accompany dyspepsia, a partial loss of sleep, and a general depression which is succeeded by a mental exaltation, physical excitement, bodily restlessness, loquaciousness, irritability of temper, etc., which ordinarily characterize the onslaught of acute mania. The attack runs for two or three months, sometimes longer, like a subacute mania—sometimes disappears in one night, the patient awakening without any expansive ideas and apparently sane and perfectly natural.

The sudden termination of the period of exaltation is the one thing that is most strongly diagnostic of a relapse. The period of quiescence and of apparent recovery is followed in a few days by great distress of mind, depression of spirits, and all the characteristics of acute melancholia. During this interval the patient bemoans his fate, begs forgiveness for his indiscretions, says he alone is accountable for the distress of himself and his family—that he has ruined his business, and that he has sinned against God, and often attempts suicide early in the attack. After two or three months, or even longer, of this mental depression, he suddenly rebounds, often in a night, awakening some morning to find himself again cheerful, happy, complacent, and in a natural mental state. This is succeeded in a few days by another period of mild maniacal excitement, completing a circle of alternating mania and melancholia often within six or eight weeks. The initiative, I think, is more frequently a state of depression which follows some business loss, some bereavement, some prolonged condition of bodily ill-health, or fatigue from too close application to business.

It is sufficient thus to point the main characteristics of this disorder, which might be mistaken for a simple mania, and which can only be diagnosed as alternating by the history.

Complications and Sequelæ of Mania.—Mention has already been made of certain complications that may arise, the most notable of which are due to the weakened condition of the vascular system, and which give rise to sudden apoplexies more or less serious. The simple hemorrhagic infarctions which frequently occur, especially in chronic mania, may produce suddenly an unconscious stertor and collapse threatening immediate death. If a reaction sets in within a day or two, however, and the temperature does not rise more than one or two degrees, the patient may recover. Such a hemorrhage is not so likely to occur in the motor centres.

There is less paralysis following than is usually seen when such

symptoms occur in persons previously sane. There is a subsidence of the acute maniacal symptoms after such an accident, and not infrequently the case takes a more favorable turn after it.

A marked instance of this sort occurred in a man of forty-five, who during an attack of subacute mania, with varying delusions, great restlessness, and an exaltation of all the bodily powers, suddenly fell, apparently apoplectic, with a suffused face, almost imperceptible pulse, labored breathing, and all the symptoms that denoted approaching dissolution. His friends were sent for, and after watching at his bedside for two or three days he gradually improved, and, though he was unconscious nearly a week, he recovered from his insanity without any paralysis. Much more frequently the apoplectic condition terminates in death, very rarely in hemiplegia or paraplegia.

An acute infectious disease, like typhoid fever, dysentery, or erysipelas, coming on in a case of acute mania is likely to prove fatal; but if any of these conditions occur in chronic mania, and the patient has a fair amount of strength so as to weather the disease, his sanity may be restored.

I have witnessed one epidemic of dysentery among the insane that seemed to be a means of restoring a number of chronic maniacs to health. The acute lung disease which appears during the course of any form of mania can often be detected only by the physical signs, the rational symptoms being entirely absent.

These disorders do not seem to produce the same favorable results upon chronic mania that the acute infectious disorders have in some cases. Tuberculosis is very common as an accompaniment of chronic insanity, and causes death in a large number of cases.

Diagnosis of Mania.—It is not difficult to make a correct diagnosis when brought to a typical case of insanity having a definite history. It is quite another thing, however, to distinguish some of the milder forms of mania from exaggerated eccentricity, from alcoholic poisoning, from delirium due to toxic conditions, and from simulating or feigning by those who desire to avoid the consequence of criminal acts. In most cases the important thing to consider is the minute history of the patient's life, his ordinary methods of doing business, his relation to his family and to the outside world, as well as his hereditary tendencies and his physical ailments.

In simple mania and in subacute mania the judgment must be made up almost entirely from such history. Expertness in this field is shown as much in the painstaking method by which the history is elicited as it is in the ability to form a correct judgment upon a group of isolated facts. There are certain peculiarities in the manner of every man with an unbalanced mind that are hard to describe, but impress the examiner very forcibly. Chief among these, perhaps, we may speak of the unnatural alertness and readiness to volunteer unnecessary information, a quickness to respond to what seems to him the slightest intimation of mental unsoundness, and an insistance in arguing the case with tireless energy.

Next comes the tendency to gather up and dilate upon unimportant trifles, and in doing so to lose sight of the significance of overt acts or of extraordinary conduct which have chiefly impressed sane minds about

him. The assertion that he feels better than usual, that he is in excellent health, and that nothing is the matter with him, made with an air of exalted complacence, is characteristic of the simpler forms of mania.

In the absence of positive delusion, which alone can determine the case definitely without the aid of other symptoms, we must seek for that period in life when the decided departure from health was observed, accompanied by a marked change in the patient's manner and method of talking. This can usually be done if the search is made intelligently; and it is absolutely necessary to do this, and to compare the former sane condition with this altered mental state, in order to make a positive diagnosis.

Then, too, certain toxic agents produce a delirium which is hard to distinguish from some forms of mania. Alcoholism is more frequently mistaken for mania than any other state. Here, again, the history is of prime importance, and the want of symmetry of all the symptoms which go to make up a case of mania must be observed. Thus, the delusions, hallucinations, and illusions are apt to be seen in a case of alcoholism without the accompanying excitement, making up a picture which is not easily harmonized with any one form of mania, being rather an aggregation of symptoms of various forms of insanity combined in one. Great depression of spirits like melancholia, with profuse weeping unlike melancholia, alternating with periods of exaltations within the same day, accompanied by delusions and hallucinations, with a thickness of speech peculiar to paresis, may be seen in alcoholism, but not in any form of mania.

The various forms of delirium may usually be distinguished by the excessive rise of temperature due to well-defined bodily disease accompanying it. The temperature is not raised, at least to any perceptible degree, in the milder forms of mania, and it is only in acute and violent manias that the temperature is raised one or two degrees. Such a patient would not be easily kept in bed or even managed without physical restraint, while a delirium that would simulate insanity at all would have a much higher temperature even though the patient could be safely cared for in bed.

"Delirium grave" is a sickness which ought not properly to be included among the manias, and yet it starts like a sudden and violent attack of mania, usually in a person of enfeebled health. Its course is short, the patient rarely surviving more than a week, and the temperature is high from the first, and gradually increases with a feeble and rapid pulse until death supervenes. This cannot be distinguished from the most acute mania, except perhaps by the temperature, and also by its rapid progress and speedy termination. The loss of flesh, the inability to take food, constant restlessness and violence, and the loss of sleep make it one of the most serious of all the ailments to manage. It ought to be classified, however, as an acute meningo-cephalitis rather than as a mania.

Acute mania is rarely simulated by criminals, though I have seen a few cases in which this was attempted. It is impossible for a sane person to maintain even a mild semblance to acute mania for a long enough period to escape detection if watched fairly well. Great phys-

ical fatigue comes on so soon that it is impossible to maintain the guard with any degree of skill. It is the only mania with delusions that can be simulated with any degree of success.

In studying a case of suspected feigning, where the basis of the alleged insanity is a delusion, the real mania is distinguished from the spurious chiefly by the constancy with which it is maintained and the unfailing control it has of the entire individual. To distinguish between the real and the assumed delusional state is a most difficult task, and often is the most tedious one an expert has to undertake. It is pretty safe to suspect that a person is feigning who, after having been caught red-handed in crime, asserts a delusion—such as that he was commanded by God to do it, or that he heard voices telling him to do it, or that his victim was chief conspirator of an intricate plot to ruin him—unless it can be shown that some intimation of this belief had been given to his friends before the act. The absence of any real motive for the crime, on the other hand, is strong presumption that the person was not acting like a sane man. This fact alone, however, cannot possibly establish a case of insanity, though as a negative proof it is worth considering.

The statement often made to me by criminals that they remembered nothing from a point of time just preceding the crime to a point of time just after the act is usually a false one—always so, I think, except in a case of epilepsy or drunkenness. This, of course, does not include those cases of maniacal violence where some person is accidentally injured. The personal habits of the patient afford the strongest indications of his condition of mind. If it can be clearly shown that these have remained unchanged—that, in the main, he dresses, talks, and in general behaves in a manner that is characteristic of him when well—his assertions concerning delusions and hallucinations may be taken with extreme caution. On the other hand, if there has been a marked change in the dress and the general demeanor of the patient, his habits, his talkativeness, his social instincts, his attention to the necessities of life, a delusion may be strongly suspected, though concealed for a long time.

Insane people rarely, if ever, admit that they are insane, and they justify their acts with an earnestness which is not easily mistaken. It is not necessary in order to establish a diagnosis that the delusion should be present or that the person should be irrational on all occasions, as is commonly supposed to be the case by the average jury. It is necessary, however, in order to establish a diagnosis that a marked change must be shown in the man's manner of thinking, feeling, and acting.

To distinguish mania from paranoia one must have a complete history, which in the latter disease shows the victim to have been degenerate from birth, and that the departure from health was so gradual as to hardly distinguish it from the onslaught of disease in a person previously sound and well balanced.

General paresis may in its early stages be mistaken for mania, but in paresis there is not the physical excitation so essential to mania, and the speech, instead of being tumultuous, is often slow, hesitating, and thick, due to the paralysis. There is also a peculiar tremulousness of the tongue, and an inability to protrude and hold it still, and a similar tremulousness of the muscles of the arm, which is detected by grasping

the fingers of the firmly extended hand and arm. The gait of the paretic will be noticed early.

Prognosis of Mania.—In general, it may be said that the prognosis in the more acute forms of mania is favorable, both as to life and as to recovery from the mental symptoms. As regards chronic mania the prognosis is most unfavorable in every respect. It cannot be ascertained from statistics taken from asylum records what proportion of simple manias recover, because so few of these cases are taken to an asylum, or if they ultimately appear on the records of the institution, it is after they have become acutely maniacal, or after such time has elapsed that they are classified among the chronic insane.

Physical restraint is not so necessary in simple mania in order to protect the community against the violence of a patient, and the friends will endure any amount of annoyance and mortification, and often loss, before they will institute legal proceedings to commit such persons to an asylum. It is not probable that so large a proportion of cases of simple mania recover as of acute mania—perhaps not more than 50 per cent.; but when the recovery does occur, relapses are not so common as in other forms of mania. Death rarely supervenes unless the case progresses into other and more serious types of insanity.

A larger proportion of recoveries would take place if all cases of simple mania that could not be easily controlled at home were removed to an asylum. The prejudice which is deep-rooted in every community against depriving any person of his liberty on the ground of insanity, unless he is violent and dangerous to himself or others, is not real conservatism. Simple mania, early restrained and properly cared for, would show a very large percentage of recoveries.

The prognosis in each individual case must depend upon the period of its duration, as well as upon the causes which operated to produce the attack. If a case of simple mania has existed for several years, it is about as hopeless as chronic mania. The facts which tell in favor of recovery are a fair condition of general health, an environment which prevents exciting causes, and a steady persistency in treatment permitted by the friends until all the symptoms disappear. The removal of cases of simple mania from asylums, sanitariums, or hospitals, or from strict discipline at home, before a complete restoration has taken place, often precipitates an attack, and even develops acute mania out of this simple type.

Prognosis, therefore, rests somewhat upon the general intelligence and courage of the friends as well as upon the individual afflicted. In general, recovery will not be so speedy as that which occurs in acute mania in the most favorable cases. From four to six months must elapse before the disease can run its course under the most favorable circumstances.

The prognosis in acute mania for all cases that come under treatment early is very good indeed. The recoveries are variously estimated at from 60 to 70 per cent. It is more favorable when the hereditary taint is slight and when the condition comes suddenly upon a young person, who from general ill-health and great mental strain is precipitated into an attack. Great age, strong hereditary taint, and organic disease of the heart or bloodvessels make a prognosis in any case most serious. As the

case goes on the prognosis varies with the varying conditions of physical strength. It is more favorable if the mental symptoms keep pace with the physical changes. Unless the patient improves in general health as his mental symptoms begin to disappear, the chances are in favor of a condition of exhaustion. If the physical health is re-established and the mental symptoms have not proportionately improved, there is danger of chronic insanity.

The death rate of acute mania varies greatly according to the age of the individual and the condition of his vascular system. Not more than 7 to 10 per cent. die during the acute attack, unless there be great age or some serious bodily ailment. There will, therefore, be left something like one case in three that passes into a chronic state. Relapses after recoveries from acute mania occur in perhaps 20 per cent. of all cases even after they have remained well for a period of years. As has been stated before, the prognosis of absolute recovery must be guarded, and, while comparative recoveries are numerous, yet few persons reach exactly the same place that they occupied before the mania came on. There is no fixed standard for absolute sanity by which every person must be measured, and, while many people recover from acute mania, all such persons are liable to show eccentricities and weaknesses or peculiar ideas not constituting insanity, yet not natural to the former state of the individual.

The prognosis of subacute mania is not quite so good as of acute mania. Fewer die during the early stages of the disorder, yet probably a larger proportion terminate in chronic mania. Recoveries from chronic mania are comparatively rare, though such recoveries do undoubtedly occur even after the patient has been insane for a number of years. The disease, therefore, cannot be said to be incurable, though the very term "chronic" indicates hopelessness regarding the ultimate restoration.

The prognosis as to the length of life is much less favorable than as if no insanity was present; for, aside from the accidents and complications peculiar to the disease, the bodily powers are sooner exhausted and the physical evidences of old age appear sooner.

Recurrent mania must always be regarded in the most serious light, especially after two or more cycles have been completed. Even if there should be a period of comparative quiet and apparent sanity lasting for weeks, the instability and uncertainty of such condition must be borne in mind and a very guarded prognosis given.

TREATMENT OF MANIA.—The preventive treatment of mania must largely rest with the general practitioner, and for this reason a more thorough appreciation of the factors which enter into the causation of the disease ought to be kept in mind than is generally the case. The teaching in our medical colleges of this branch of medicine, which has within a few years been included in the regular curriculum, must undoubtedly bring about good results of the greatest importance in the future.

As has already been intimated in discussing the question of causation, much can be done toward the prevention of insanity by judicious advice regarding the up-bringing of so-called nervous children. Precocious children should never be urged to develop that lowest of all the intellectual faculties, the memory, to the neglect of the higher faculties.

Such children should in early life be indulged in out-of-door sports, and especially should they be encouraged to play with animal pets, and thus to learn humanity, self-control, the appreciation of the feelings of others, and the exercise of consideration and pity for suffering, which alone can counteract the dangerous selfishness and egotism that make them in later years an easy prey to the passions.

The somewhat antiquated method of teaching obedience, reverence for their elders, and respect for authority and sacred things made stronger characters than we see developed now-a-days in children who are indulged in every direction.

Puritan New England of a hundred, or even fifty, years ago had fewer cases of insanity or of nervous diseases generally than may be found in the New England of to-day. The industry, self-denial, and patience necessary to the acquirement of an education in former years developed men with more poise, strength, and self-control than the kindergarten system of to-day can hope to do when supplemented by private tutors, luxurious college life, and followed by an easy entrance into the professions, which great wealth may provide.

Any tendency in the public-school system to make the teachers do the work, and to give the children to understand that tasks which are not pleasant are unnecessary, and that the acquirement of real knowledge must always be made a pleasure instead of a hardship, will not bring out the strongest and best elements of character, which must stand against the shocks, the accidents, the disappointments, and the anxieties of life.

The ambition of parents of neurotic tendencies should not carry them so far as to insist that their children shall become highly educated in the common acceptance of the term. In general, good manners persistently taught in youth exercise a wholesome control over the lives of men.

It is very doubtful whether teaching children the evils of vices, the dangers of personal indulgence in questionable habits, especially as regards social vices, acts as a deterrent force against unbridled license in later years. Wholesome occupation of body and mind, with careful avoidance of unnatural and prolonged emotional conditions, must necessarily prevent persons of unstable inheritance from crossing the line of demarcation between sanity and insanity. On the other hand, the too intense pursuit of exciting occupations without occasional mental relaxation frequently leads to insanity.

It is a curious thing that misers rarely become insane and usually are long-lived. The same may be said of other men who pursue rationally and earnestly any legitimate object in life.

Good living is certainly one of the strongest preventive measures against the onslaught of mania. The hard workers and hard thinkers, as well as hard fighters, are usually good livers. This may be made to cover a moderate use of alcohol, though never its abuse.

In rural communities mania most frequently develops after the summer season of hard work among farmers who have labored for long hours, and who have not had an adequate food supply or who have lived upon improperly cooked foods. Melancholia, on the other hand, is more likely to occur in the winter and spring months. The poverty

of diet and the ambition to accomplish much work during the time when the crops must be gathered brings about an impoverishment of the blood which predisposes an attack of mania.

General ill-health, therefore, is often all that can be discovered as the immediate cause of an attack of mania. One may sum up the subject by saying that "moderation in all things" tends to sanity, as well as to longevity.

Excesses in alcohol, venery, or gluttony tend to produce general ill-health, as much as long hours of hard work and an insufficient diet.

When an attack has once thoroughly established itself the treatment of all forms of mania must be chiefly hygienic and dietetic, not medicinal. In milder cases, and especially in simple mania, if the environment of the patient is bad, a radical change of scene, if it can be accomplished, with a fair amount of mental occupation, is most advantageous. On the other hand, it is folly to suppose that change of scene can, of itself, effect much in a case thoroughly established. A man cannot be amused unless he can reason, and if he has a delusion, change of scene will not make him forget it.

We must then consider chiefly all those means that tend to increase the bodily strength, to promote sleep, and to secure quiet both of body and mind. As the absence of sleep is one of the earliest and most prominent symptoms, so it must be the chief thing to consider in the question of treatment. It is well known that a diet composed largely of milk, together with free exercise in the open air to the point of muscular fatigue, tends to produce sleep, and that the avoidance of exciting discussions and of stimulating foods and drinks is also important. While the patient should, therefore, be fed freely and generously, the diet should not be chiefly of meats. Coffee should be excluded, and stimulants, except in debilitated cases, should be withheld or limited to the meal-time.

For the promotion of sleep many drugs have been recommended, but very few can be used with uniform success in all cases. In full-blooded persons with simple mania, where the appetite and digestion remain good, the bromides may be given with great benefit in doses varying from thirty to sixty grains, three or four times a day, to allay the muscular restlessness. The hot general bath or the hot spinal sponging at night is of great service. A single dose of chloral of from fifteen to twenty-five grains, well diluted in water, may be given at bedtime. If this is not sufficient to secure sleep, it is better to give some hot broth, hot milk, or hot gruel than to repeat at once the chloral.

Newer hypnotics are much better for simple insomnia. These are sulphonal, trional, and tetronal. They are much alike in their action, but none of them are very efficient when there is pain, great bodily restlessness, or intense maniacal frenzy. Sulphonal, which has been longest in use, is best administered in hot milk or hot broth in doses of from fifteen to thirty grains, about two hours before bedtime. Its action is so slow that it is sometimes better to give it three or four hours before sleep is expected. When sleep is secured in this manner it is profound and usually prolonged and refreshing. If the dose has to be repeated, an interval of at least two hours should intervene, and if two

full doses do not secure the result desired, it is not worth while to push the drug.

There are some objections to this hypnotic: First, its influence is indefinitely prolonged, so that the symptoms are obscured during the next day and sometimes for forty-eight hours, and there are some cases in which vertigo seems to be produced by it. One advantage is that a single full dose will sometimes secure sleep for two nights, its influence the second night seeming to be fully as pronounced as that of the first.

Trional seems to be the better drug of the two, but it has the same difficulties and is administered in the same manner, because it is so hard to dissolve. In those cases of simple mania in which this drug acts favorably there is less objection to its administration than to that of almost any other medicine used for the purpose of procuring sleep. It does not seem to be followed by vertigo or by any objectionable symptoms the next day. Tetronal is very much like it—not in any way better—and of these three hypnotics trional seems to be the best.

Experiments have been made with these newer remedies regarding their influence upon maniacal excitement when given during the daytime, and the conclusion reached has been that tetronal is a better drug than either of the others of this class when given two or three times during the day to prevent excessive restlessness. The dose in this case is ten grains, and may be repeated at intervals of eight hours. In perhaps one quarter of the cases in which it has been used it has exerted a marked influence over the maniacal excitement.

If sleep is not obtained by any of these methods, then hyoscyamine may be administered hypodermically—one tenth-grain doses—in the latter part of the day, and one fourth of a grain of bromide of morphine may be given hypodermically at bedtime, and this may be followed by a full dose of bromide of sodium. When these drugs fail the simpler remedies—valerian, fluid extracts of hops and of skull-cap—may be given during the day and evening with benefit.

The securing of sleep by any drug is one of the most difficult features of the treatment. Wherever it is possible, prolonged exercise in the open air during the daytime is much more effectual in procuring sleep than any other means. This will depend very largely upon whether the case is to go to an asylum, or whether the patient has sufficient money to pay the very large expense of proper care at home. This question of money determines more than any other factor whether the patient shall be restrained in a hospital or whether he be given greater freedom and a better chance of recovery by having all the paraphernalia of a hospital at his own home. A small private hospital answers the purpose, but that is also so expensive as to be beyond the reach of the average person.

If a maniacal patient has been sleepless for a week and cannot be controlled by moral suasion, then it is always best that he should go to an asylum, unless he has sufficient money to meet the great expense of nurses, and unless also there is a skilful physician at hand.

With regard to the treatment of cases within the asylums this discussion has nothing to do, for it is intended for the general practitioner. If a case is to remain at home, at least three attendants are necessary, with house-room enough, so that several rooms may be set apart for the sick

man. All breakable articles having been removed from these rooms, one attendant must always be on duty, with a second one at hand to be called in an emergency, while the third sleeps. The patient must never be left alone. He must be talked to as if he were sane, for there are gleams of reason in most cases of insanity. He must never be lied to or cajoled, tricked, or humored to his disadvantage, but must always be told the exact truth in plain terms, both as to what ails him and as to what it is necessary for him to do. It is better not to restrain him, excepting so far as it is necessary to prevent violence to himself and others. He ought to be out of doors as much as possible, and therefore in as secluded a neighborhood as possible, so as to be allowed a fair amount of exercise on foot. It is better to have the patient, unless he is boisterous and noisy, driven by his attendant in a carriage, for the motion of the carriage in the open air does not exhaust the strength, and is very efficacious in producing sleep. Thus, especially in the milder cases, if a small farm-house is secured, where comfortable board can be procured, and the patient is driven many hours a day in the open air, the very best results may be expected.

I have witnessed the recovery from acute mania of more than a year's duration brought about by this method of driving for many hours each day in the open air, which produced sleep and a wholesome appetite when drugs and restraint had failed.

The difficult part of all this is to secure proper attendants. They must be thoroughly trained to this work, must be strong and even-tempered, and must be paid enough to secure their best energies.

As before intimated, foul breath and a sluggish condition of the secretions must not be taken as an evidence of indigestion, and therefore requiring a limited diet. The diet should be full and generous, consisting of meats during the height of the attack in moderation, milk in great abundance, as well as eggs, fruits, and vegetables. Alcoholic stimulants during the height of the attack do not seem to increase the excitement, but rather to lessen it decidedly. Anæmic or feeble patients may be given ale or whiskey and milk with freedom, whichever is best borne by them.

The manifestations of erotic excitement are best controlled by the bromide salts, and especially the bromide of camphor. This may be given in three- or five-grain doses three times a day, and will usually be found efficient.

Where syphillis is known to exist the regulation treatment of this disorder should be begun at once, and if it is an old case, the iodide of potassium should not be relied upon alone. Frequently small doses of bichloride of mercury or iodide of mercury are more efficient and stimulating to the absorption of syphilitic exudate than is iodide of potash alone.

Likewise rheumatism, gout, bronchitis, asthma, or any other well-defined disorder must be treated upon rational principles, the same as if insanity was not present. In alcoholic cases it has been found that the use of strychnine, preferably given hypodermically, aids very much as soon as convalescence begins. These cases should also be detained in asylums longer than those whose attacks have been caused by some sudden condition of ill health. It is better to withdraw the drug which

has produced the delirium, no matter what particular drug forms the habit. The exhaustion following such course is often very great, but the judicious administration of cardiac stimulants, especially of strychnine, renders it safe to do this, and the cases run a better course when this treatment is instituted at first.

When the period of convalescence sets in and the reaction occurs after the violence, stimulating tonics are of great service. These may be of the various preparations of phosphites or hypophosphites, with iron, quinine, and strychnine. This latter remedy is of great service during the period of convalescence, when the circulation becomes rather depressed, the extremities cold, the skin greasy and turgid, with the finger-tips, lips, and ears of a purplish tint, showing venous stasis. In this stage the use of hot baths, massage, and faradism to stimulate the vasomotor system is helpful.

This is a general outline of treatment for the cases of simple mania, and even for those milder types of acute mania and subacute mania that can be managed outside of an asylum.

The persistent refusal of food, requiring artificial feeding, usually makes it imperative to place the patient in a hospital, mainly for the reason that he needs more frequent attention on the part of the physician than can be secured outside. When there is absolute refusal of food the stomach-tube is passed and a pint of hot milk, with an ounce of whiskey and two or three ounces of the expressed juice of meat, may be administered three times in the twenty-four hours. Very frequently, when there has been a refusal of food for several days, vomiting comes on, and it seems impossible for the patient to retain anything. These cases are best managed by the stomach-tube, washing out the stomach, if necessary—first with warm water, then adding one pint, or even two pints, of predigested milk, together with an ounce or two of rye whiskey. This is often found to remain when smaller quantities of food would be promptly rejected.

The examination of the urine will frequently disclose the fact that the amount excreted in twenty-four hours is below the standard of health. At the same time, the specific gravity may not be proportionately high, to indicate that there is an elimination of the solids which ought to take place. If six hundred grains of solid matter are taken as the average for a person in health when no unusual physical exercise is indulged in, then it is apparent that a much larger amount than this ought to be excreted by a person under great physical excitement. It is more important to test for the amount of solids eliminated by the kidneys in the twenty-four hours than to consider any other indication which the urine may afford, excepting, of course, grave kidney disease.

A simple method of arriving at a fairly correct conclusion is to take the whole number of ounces of urine for twenty-four hours, and multiply this by the last two figures given by reading the average specific gravity of the urine. This product is in grains, and ought never to fall below five hundred. It will be found necessary often to administer diluents, of which perhaps the best is distilled water or some alkaline carbonated table water, in order to secure the elimination of detritus produced by rapid waste of tissues during the attack. The condition of the skin itself is also very important, both as an eliminative

organ and also as to its irritability, which unnatural dryness and an unhealthy condition generally may produce. Tepid baths and oil rubs are often of great service. No very brilliant therapeutical results have followed the excessive use of hydro-therapeutics, which have been so frequently tried. In general, therapeutic treatment must be combined with the dietetic management, so as to maintain as far as possible the bodily strength during the height of the seizure, to restore the blood to its natural condition, and to repair the wreckage that has been done when convalescence sets in.

Simple mania may frequently, at its outset, be modified by the administration of such sedatives as the bromides, valerian, or even morphine during the day and trional and chloral at night, so that the attack may be cut short or very much modified in its violence.

The same may be said, though to a less degree, of subacute mania, but the abortive treatment of acute mania is not so successful, and indeed is rarely seen.

There is no doubt but that simple mania, coming on suddenly as the result of great emotional excitement or great mental strain in persons who have unstable nervous organizations, may be aborted by the persistent and careful use of morphine given hypodermically, so as to ablate sensation and to cause sensory impressions to disappear. The cases most likely to be benefited are those where the mania comes on in a young person, developing out of an hysterical state perhaps, and which has been preceded by some great mental shock. I have seen many such cases recover after two or three days of half-consciousness produced by this drug; but in every instance the treatment was begun at the very outset of the disorder. I have not seen this use of narcotics succeed in any case that has had a history of even two weeks' duration.

Chronic mania ought not to be treated in a private house, unless the delusions are fixed, well defined, and of a harmless nature. Even then the environment of the patient ought to be such that no unusual emotions should be excited, for it must always be borne in mind that a diseased brain will never act in exactly the same way that a sound brain will in an emergency. A delusional state, with varying hallucinations and illusions, is too dangerous a condition to permit the patient to go at large. Chronic mania ought, therefore, as a rule, to have the restraint of a well-regulated asylum, where the treatment is disciplinary as well as dietetic and hygienic.

DEMENTIA.

By WILLIAM BROADDUS PRITCHARD, M. D.

DEFINITION.—The term dementia in its broad etymological significance means, literally, "without mind." To the layman it has long been a generic term for insanity. Technically, the word is restricted in application to a condition of mental incapacity, varying from slight to profound degree, which represents a quantitative impairment in contrast with the simple perversion or qualitative deterioration in mental functions in other forms of insanity. There is much vagueness and confusion, however, even among alienists in the clinical application and significance of the term. It has been a sort of waste-basket into which types not clearly understood have been thrown more or less arbitrarily.

CLASSIFICATION.—Dementia may be either a congenital or an acquired condition, although the word amentia is more correctly descriptive of the states of mind-absence met with in congenital cases. Amentia represents a primary defect of organization; dementia, on the other hand, reflects a state of disorganization. With the former we have nothing to do in this paper, the subject being covered in the articles upon idiocy, imbecility, etc., appearing elsewhere (p. 859).

Any basis of classification of practical value in obtaining a working clinical conception of acquired dementia must, of necessity, be more or less arbitrary. We have pathologically two groups or types—one in which certain causes produce certain constant organic changes in the tissues of the brain; the other group includes cases lacking a constant or fixed organic basis, though in many of them determinable but inconstant lesions exist, either primarily or secondarily. It is quite probable that further advances in methods of investigation will justify the popular impression that structural changes exist in all cases, but we have not yet reached that point. We have therefore two broad groups in dementia—the vesanic or functional and the organic. Of the so-called functional dementias some exist as such primarily, while others are secondary or sequential to other psychoses. Clinically, the fundamental affection of mind is the same in all cases, and yet the symptom-picture varies considerably in the degree of quantitative impairment, with correlated variations in the prognosis. These facts constitute the basis for the classification herewith submitted:

VESANIC:

1. *Primary,*
   - Simple (including partial) dementia.
   - Hebephrenia (insanity of pubescence).
   - Acute primary dementia or stupor.
2. *Secondary,*
   - Secondary confusional insanity.
   - Terminal dementia of mania, melancholia.

Organic:
1. Paretic dementia.
2. Dementia due to gross brain disease, including traumatic, post-hemiplegic, meningitic, etc.
3. Dementia due to syphilis, myxœdema, etc.
4. Toxic, including post-febrile, alcoholic, etc.
5. Senile dementia.

Simple Dementia.

Definition and Symptoms.—Among the familiar types common to village or town life in almost every community is the harmless dement. Negligent and dirty in dress and appearance, foolish and silly in manner, taciturn or garrulous in speech, generally peaceable and easy-going in disposition, though sometimes irritable and morose, the butt of the small boys' practical jokes, he lives for years perhaps the same routine of useless existence. These cases are often classical symptom-types of simple dementia. Some of them represent terminal stationary stages of acute insanities, others are post-hemiplegic or traumatic in origin, while others still are cases of congenital defect or imbecility. A certain proportion of these dements, however, belong to a class *sui generis*, and represent a condition of gradually acquired intellectual and moral enfeeblement, beginning insidiously (rarely abruptly, and even then without active symptoms of either maniacal, melancholic, or stuporous character) and without demonstrable direct cause. Often in such cases there may be a history of ordinary or even unusual intelligence in early life, but with apparently a lack of stamina, resulting in early exhaustion of intellectual function. In other cases, probably the majority, there is a history of backwardness or slight stupidity in early years, not particularly noticeable, however, until some shock or fright or other active cause precipitates a total mental collapse. The explanation suggested in many of these cases for the mental phenomena present is that of precocious senility in the psychic sphere, the physical man remaining normal or nearly so. It is, so to speak, a juvenile form of senile dementia, with absence of physical senility. The period of second childhood has anticipated by several years the normal involution of the individual. The principle involved in the theory of the "extinction of the unfit" is strikingly suggested in this class. Often such patients represent etiologically a most intense degenerative heredity in consanguineous marriage or vicious excesses in the parents, particularly alcoholism. These patients are of little practical interest to the physician except from a sociological or medico-legal standpoint. The determination of the legal status of such cases is ordinarily simple, but in rare instances fixing the degree of responsibility where a crime has been committed is not so easy. The prevalent impression that these dements are harmless is sometimes very unexpectedly and unpleasantly refuted by an act of violence with or without provocation. Certain of these patients may at times suddenly and without warning develop furiously maniacal attacks, with purposeless tendencies most violent and destructive. The safety of the patient and of the community is positively assured only when such individuals are under restraint or control, preferably in an asylum. In many of these cases such care and control become necessary, although no violent

tendencies or destructive propensities exist. The dementia itself may be or become so profound and complete as to render the victim absolutely helpless. Such patients have to be clothed and fed and protected from the weather, and all their natural wants attended to, being just as dependent as infants.

So-called partial dementia differs from the type just described only in the lessened degree of impairment of mind which is present. In some cases the enfeeblement of memory, judgment, and attention is comparatively slight, the individual retaining intelligence sufficient to enable him to care for himself, and even in some instances to provide for a wife and family. Such individuals are able to follow some simple employment at manual labor and to earn a scant livelihood, though the struggle for existence is almost upon the plane of purely animal competition. The moral sense is defective, and there is total absence of all higher ethical principles. Many of these partial dements are obtrusively religious, though none the less unreliable as regards truthfulness, virtue, and honesty. They are extremely credulous, and readily acquire new doctrines and beliefs, being, as a rule, superstitious (delusional) in intense degree. Ordinarily they are harmless, and may be safely left to work out their own not altogether miserable existence. They constitute the comparatively numerous class described by the layman as the "weak-minded."

Hebephrenia.

Definition and Symptoms. — The term hebephrenia (ἥβη, the mind φρήν, puberty) was originally employed by Hecker and Kahlbaum to describe a form of mental derangement occurring at or about the age of puberty, characterized by a period of primary melancholic depression, followed by mental enfeeblement and quasi-hysterical phenomena. The true type is not at all common, and in many instances in which it occurs hebephrenia does not constitute an insanity at all, except in the most strictly literal interpretation of the word.

It is quite common, for example, to find in the young who are passing through the period of transition from childhood to adult life a slowness or awkwardness in adapting themselves to the new sensations and environmental influences attendant upon the change. There is a physical awkwardness and embarrassment often observed, and correspondingly there exists an awkwardness or embarrassment mentally. Such individuals are silly and mawkish often in manner and speech, unstable in emotional control, lacking in equilibrium in their mental processes of ideation. Should there be inherent or acquired neurotic weakness, all these conditions become exaggerated, and a true insanity develops, the tendency of which is to pass into a terminal dementia, the mind showing an enfeeblement from the very first.

The beginning of the affection is usually marked by a period of sadness and depression suggesting melancholia, but the facies of melancholia is lacking, the sadness is superficial, and there are no profound delusional states. There is none of the furtive secretiveness and reserve of melancholia; on the contrary, the subjects of hebephrenia are often voluble and obtrusive in speech, desirous of attracting attention. They pass from grave to gay in mood, changing quickly and without com-

mensurate cause from melancholy sadness to hilarious laughter or silly jokes, strongly suggesting simulation or hysteria. They display a mawkishly sentimental turn of mind in certain cases, spending much time in writing maudlin poetry or sophomorish effusions in prose. They are incapable of steady application, and are shiftless and thriftless in business. As a rule, they are not vicious or malicious, but they may be guilty of apparently purposeless acts of a mischievous or malicious character, and suicidal attempts occur occasionally, though even here there is often the same suggestion of simulation or "faking" in the lack of earnestness and the half-hearted method of the attempt. Masturbation is said to be the most common cause, and, as might be expected, the male sex is largely in excess among these patients.

PROGNOSIS.—The simple cases, those in whom there is really scarcely any insanity at all in its true sense, recover. After the affection has assumed a well-developed form, however, the ratio of recoveries is very small. A small proportion of cases remain for years in a stationary mental condition, but the majority pass more or less quickly into a terminal dementia.

TREATMENT in these cases is entirely symptomatic. Phosphorus, strychnine, arsenic, salts of gold, protonuclein, and other drugs of the class known as nerve tonics and brain foods are indicated theoretically, but there is little recorded evidence of any positive good from their use. The pathology involved is also problematical, but there is reason to believe that the essential condition is one of developmental arrest primarily, with subsequent premature degeneration of simple type.

ACUTE PRIMARY DEMENTIA.

DEFINITION AND SYMPTOMS.—The form of insanity commonly described under this title differs from the ordinary conception of dementia in several particulars, the most important of which is that it is recoverable. Because of these clinical and prognostic differences many writers do not regard the affection as belonging to the dementias at all. By the French alienists it is known as stupidité, and by certain American writers, notably Spitzka, it is designated primary stuporous insanity. It is in most instances an affection of early adult life, and is characterized by a more or less profound stuporous state or stupidity, with muscular atony and relaxation. Cerebral activity is usually in almost total abeyance, the mind presenting a vacuum. There is no initiative in thought or action. Memory, judgment, and even the emotions, are in a state of paralysis, so to speak. At times in the early stages there is some evidence of emotional action, but it is transient, causeless, and silly in type. All conscious relations to surroundings or to self are soon lost, and the individual in the fully-developed attack is reduced to the lowest level of purely vegetative existence. Somatic function and common sensibility are proportionately involved. The appetite disappears, or, rather, all appreciation of the necessity of eating to sustain life is lost. If food be placed in the mouth, however, the patient will masticate and swallow it mechanically, although the quality, character, or quantity of the food is a matter of indifference. Sometimes it is necessary to push the food back against the pharynx, the patient swallowing purely through

reflex action. There exists an utter negligence and disregard of personal appearance; the habits become filthy, the clothing soiled with urine and fæces. The sphincters are not paralyzed, but there is total absence of any conscious appreciation of their action. Common sensibility is so far impaired that a pin may be driven into the skin with force or the surface burned with hot iron without appreciable indication of pain or suffering on the part of the patient. Such experiments are, however, not free from hazard, in that the wound or burn may prove the starting-point of ugly ulceration or sloughing, the low state of trophic vitality in these patients predisposing them to such accidents under slight provocation. Evidences of this lowered trophic state are to be found in the subnormal temperature, feeble pulse and heart action, dilated pupils, and blue discoloration of the extremities, with coexistent œdema, particularly of the feet, often observed in this affection. Spitzka is authority for the statement that Raynaud's disease is not uncommon —a statement which I do not doubt, though personally I have never seen the two together.

Acute primary dementia is usually of rather abrupt onset, passing quickly, after an initial period of short duration of mental confusion, loss of memory and attention, and silly morbidness, into the state of stupidity already described. It may be caused by any factor or combination of factors tending to produce a state of mental exhaustion. Masturbation and sexual excesses, insufficient food, and physical hardship or privation, debilitating diseases, especially those attended with prostrating discharges—chronic diarrhœas, dysenteries, or suppurations—may act as immediate causes of an attack. Sudden and profound mental shock has been noted as immediately preceding an attack acting apparently as the only cause. The most common and potent immediate causative factor, however, is over-exertion mentally in study and competition at school. In many cases there is a predisposition apparent either in an acquired neuropathic instability or some form of vitiated heredity of neurotic type. The two sexes are about equally affected numerically, with possibly a slightly greater predisposition in males.

Diagnosis.—The diagnosis is ordinarily a matter presenting few difficulties: the age of the patient, the determinable cause, the absence of emotional disturbances in the direction of either exaltation or depression, the relative infrequency of delusions or hallucinations, the total abeyance or annihilation of all mental reflexes—serve to distinguish the affection from either mania or melancholia with ease and promptness. In the cases which are less typical, and in which transient emotional disturbances occur, the melancholy, if it be present, is less fixed and profound than in true melancholia, the facies is absent, and there is an absence of the delusions of dread and terrible foreboding characteristic of melancholia. The initial stage of general paresis may simulate clinically the symptom-picture in primary dementia in some respects, but in paralytic dementia there are superadded the physical symptoms of tremor, abnormal speech, pupillary inequality, and vasomotor disturbance, etc., conspicuous by their absence in the affection we are considering.

Prognosis.—The prognosis in acute primary dementia is good. Gray states that the majority recover, and Spitzka gives the proportion of recoveries as 90 per cent. The more sudden in onset the better the

prognosis. If recovery does not occur, the patient passes into a condition of chronic or terminal dementia, or death occurs from physical disease, usually phthisis.

The duration is from two, three, or four weeks to as many months in those cases which recover.

Treatment is based chiefly upon the physical condition present. Asylum restraint is rarely necessary, provided proper care, food, and medical attention can be secured at home. Such patients rarely manifest homicidal, suicidal, or destructive tendencies, though they may do themselves injury in purely negative ways, involving the necessity of constant oversight and care. From a distinctly therapeutic standpoint all measures calculated to restore bodily vigor, such as abundance of nutritious food, proper rest, bathing, massage, etc., are indicated. Among the drugs which I have found of positive benefit in these cases are strychnine, protonuclein, thyroid extract, and iron, preferably in the form of pepto-manganate or ferratine, the former in doses of a tablespoonful in milk or sherry wine four to six times daily, the latter in doses of five to ten grains three times daily. Strychnine may be advantageously combined with the iron in doses of one-sixtieth to one-twentieth of a grain three times daily. The dose of thyroid extract, perferably given in tablet form, is five grains three or four times daily. Protonuclein may be given in the same way. In one case recently observed general faradization seemed to be of much value in promoting an improvement in general bodily nutrition, with commensurate amelioration of the mental stupidity. There is little if any tendency in these cases to relapses, and if recovery takes place at all, it is usually perfect, leaving no residuum of the mental perversion as an evidence of the attack.

Secondary Confusional Insanity.

Definition and Symptoms.—This is really simply a stage in the development of terminal dementia succeeding the acute psychoses. It represents a transition period rather than an entity, and yet the symptom-picture is often so different from that of ordinary terminal dementia that it is entitled to a separate reference and description. It corresponds by analogy somewhat to the partial type of simple dementia. The mind is not completely obliterated, but there exists a state of confusion and agitation. Following an attack of mania or of melancholia of prolonged duration and obstinate resistance to treatment, the patient is noticed as being less excited on the one hand or depressed on the other. He is, in other words, less subservient to the exaggerated emotional element which had previously dominated mental activity. His delusions and hallucinations are partially retained, but they are mere fragmentary elements and no new ones are elaborated. The morbid propensities to suicide or homicide or destructiveness are either lost or if persistent they lack premeditation or elaboration. Violence, if it occur, is purposeless and without emotional basis. The maniac remains talkative, and even verbose, but less so than formerly. The melancholiac loses his introspective silence, and may become garrulous, but speech is silly, incoherent, and often unintelligible. Such patients exhibit in equal degree at times a purposeless motor activity or restlessness. They are utterly incapable

of any degree of concentration of mind or logical sequence in ideation, and even the identity of the personal *ego* may be lost completely. In some of these cases the residua of the original or primary psychosis persist with sufficient distinctness to enable the observer to determine its nature. These patients are ordinarily far less helpless and dependent than is the case in terminal dementia proper. They are less negligent of the laws of decency, and are far less filthy in their habits. They have sufficient intelligence with which to recognize and make known their more imperative physical necessities, such as food and warmth. They do not require to be fed, and ordinarily are possessed of active appetites, the bodily nutrition remaining good.

The DURATION of this transition period is variable. It may last a few weeks or months or many years. Usually, however, the symptoms will be found upon comparison from month to month to be steadily progressing toward a complete dementia, which is the inevitable termination, since treatment is utterly ineffectual. On account of the occasional outbreaks of excitement or violence which may occur in these cases, and their more or less dependent condition, institution care and control is advisable.

These outbreaks of excitement, by the way, gave rise through Krafft-Ebing to the term dementia agitata, by which this form of alienation is designated in certain textbooks. They are relics, perhaps, of the original mania or periods of agitated agony in melancholia.

TERMINAL DEMENTIA.

DEFINITION AND SYMPTOMS.—While the expression "terminal dementia" might be appropriately applied to all forms of mental decay which are secondary in onset to some acute organic or psychical affection, technically the term is restricted by most writers to that form of progressively increasing mental deterioration which succeeds in the uncured cases of acute vesanic insanities, such as mania, melancholia, etc. Terminal or sequential dements constitute a large proportion of the insane population in all countries. Clouston has estimated them as numbering two thirds of the asylum insane in Great Britain. Here also, as in simple dementia, the degree of mental enfeeblement may vary greatly, though the fundamental defect is the same in character, the essential symptomatic condition being one of quantitative loss of mental function. It is difficult in the dementia which terminates a melancholia of the stuporous or katatonic type to determine the period of transition from the primary psychosis to the terminal dementia. Sometimes this transition is represented by an intermediate condition of confusion and incoherence referred to under the preceding paragraph, entitled Secondary Confusional Insanity. In other cases the element of time alone determines the change in diagnosis. A melancholia with profound stupor or an acute primary dementia continuing unaltered for more than a year or two is probably already across the border-line of a terminal dementia. Ordinarily, however it is not difficult to recognize the symptoms which both positively and negatively indicate the onset of dementia. The first suggestion may occur through a rapid accession in flesh and weight without corresponding mental

improvement. The facies of melancholia becomes less characteristic of mental suffering, showing an increasing tendency to absence of all expression, or vacuity. The tension in the facies becomes relaxed. Emotional disturbances in both mania and melancholia subside in degree or disappear altogether. The maniac becomes calmer, less aggressive, less voluble in speech, more collected, or rather less agitated, in manner. The melancholiac loses that vestige of intelligence which, no matter how profound his stupor, was still apparent to the trained observer. He loses that semiconscious, semispastic resistance to forced movements; he no longer refuses food, and his insomnia may disappear altogether. The circulation is better, the pulse rate dropping from 100 or over or rising from 40 or 50 to the normal. Gradually or quickly the more characteristic symptom-picture of dementia is developed, and the nature of the case is no longer in doubt. There is a period of stupor which apparently represents an exhaustion reaction which sometimes immediately precedes recovery in certain cases of both mania and melancholia, which has been not infrequently mistaken for dementia. It is usually of short duration, and does not long obscure the diagnosis, but its existence should always be borne in mind. When once fully developed, terminal dementia represents the most degraded and repulsive level to which man can descend. Such dements are utterly disgusting and indecent in habits, defiling themselves with filth and often filling their mouths with dirt or refuse. They usually sit or lie upon the floor in squalid dishabille, passing the time in noisy, meaningless chatter or senseless grimaces and gesticulations. Sometimes they are destructive and noisy, especially at night, requiring hypnotics. Others are apathetic or stupid in intense degree, the mind being apparently in a state of as complete extinction as it is possible to conceive. Such patients are mere automata, and have to be cared for with the same attention which is given to infants. In certain cases there is for years some preservation of intelligence in modified and attenuated degree. The patient can understand simple instruction, and can perform certain duties, as with simple partial dementia. He must be told what he is to do again and again, however, and has absolutely no power of initiation. Such patients make up the class of working patients in asylums, and are usefully and beneficially employed in simple duties. All dements, no matter what the degree or type, are liable to develop sudden outbreaks of excitement, attended with impulsive violence or destructiveness—a fact to be borne in mind, especially if the attempt is made to care for the patient at home.

TREATMENT.—Treatment in dementia of the sequential or terminal type is directed solely to the maintenance of physical health, since there is no cure or relief for the mental condition. The food especially should be wholesome and abundant, since there is quite characteristically present a tendency to emaciation and malnutrition even where the appetite is enormous. Patients with dementia should never be left alone when eating, since they have the habit of bolting food in large masses, sometimes with fatal strangulation as a result. Careful, patient training will occasionally cause some improvement in filthy and injurious habits, but the task is a difficult one and ordinarily hopeless.

The Organic Dementias.

The dementia which occurs as a sequence of, or in association with, gross structural disease of the brain is merely one symptom in the clinical history of the case and often a symptom of secondary importance. Any intelligent conception of such dementia must necessarily involve a discussion of the primary cause. Such discussion is not only impossible within the limits of this article, but it would be equally inappropriate here. The reader is therefore referred to the various articles found elsewhere upon paretic dementia, syphilis of the brain, cerebral traumatisms, hemiplegia, epilepsy, alcoholism, etc. (See Index.) Senile dementia, since it constitutes an entity and is not described elsewhere, I shall include as the final sub-type to be considered.

Senile Dementia.

Definition and Symptoms.—Senile dementia proper, using the term in its strictly literal significance, is a condition which, as it occurs ordinarily, might be considered almost a normal epoch in human life. It is the seventh stage of Shakespeare's man. Just as with advancing years the physical man begins to degenerate and disintegrate, so the mental man takes on a process of corresponding weakness and decay. The somatic substratum of mind takes part in the general retrograde metamorphosis incident to increasing years, and the dependent psychic functions of memory, attention, and reason gradually fade and dim in commensurate degree. When slight in degree and somewhat proportionate to the physical decay in its advancement, the mental state is simply one of slight weakness, and can scarcely be termed a true dementia. In certain individuals the mental decay advances more rapidly than the physical, and reaches comparatively quickly a low level. The most prominent symptom is loss of memory, especially for recent impressions and events, the details, even in minute degree, of incidents and events long past being retained. There is exaggerated emotional excitability, tears and anger alike resulting from causes the most trivial and insignificant. Such patients are credulous and impressionable as children are, and are easily influenced by others, judgment and reason both being seriously impaired. Their habits are careless, they lose all recognition of time and place and even, at times, of identity. Rarely, if ever, is there observed any homicidal tendency, nor are they, as a rule, actively destructive. Suicide is, however, occasionally attempted, and sometimes successfully. It is essential to the diagnosis that evidences more or less conspicuous of physical senility shall be coexistent. These consist in varying degrees of bodily decrepitude, manifested in tottering gait, wrinkled skin, and tremulousness in muscular action and in speech. Usually superficial evidences of a general atheroma are to be found, and arcus senilis is common. The special senses are impaired sometimes in marked degree, notably vision and hearing. It is partly through this impairment or defect of special and common sensibility that the mind fails to receive new impressions, ceases to advance, and begins its backward march, which ends in the annihilation of intelligence.

The age at which senile dementia occurs is ordinarily anywhere between sixty and ninety. Premature senility, both physical and mental, occurring under the age of sixty is by no means uncommon, and cases have been reported under the age of fifty. Any of the numerous causes of vascular degeneration may predispose the individual to premature decay. Early senility is sometimes observed as a sort of family trait. Worry and want are factors tending to aggravate the condition and to hasten its advance.

Prognosis.—Although the prognosis is in most instances hopeless as regards recovery, cases are occasionally observed in which marked improvement temporarily follows active tonic and supportive treatment, directed chiefly toward the betterment of the physical state. One of the most important aspects of the subject of senile dementia relates to the medico-legal status of such individuals. The testamentary capacity of senile dements is especially important because of the frequency with which it is made the basis of will-contests. Contracts, marriage, partnership agreements, and many other relations in civil law are quite often subjects of litigation in connection with this type of alienation. Of course in well-marked cases no doubt can exist as to the mental incapacity, but occasionally it is exceedingly difficult to determine the problem. No general test or standard of capacity can be fixed arbitrarily in these cases, but each must be studied individually with regard to this point.

Pathology of Dementia.—It is exceptional to find a normal brain in post-mortems upon dements, no matter what the clinical type. The pathological findings are, however, exceedingly varied in both character and degree. There is a special pathology in paretic dementia and in the dementias associated with epilepsy, syphilis, and alcoholism, which will be found in the separate articles upon these subjects. In the dementias dependent upon gross organic cerebral disease, such as tumor, meningitis, apoplexy, and other acute vascular lesions with ramollissement, the pathology is that of the organic lesion, and has no place here. The pathological changes observed in the vesanic dementias may, and usually do, include the skull, the meninges, the bloodvessels and lymph spaces, and the tissues of the brain itself, the latter either grossly or in minute cellular and connective-tissue alterations. The skull is usually thickened; it may be of increased weight or, as is sometimes the case, lighter in weight, with or without thinning of the walls. The thin skull-cap is rather characteristic of senile dementia, while the thickened, heavy, and sometimes enlarged skull is quite common in secondary dementias. Bevan Lewis states that cranial hyperostosis is present in from one fourth to one third of all fatal cases of insanity. The skull may show other abnormalities in osteophytes, plates, and small exostoses are occasionally observed. The meninges may be the site of adhesions, especially frontal, so-called arachnoid cysts, dural hæmatoma, or opacities. As a matter of fact, one or more of these conditions is quite common. The walls of the bloodvessels are often thickened and infiltrated with fatty granules and hæmatoidein. The perivascular spaces are enlarged. The brain as a whole is sometimes altered in size in the direction of an atrophy, or there may be

areas of wasting in certain regions, with partial or complete effacement of convolutions. The most characteristic and frequent alteration in the tissues proper of the brain consist in a diminution or relative deficiency in the pyramidal cells, with increase, sometimes enormous, of the spider or scavenger cells and neuroglia. Sometimes the cells are not so markedly diminished in number, but they present a shadowy indistinctness of outline; the nuclei are enormously swollen or shrunken and changed in form, and they do not stain well with carmine. These cells may exhibit all stages of granular or pigmentary degeneration in some cases. The medullated fibres of the white substance are, according to Bevan Lewis, affected with a chronic degenerative process which occurs as so-called miliary or genuine sclerosis or as colloid degeneration. The pathology of dementia is, in short, as has been stated, one which may include any and all forms of degenerative change in the brain.

PARANOIA.

By WILLIAM NOYES, M. D.

SYNONYMS.—Monomania; Systematized insanity or Systematized delusional insanity; Intellectual insanity; Primary insanity; Primary delusional insanity; German Primäre Verrücktheit; Originäre Verrücktheit; French Délire partiel; Délire systématise; Folie systématisée; Monomanie intellectuelle.

DEFINITION.—Paranoia is a primary, chronic mental disease, usually arising on a degenerative basis, and characterized especially by systematized delusions.

The name "paranoia" (ἡ παράνοια) is derived from *παρα*, near to, at the side, near, across; and *νοεω*, to think, to be wise. The term may thus be taken to indicate a qualitative and quantitative alteration of intelligence. Plato in his *Phädrus*, and later Aristotle, used it as synonymous with *μανια*.

HISTORY.—The history of the term paranoia may be said to be the history of the attempt to find a suitable and universally acceptable designation for that large class of mental diseases where the intellectual processes are involved, in distinction from the feelings and emotions, as in mania and melancholia. Ten or fifteen years ago it would have been possible to give a definition of paranoia that would meet with fairly general acceptance, but at present (in 1898), this cannot be done, and the confusion has now become so great that there is almost no form of mental disease apart from mania, melancholia, and the organic brain diseases that is not classed by some writer as a form of paranoia. The inability of alienists to frame a classification of mental diseases that all could unite upon has always been a standing reproach to psychiatry, and regret must now be keener than ever that a term that at one time bade fair to be of more service than perhaps any other single term in psychiatric nomenclature should be so loosely used and so widely applied as now utterly to fail to call up a definite clinical picture, unless the term is itself qualified by the name of some particular author. No difference could be greater, for example, than the use of the term by Krafft-Ebing and Spitzka on the one hand, and on the other by Ziehen, who recognizes a paranoia following hereditary degeneration, trauma capitis, chronic alcoholism, puberty, the senile state, the climacteric, acute febrile conditions, epilepsy, hysteria, exhaustion, and periodic, circular, and recurrent insanities.

It will thus be seen that paranoia has the most extensive, but at the same time the most ill-defined, acceptance of any word in psychiatry.

The term was used by Vogel in 1764, but with nothing of its present

signification, and by Heinroth in 1818, to describe certain secondary states of mental exaltation. Esquirol in 1838 described the intellectual monomanias, and reported several observations in which he laid stress upon the ideas of grandeur. The first to advance the modern conception of paranoia was Griesinger in 1848, who described systematized insanity under the title *Die Verrücktheit*, but he considered it to be always secondary to melancholia or to mania.

The modern conception of paranoia as a primary disease dates from Snell in 1865, who first clearly described a fundamental form, distinct from mania and from melancholia, and characterized by the primary appearance of a series of delusional ideas of a mixed nature (persecution and grandeur) and accompanied by hallucinations. Snell still held to the older term, monomania, but his description marks a distinct advance in psychiatric classification. The delusions are not, Snell held, as in other forms, an echo of the whole mental life, a tendency to generalization. The most striking symptom is a delusion of persecution, with an exaggeration of the sentiment of personality, and a tendency to activity rather than to passivity, differing in this from melancholia. There are also delusions of grandeur uniformly primary, contemporary, or consecutive to the delusion of persecution, and bringing about a change in the personality. The development of these forms of insanity is slow, but sometimes they develop quickly on account of the mental excitement. The prognosis is bad, yet in these cases a true consecutive dementia is never observed.

Griesinger in 1867 retracted his former opinion, and agreed with Snell as to the primary origin of the mixed states (delusions of persecution and of grandeur), and he gave them the name of *primäre Verrücktheit*. He also described the hypochondriacal and erotic forms. This German term, *Verrücktheit*, introduced by Griesinger, is in many ways an admirable one; in English its nearest equivalent is cracked or crazy, and no word better describes the mental condition of the paranoiac than craziness.

Westphal in 1878 first described the acute, curable form of paranoia, "characterized by the sudden explosion of hallucinations, especially of hearing, accompanied by ideas of grandeur. At the height of the disease the incoherence is such that it suggests a febrile delirium. In certain instances there are impulses; in others, on the contrary, there is complete dejection." It is concerning this acute form of Westphal that there have been the greatest differences of opinion, and now, after almost twenty years of discussion, the question cannot be said to be any nearer a settlement. Schüle, Meynert, Mendel, and others follow Westphal, while Krafft-Ebing gives the weight of his great authority against the existence of an acute paranoia. In this condition of the subject, a general agreement apparently being out of the question, it merely remains for each writer to state on which side he finds himself, and the present writer prefers to follow Krafft-Ebing.

To the writer's mind the most complete and philosophical conception of paranoia is unalterably bound up with the idea of *chronicity*. There can be no more beautiful clinical pictures of mental disease than are furnished by the life-history of many saints, martyrs, religious enthusiasts, and fanatics, for whom, if we agree to call them paranoiacs, we have a gen-

eral descriptive term than which no better has ever been found, and which gives us at once a clear insight into their mental processes; and at the same time the logical and systematic evolution of their false ideas furnishes us with a clue to the immense influence these people have exercised in many periods of the world's history. To read history with this conception of paranoia is to gain a new light on the forces that have moved men and overthrown kingdoms. Under the guiding and overmastering influence of a paranoiac we see France victorious under a Joan of Arc; thousands of men, women, and children leaving their homes to perish in foreign lands in the crusades under a Peter the Hermit; a new religion founded through the visions of a Swedenborg; a kingdom, Bavaria, nearly bankrupt through the extravagances of a Ludwig; a rise of the people to full control of the government under the teachings inspired by a Rousseau; a nation thrown into mourning by the act of a Guiteau; and men and women the world over regulating their daily lives and sustained in every hardship and misfortune that the world can offer by the ecstatic visions of some saint or martyr. Surely, the mental conditions of which the above are types, extending over years and disappearing only at death, are, at the last analysis, essentially different and distinct from an acute, curable, mental disturbance that may follow delirium tremens, childbirth, or an attack of typhoid fever. It will be of marked assistance in gaining a clear idea of the narrower and, in the writer's opinion, more proper conception of paranoia to consider what mental diseases it is allied to. Thus, Krafft-Ebing places it under the general broad grouping of the mental degenerations, the other members of the group being constitutional affective insanity (folie raissonnante), the periodical insanities (which include the circular forms), the forms with morbid impulse, such as dipsomania, and periodical insanity arising sympathetically, as menstrual insanity. The near and intimate relationship of these conditions with the classical cases of paranoia that have been cited will be apparent at once. They are all dependent on some inherent vice or weakness of the nervous system, something that came into the world with them, and only needing the proper exciting cause to burst forth into full-developed mental disease.

ETIOLOGY.—Of all the factors entering into the etiology of paranoia, mental degeneration is by all authors admitted to be the chief. Paranoiacs above all others have the neuropathic constitution and the *reizbare Schwäche*, or irritable weakness. As signs of the neuropathic constitution may be mentioned severe reactions to atmospheric changes; very high temperature, with striking irregularities of the curve in febrile conditions, with typical course at other times; severe nervous reactions, even to the point of convulsions; neuroses and psychoses at the physiological periods of life (dentition, puberty, menses, climacteric); hyperæsthesias; vasomotor disturbances; nystagmus, strabismus, stuttering, contractures, and even innervation disturbances of muscles (grimaces, etc.); disturbances of the sexual life are especially common, either failing entirely or being abnormally strong, appearing even in early childhood and seeking perverse gratification. These individuals are especially the subjects of their emotions, quickly running the whole gamut from extreme gayety to deep hypochondria. Prolonged and continuous exer-

tion, either physical or mental, seems to be an impossibility to many of them, through the quick exhaustion of their nervous force and their special liability to ennui. The higher ethical feelings and strivings are lacking, but there are often a sham religiosity and a fondness for the emotional forms of religion.

The etiological factors vary, of course, according to the views of the individual writer—whether he construes paranoia in the broader or more restricted sense. Those describing the acute hallucinatory form, for example, give as etiological causes hereditary predisposition first of all, then epilepsy, hysteria, alcohol, lead and cocaine poisoning, and the majority of febrile and post-febrile psychoses; and it is here that are classed the inanition psychoses, those following mental and bodily overexertion, insufficient nutrition, severe anæmia, repeated childbearing, prolonged lactation, and sexual excesses. The majority of these causes are what go to make up a typical inanition psychosis, but to designate such a psychosis acute paranoia because the dominating symptoms are hallucinations and delusions appears extremely illogical. Ziehen, who is the author above quoted, gets a complete recovery in 70 per cent. of the above class of cases. Undoubted exciting causes of the outbreak of paranoia in its more restricted sense are puberty, the climacteric, uterine diseases, and masturbation.

Pathology.—At the present time it is not possible to say that there are distinct and clearly marked pathological changes peculiar to the brains of paranoiacs. Spitzka gives as signs of the insane temperament—(1) atypical asymmetry of the cerebral hemispheres, as regards bulk; (2) atypical asymmetry in the gyral development; (3) persistence to embryonic features in the gyral arrangement; (4) defective development of the great interhemispheric commissure; (5) irregular and defective development of the great ganglia and of the conducting tracts; (6) abnormalities in the development of the minute elements of the brain; (7) abnormal arrangement of the cerebral vascular channels.

Donaldson's attitude, in his *Growth of the Brain*, is very conservative: "Since the hemispheres of the brain are, in the fœtus, smooth, and only gradually become marked by fissures, it is observed that early [*sic*] disturbances in their growth, which later are almost invariably associated with disturbances in the intelligence, are also accompanied by abnormalities in fissuration. But farther than this it is hardly safe to go."

Several marked examples of these changes in the brains of paranoiacs have been reported. One of the best known is that of Muhr, where there was notable and unusual asymmetry in the two halves of the brain, attributed to a reduction in volume of the left half of the cranial cavity and of the left internal carotid artery. In a case reported by Kirchoff the skull was microcephalic, the left hemisphere did not cover the cerebellum, and the cerebral commissures were defective. Others have found evidences of inflammatory processes in the meninges; ependymitis of the ventricles, especially the fourth; anomalies of the basal arteries; and atrophy of the cortex.

Regarding the changes found in these conditions, Folsom holds with Schüle that similar and as extensive gross intracranial anomalies are found in persons who could not be called of unsound mind; but any defect of brain is far more common among persons of unsound than of

sound mind, and therefore in doubtful cases it is of a certain value as corroborative evidence of mental infirmity or impairment.

We may therefore conclude, with Sander, that paranoia is not a single process, but frequently a conglomerate of different processes displaying themselves in the same individual, and we must therefore expect various pathological changes in the brain. How varied these processes may be is seen from the findings in the brains of the two most noted paranoiacs of modern times—Guiteau and Ludwig II. of Bavaria. In Guiteau's brain there were adhesions of the dura mater to the inner table of the skull along the longitudinal sinus, and adhesions of the dura to the pia and to the brain at a spot on the vertex. There were thickening and milky discoloration of the "arachnoid" over the sulci in the whole convexity of both hemispheres of the brain. The walls of the bloodvessels of the brain were affected with granular degeneration in numerous minute areas throughout the cortical layers. In many places there was degeneration of the nerve cells and cells of the neuroglia in the gray matter. There was also asymmetry of the convolutions. There were thus evidences of past inflammation. But all these changes are frequently found in the brains of the chronic insane from other forms of insanity.

Ludwig's brain presented many similar appearances. The skull was somewhat small for the size of the body (he was six feet three inches tall), and was asymmetrical. The calvarium was unusually thin. There were degenerations of the bony tissue on the inner plate of the skull, especially at the frontal bone. There was an osseous growth two millimetres in length springing from the clivus, and the surrounding bony tissue was porous and brittle. The left petrous bone bulged into the temporo-sphenoidal lobe about one centimetre; the pia was thickened, especially in the frontal region; the "arachnoid" was thickened, with milky discoloration. At one point the pia was thickened and hardened, and had impinged on the table of the skull, causing absorption. Here, again, there are evidences of inflammatory processes, but nothing that can be looked on as absolutely pathognomonic.

Symptoms.—Paranoia may be considered an exaggeration or still further development of the insane diathesis or the psycho-neuropathic temperament. It is primarily a disease of the ideational life, its chief symptoms being delusions and imperative conceptions, with outbursts of sudden overmastering hallucinations. An all-pervading and ever-present egoism is its most notable characteristic. The paranoiac is above all a reasoning being, but his conclusions are faulty through an inherent defect of his logical apparatus, with a weakened power of criticism and reflection. In many cases the evolution of the disease is so slow that it may be years before the subject is recognized to be insane, and his insanity even then may seem to be but the exaggeration of his natural temperament. The paranoiac is exalted or depressed according to the nature of his delusions, but there is this difference between the depression or exaltation of paranoia and of melancholia and mania—that in paranoia the condition is primary and a reaction to the delusions, and not, as in the latter cases, a secondary reaction to the disturbed emotional state. In melancholia the patient is depressed because of his own misdeeds or sinful actions toward the external world, while in

paranoia it is the external world that is all wrong and keeping him out of his rights through no fault of his own. Through his inability properly to orient himself the paranoiac builds up a most imposing structure on entirely erroneous and fictitious ideas and conceptions. Grant the premises, and you often cannot find a flaw in the argument. Times of great political excitement are especially favorable for bringing to the front these erratic and often dangerous individuals, and the murder of Garfield is still recent enough for all to recall the trains of false reasoning by which Guiteau finally brought himself to believe that he was being kept out of his just rights by the government, and that the only way to obtain justice for himself and to save the country was to remove the President. The paranoiac therefore believes and acts exactly as if his delusion were true.

The mental powers of paranoiacs are usually fair, and there are cases where they have risen to the highest rank intellectually and have left an indelible stamp on the world's history. Such a case was that of Rousseau, who was the first to voice the aspirations and longings that a few years later bore fruit in the French and American Revolutions, yet the life-history of Rousseau would make a classical description of the persecutory form of paranoia. Nowhere can the workings of the paranoiac mind be better seen than in his *Confessions*, beginning as it does with an impious reference to the Deity, and proceeding with an egotism that has never been equalled to lay bare the coarseness and foulness of his own youthful mind; appealing with most fervid terms to the mothers of France not to give over to others the care and rearing of their children, and then without a qualm leaving his own children at an orphan asylum. Time after time he was obliged to flee from his imaginary enemies, misconstruing all the efforts of his friends to assist him, and finally, in all probability, dying by his own hand when the fear of his enemies became too acute to be longer borne. And yet this was the man whose reformatory ideas in education have lasted to the present time, and whose political ideas are practically embodied in the Declaration of Independence.

In their youth these patients are the victims of morbid subjectivity, and often have hallucinations at a very early age, as was the case with William Blake, the poet and artist, who at ten years of age saw a tree filled with angels. Every conceivable incident of their daily life and the chance remarks, tones, and gestures of those they meet are seized upon by these patients and woven into their cobweb structure of delusions. The friends of a patient under the writer's care have recently brought him some magazines, four or five years old, in which he finds mentioned two names the same as those of two fellow-patients; this is to him proof positive that these two fellow-patients are concerned in the scheme by which he is deprived of his liberty. The hallucinations are often kept under control to a remarkable degree, and there may be years at a time that the patient has an intermission with a full appreciation of his condition, but after a time the disease begins again where it had left off. Dissimulation, too, must always be guarded against. A noted historical example of this is where a French patient was able to convince every one of his mental soundness, and was about to be discharged by the government inspector; but it being necessary to sign his

name, he could not, even with liberty so near him, give up his delusion, up to that time successfully concealed, and he signed himself "The Christ."

Hallucinations of all the senses occasionally break out with such great violence that the patients become acutely maniacal, becoming wholly unoriented and confused.

One of the most marked characteristics of the disease is the so-called *transformations* of the delusion. The patient, imagining himself watched and spied upon, his footsteps dogged, and himself made the subject of derogatory remarks, concludes that he must be a person of importance, and all his delusions become tinged with self-importance, and he demands that he shall be taken at his own valuation. The process may be reversed, and delusions of persecution may be the sequelæ of delusions of grandeur, or the two forms of delusion may exist together; indeed, Spitzka holds that a demarcation between paranoia in which persecutory delusions predominate and that in which ambitious ideas prevail cannot be defended—that they are merely the same disease manifesting its symptoms under different external guises. A marked characteristic of certain paranoiacs with exalted ideas is their attitude and gait; and it is often possible to make a diagnosis in a group of asylum patients from this *paranoiac gait*, with the body erect, even to bending backward, arms carried stiffly by the side, and measured steps, all indicating the height of egoistic self-importance.

To attempt a description of the delusions of paranoia would be to catalogue the contents of each paranoiac's mind, so varied and innumerable are the delusions; but it is the *process* that special stress must be laid upon, by which a *Wahngebaude*, or "castle of delusions," is erected in which the whole life of the patient is spent.

The most trivial and simple incidents of the daily life of these patients are distorted and twisted until they become the foundation-stones of the delusional structure. Chance remarks overheard in the street, references in books and newspapers, and even reminiscences of dreams, all furnish material which their perfected logic weaves into the warp and woof of their new life; for it must never be forgotten that an essentially new personality is the final outcome of this delusion-building —a personality that is in every way consistent with the dominating false ideas.

There are three classes into which the delusions naturally divide themselves: (1) of delusions of suspicion and persecution, often of unseen agency, based on hallucinations; (2) of exaltation; (3) of delusions transformed from sensations. The outbreak may be acute, with overmastering hallucinations, giving rise to delusions, and with secondary reactions of assault and violence or even suicide; or the onset may extend over months or years. Perverted sensations, anæsthesias, and paræsthesias lead to transformed sensations. Here arise the delusions of conception by the Deity or of ravishing, and the gravest scandals may arise from false accusations of men above reproach. Hallucinations of all the senses may occur, the frequency being given as first hearing, then feeling, vision, taste, and smell.

COMPLICATIONS AND SEQUELÆ.—Melancholia, general paralysis, alcoholic and epileptic insanity, and the periodical forms of mental dis-

case are reported as complications of paranoia. When these develop, as well as when senility comes on, the resulting dementia must not be considered as a result of the paranoia, but of the complicating disease.

DIAGNOSIS.—From the slow development of the disease, often seeming but a part of the character itself, a diagnosis is often impossible until the delusions have become so firmly fixed that all hope of doing the patient any good has long gone by. Paranoia may at times simulate mania, melancholia, hypochondria, and general paralysis. Hallucinations are very rare in mania, and the characteristic "flight of ideas" of mania is absent in paranoia. Delusions of poisoning, of persecution, and of grandeur are present in general paralysis, but their unsystematized character and the behavior and mental failure in general paralysis should offer the means of diagnosis. In melancholia and hypochondria the depression and self-depreciation are usually subjective and referred to the unworthiness of the patients themselves, and not to external agencies, as in paranoia.

PROGNOSIS.—The prognosis of paranoia is extremely grave, almost hopeless. In over 500 cases Krafft-Ebing has never seen a recovery, although admitting the occurrence of lucid intervals, especially in the beginning of the disease, but of short duration, while later there may be deep and lasting remissions with complete latency of the symptoms. A remarkable historical example of almost complete recovery was the case of John Bunyan, who had mental strength enough largely to overcome and hold in check his hallucinations and to prevent them from gaining the complete mastery. The tendency is toward a progressive mental enfeeblement, but not to complete dementia. Many of the most useful and hard-working patients of the asylums are the paranoiacs who have passed through the storm and stress of their disease, and have settled down into a quiet, contented routine.

The great advances of the study of educational processes in recent years and the present universal interest in child-study are hopeful signs that there may be in the future a more general early recognition of the psychopathic temperament and the insane diathesis, and the adoption of judicious educational methods with these unfortunates. In this only does there seem to be any hope of reducing the number of paranoiacs in the world.

TREATMENT.—The protection and safety of society are the chief considerations in dealing with paranoiacs, and for this reason asylum treatment in the majority of cases is absolutely demanded. There is no other class of the insane from whom asylum physicians and court officers stand in so much danger. Matthew D. Field has made a careful study and report of Mary Anderson's persecutor, the paranoiac Dougherty, who was released from the asylum against the physicians' advice, and then justified their diagnosis by returning and killing one of the assistants. It is the possibility of such tragedies as this that must always be borne in mind. The suddenness with which the delusions against one person may change to another affords no permanent safety except in seclusion. Suicide is rare, but may occur as a result of the terrible and distressing delusions.

GENERAL PARESIS.

By LANDON CARTER GRAY, M. D.

DEFINITION.—General paresis is a cerebral disease of chronic, remittent type, characterized by dementia of a very gradual onset, usually merging into mania or melancholia, generally with stupid and expansive delusions, and accompanied by tremor, ataxia, pupillary alterations, and eventual paresis.

ETIOLOGY.—The conditions which bear a causative relation to general paresis are—sex, age, heredity, occupation, social position, other insanities, cranial traumata, climate, locality, and race, syphilis, alcoholism, improper food or poisons, spinal disease.

Males are much more frequently attacked than females, and a careful analysis of statistics made by Mickle shows that about four of the former are affected to one of the latter. To the greater tension and exposure of men in active life, and their greater tendency to errors of hygiene and diet and to dissipation, is probably largely due this difference between the sexes, although it should be remembered that the female is by nature the more viable organism.

The disease chiefly occurs between the ages of thirty and thirty-five, although exceptionally it may occur before or after these dates, sometimes as early as the sixth, and occasionally as late as the sixtieth, year.

In my experience hereditary paralytic dementia has been rare, although in most cases a neurotic heredity has obtained.

Naval and military careers are said to be conducive to paralytic dementia. Mickle found that the regiments of the English Guards furnished many cases, and the disease was frequent among the soldiers of the First Napoleon.

It is stated by the same author that of 64,642 persons admitted to insane asylums in England and Wales, 5.91 per cent. of the private admissions were general paretics, while of the pauper admissions 8.21 per cent. were such. Jung asserts that 31.8 per cent. of the males of the better class were paralytic dements, but only 1 out of 109 females; and Laehr found only 3 cases of paralytic dementia among 786 females of the better class. Colin maintained that in the French army three fourths of the cases of insanity among the officers were of this variety.

Without doubt the ordinary psycho-neuroses of long duration sometimes pass into paralytic dementia, and this has been especially noticed by Calmeil and Marce in cases of hallucinatory insanity, their patients having lasted three, four, five, and thirteen years respectively.

That in certain cases cranial trauma acts as a cause there can be no doubt.

There are many curious facts about race, climate, and locality in the causation of paralytic dementia. It is said to be extremely rare in

Ireland, many of the large asylums containing no case of it, and but 2 to 3 per cent. of the admissions to the Dublin Asylum consist of this disease, while it is almost unknown in the Belfast institution, where, Mickle has stated, "the population is chiefly of Lowland Scotch origin, and really of Saxon blood, while the Celts of Wales, Cornwall, and the Scotch Highlands have a considerable share of the disease." Yet the Celts in the south of Scotland are seldom affected by it. The disease has increased in the south of France during the last two generations. Dr. Luther Bell is said to have first recognized it in this country in 1843. There was not a single case in the Toronto Asylum when Dr. Workman entered it in 1853, but from January, 1865, to July, 1875, there were 72 deaths accredited to paralytic dementia.

The different provinces of Germany, various parts of Belgium, the several counties of England, and the separate states of Italy exhibit widely differing percentages. In Portugal the rate is about 3 per cent. of all the insanities. In Constantinople, judging by the institution on the Asiatic side in Scutari which was visited by Mendel, the disease was relatively frequent, and increasing in number, according to the statement of the superintendent. In Rio Janeiro, Brazil, there were 11 cases in 297 insane. It is a rare disease in Cuba, where it occurs oftener among the negroes than with the native whites. It is said to be frequent in Australia.

The relationship of syphilis to paralytic dementia is very thoroughly set forth in a most excellent and carefully compiled monograph written by Morel-Lavallee and Belières in 1889, in which the authors very decidedly show that syphilis is admitted by competent observers in all the countries of Europe to be a frequent cause of this disease, though there is no means of differentiating these cases from others of non-specific origin. It is furthermore very conclusively proven by these authors that a disease which closely simulates paralytic dementia is the syphilitic pseudo-general paresis of Fournier, which may be induced by focal lesions of the cortical or subcortical regions of the cerebrum, or by syphilomata, causing fusion of the cerebral membranes and eventually cortico-meningeal infiltration. These conclusions were illustrated by some 46 cases which were carefully collated and presented in detail. Although the authors offer no tangible means of differentiating syphilitic pseudo-paresis, yet a study of their cases will show that there was a precedent history in every case of cerebral syphilis, and this was also shown in my cases. The duration of the disease may be said to vary from a few months to many years, although the average expectancy is from three to five years.

Pathological Anatomy.—It is only recently that we have begun to obtain a clear knowledge of the pathological alterations of the cerebral tissues characteristic of this disease. In the past, striking results of morbid alterations have been described and have passed as explanatory of the textural changes, although they have been in reality the results of a long series of microscopic alterations that have themselves eluded our ken. Our lack of complete knowledge of the histology of the cerebrum and our defective hardening methods are chiefly accountable for these failures. By the marvellous bichromate-of-silver stain, first discovered by Golgi and subsequently applied by the genius of

Cajal, and its subsequent modifications and alternatives, our knowledge of the wonderful mechanism of the brain has been revolutionized; and this, in conjunction with the method of so-called "control" experiments —*i. e.* investigations of normal tissues by the same method—has set aside many descriptions of deceived investigators. For example, cerebral pathologists are not agreed as to whether paralytic dementia primarily begins in the nerve fibres and the nerve cells, or whether its onset is to be found in the bloodvessels and their lymphatic appendages. This is an idle strife of words as a matter of fact, for there have been recorded no pathological observations capable of proving that all the fine medullated nerve fibres of the cortex, such as are described by Cajal and his followers, have been thoroughly examined, for the simple reason that the staining fluids used in the pathological cases will not affect all this network of fibres, and it is therefore impossible to say that authors who have described only vascular lesions in the early cases may not have unconsciously and unintentionally overlooked certain degenerations in these nerve filaments.

Binswanger and Frohman have demonstrated that all the hardening methods and preservative solutions, such as absolute alcohol and chromic acid, alter, and often entirely destroy, the original structure of the cells. And this is not all, for certain alterations described as pathological have been proved to be due to immediate post-mortem change. Thus, Zacher describes with great particularity certain microscopical details in nerve fibres which he would have us regard as the earliest signs of the disease, such as brittleness, varicosities, irregular nodules, normal axis cylinders shown through transparent medulla, irregular curved shape, atrophy, and final disappearance, observed especially in the tangential fibres and the sublying strands; and yet Binswanger has ruthlessly demonstrated the same appearances in the brain of a man who has been beheaded! In spite of the difficulties of the histological technique, however, it can now be said that the changes affect—1, the nerve fibres; 2, the nerve cells; 3, the neuroglia and the vessels; 4, the membranes.

In 1880, Tuczek thought there was a progressive disappearance of nerve fibres from the anterior, basal, mesial, and lateral portions of the cerebrum as far back as the anterior central or ascending frontal convolutions, the gyrus rectus at the base of the brain being also implicated in some acute cases. The fibres which are known as the tangential fibres of the cortex, as well as the superradiating and interradiating fibres, were those involved. In these cases there was an increase of fibrous transformation of the interstitial tissues, as well as a decrease or disappearance of the nerve fibres, with an abundant development of abnormally large spindle or Deiters' cells. Tuczek claimed that these pathological appearances were pathognomonic of paralytic dementia, and he still holds this view; but, while the facts to which he first called attention have been abundantly confirmed, his interpretation of them has not met with the same happy fate, as the same fibres are frequently wanting in other forms of mental disease. Zacher, examining the brains of 12 paralytic dements, endorsed Tuczek's statements; but in 2 brains of paranoia, 3 of ordinary paralysis, and 3 of senile dementia he found the same peculiar disappearance of nerve fibres as did Eminghaus in post-febrile dementia, Keraval and Targoula in secondary dementia, Jendrassik in tabes dorsalis without mental impairment, and Greppin in several cases

of what he calls post-melancholic dementia; whilst Kostiurin, examining the brains of 12 perfectly sane people varying in age from sixty-five to sixty-eight years at death, observed a considerable loss of tangential fibres in the parietal lobe. Greppin, moreover, could not find in a case of paralytic dementia of nine months' duration any lack of the intracortical nerve fibres. Indeed, Fischl points out that there are great physiological differences in the number and calibre of the cortical nerve fibres, and that a sufficient regard has not been paid to these physiological conditions. Binswanger, who has lately made the most thorough study of the pathological data about this disease, believes that it is only possible to reach conclusions as to the disappearance of fibres in the uppermost or tangential layer and the interradiating fibres, whilst the findings in the superradiating fibres are uncertain. There can be no question that the bloodvessels are implicated at an early stage, although, as has been said previously, there is no doubt as to whether they are at first affected. These vascular changes are in the nature of a hyaline degeneration. A functionary and passive hyperæmia results, which is attended by an outflow of numerous red and white blood cells through the wall of the bloodvessels. Because of a certain amount of destruction of tissue, as well as of the transudation, there soon follows a dilatation of the extravascular lymphatic system, and the veins become distended with red blood cells, although, curiously enough, no white corpuscles are seen within them.

Of no less interest have been the alterations in the basic and connective-tissue substance of the brain, as well as in the vessels, inasmuch as upon the determination of them rests the question as to whether the disease is really primarily an interstitial encephalitis. At the outset the important question arises as to how much of what was formerly regarded as basic and connective-tissue substance is really nerve processes which have been shown to exist by the newer methods. The significance of the increase in the number of free nuclei, the alteration of the basic substance into thickened and fibrous tissue, the appearance of spindle cells, and the proliferation and thickening of the neuroglia have variously impressed the different authors. The older writers regarded such appearances as due to a true inflammatory process beginning in the bloodvessels, the nerve elements being at length implicated. In an elaborate paper upon this subject as long ago as 1880, Binswanger demonstrated that there were varieties of nuclei coming from the different structures of the cerebrum—namely, from the smooth muscles of the vessels, the neuroglia, the ganglion cells, the adventitial sheaths, the pericellular spaces, and also certain free nuclei which are found strewn throughout the cerebrum. Some of these color readily, whilst others do not, the former being generally the smallest. These nuclei, which are more numerous in advanced age, are irregularly distributed in the white and gray substances, varying greatly in different individuals. In addition to these facts, investigations by Lubimow, His, Golgi, Kölliker, Witowski, Gierke, Cajal, and Retzius have only served to create great doubt as to what the so-called basic and connective-tissue substance is, and this uncertainty has naturally reflected itself in the observations concerning the nature of paralytic dementia, as a consequence of which nothing can be more definitely said than that the neuroglia is sometimes atrophied and

sometimes proliferated, whilst the spindle cells in the middle and deeper layers and the cortex are not increased in number except in the later stages.

A diminution in thickness in the gray matter of the cortex, especially in the central convolutions and the frontal brain, partially also in the occipital lobe, has been found by Cionini. It would be inferred, therefore, that a certain amount of cortical substance must have been destroyed, and what has remained has become more compressed, whilst the physiological spaces around cells and bloodvessels have increased.

An active proliferation in the endothelial adventitia of the arterioles, capillaries, and veins, leading to a thickening of these structures and proliferation of the endothelial nuclei, is observed to begin at this time, and may be regarded as a reparative effort. At a later stage a portion of these new formations also undergo a hyaline degeneration. The process of proliferation in some vessels even implicates the true wall of the vessel, the lumen becoming narrowed by these means, even to complete obliteration in the later stages. A new formation of vessels is doubtful, although Bevan Lewis thinks he has seen it. The intra-adventitial lymph spaces are only affected in places, separated from the media, and containing red and white blood cells, coagulated lymph, and amorphous and finely granular pigment. The white blood cells are only occasionally found, however; indeed, their presence in large masses is regarded as an indication of the course of the disease by some authors. Binswanger believes that they are seldom found in the intra-adventitial lymph spaces in large number in slowly progressive cases with paralytic intercurrences, but are present in those with chronic and frequently repeated venous congestions. A generally congested condition of the extra-adventitial lymph space, together with the pericellular space, is common. In the former is also found, though in a less degree, white and red blood cells, fibrinous coagula, and pigment; but in the lymph spaces of the cortical substance itself, as well as in the pericellular spaces, no distinct increase of the lymphoid elements has been observed. As the disease progresses, to these alterations may be added true inflammatory or exudative processes which are demonstrated clinically by somnolence and irritative and paralytic phenomena. Should death then occur, there may be marked infiltration of both the extra- and intra-adventitial lymph systems, although it has been demonstrated, on the other hand, that these inflammatory changes are sometimes absent, in which event the clinical phenomena may be explained by the obstructed flow of the lymph stream produced by the altered cerebral pressure.

Lesions implicating the pia mater play an important part in the later stages of the disease. In the early stages the pia mater is only moderately affected over the convexity of the hemisphere, particularly in isolated places in the frontal lobes, being thickened and proliferated in its endothelial nuclei; but in the course of time the thickening and nuclear proliferation of the endothelial adventitia of the other vessels extend to the similar structures in this membrane; and these hyperplastic processes of the pia are regarded as an extension of the proliferation of the vessel sheath to the endothelial membrane. Partial adhesions of the pia to the connective tissue lead in places to imperfect obliteration of the epicerebral spaces, this being observed in the region of the vessels

going to the cortex, as well as in the portions of thickened pia; and this is due to an increased growth of the pial connective fibres and of the endothelial septa coming from the pia and traversing the epicerebral space. There are thus induced an obstruction in the flow of the lymph stream, and a stasis in the lymph spaces of the connective tissue, as well as those in the deeper portions of the cortex. It is easy to understand that this altered lymph, thus obstructed in its flow, should coagulate cerebral tissue which is already advanced in degeneration. These changes do not seem to differ in kind, whether the case be chronic or relatively acute, except in the rapidity of their progress. A certain number of the violent cases are fatal because of the exhaustion produced by the great motor excitement, the deficient nourishment, the pulmonary or intestinal lesions, etc., rather than because of any intensity in the pathological phenomena of the cerebrum; and, indeed, true inflammatory phenomena are entirely wanting in many of them. These alterations in the tissues especially affect the frontal brain, the convolutions of the island of Reil, and the parietal lobe, and finally extend to the temporal and occipital lobes.

Discussion of the alterations in the cells has not been had in this general description because of the uncertainty regarding them.

The ganglion cells of the cortex, especially in the frontal lobe, often possess two or more nuclei, one of them, however, being probably the nucleus of a wandering leucocyte. The body of the cell is jagged in places, the space thus formed being filled with the nuclei of leucocytes, whilst the pericellular spaces are widened, and contain one or more leucocytes, as well as pale, ovoid nuclei which are coarsely granular. It is uncertain what these large ovoid cells, which have been mistaken for ganglion nuclei, are, although Binswanger suspects them to be of endothelial origin.

Various other cell alterations have been described by different authors. Binswanger states that the nucleoli are often swollen, and contain peculiar white, glittering, irregular lines, producing a cracked appearance; or the place of the nucleoli is taken by two to four irregular lump-like large granules, the body of the cell remaining unaltered. Again, the same author has seen the nucleus enlarged, puffed, cloudy with granules or holding glittering plates, or its contour is irregular or merges with the surrounding cell-protoplasm, whilst in both cases the nucleolus is unaltered. The body of the cell in other cases appears normal, but the nucleus has been replaced by a loose, finely granular mass, and in still other cells the nucleus has disappeared, a brown pigment appearing in its stead. The nucleolus is also destroyed. With these alterations of the nucleus and the nucleolus the body of the cell is irregular, bellied, diminished, darkly pigmented, and contains small nuclei without nucleoli. Associated with the cells thus altered are found others perfectly intact.

The central, the third frontal, and neighboring convolutions, and the island of Reil, are the chief sites of these cellular changes. These observations of Binswanger have been confirmed by Liebmann, Zacher, and Bucelski, but in certain investigations undertaken by Fischl he found that in some hardening methods, notably in Flemming's, Ehrlich's, or Müller's solutions, the nucleoli of the cells would disappear or diminish, the place of the nucleolus would often be taken by pigment, and its shape

would be altered, and the nuclei would lose its sharp outlines, whilst pigmentary collections would almost constantly be found in the ganglions. Nevertheless, as pathological he regarded pigmentary degeneration with fattiness and entire destruction of the ganglion cells, and nuclear and nucleolar alterations found in normal brains are seen more widely distributed and implicating a larger number of cells in pathological instances. Fischl was never able to obtain the hyaline degeneration of the cell-body (spoken of by Liebmann and Lubimow) in alcohol preparations. That the cortical cells diminished in number in the early stages and in the galloping cases has been observed by Binswanger, although this was not very marked, and the cells in the frontal lobe and the central convolutions were often only affected singly or in individual groups. From some very interesting observations upon the early alterations in the ganglion cells of the anterior horn Friedmann has concluded that the death of the cell is effected in several ways: (1) By a homogeneous swelling of the cell, which is really a hyaline transformation of it, commencing in the centre, whilst a small peripheral portion of the cell may remain intact for some time, containing the chromatic substance, and even the nucleus, which latter disappears late, but yet before the nucleolus. (2) By a granular destruction or fatty degeneration. In this the diseased portions are transformed into a pale, granular mass as the chromophile substance disappears. The first step in this type is the granulation, and not the fattiness. Types 1 and 2 are often combined. (3) By sclerotic degeneration, which is less frequent than the other forms in the acute inflammatory period, whilst it is more often found in the chronic cases. (4) By the transparent light cell. This is a very rare form, characterized by the destruction of all but a small portion of the chromatic substance, the nucleus often remaining intact. This author does not believe in the existence of simple atrophy or shrinking of the cell, or in the so-called cloudy swelling; the first being, according to him, only the result of the homogeneous degeneration, and the latter a combination of swelling with the stage of the molecular destruction. These observations have not yet been confirmed, however. They have led to a great doubt as to whether the active inflammatory changes in the ganglion cells described by others really occurred, inasmuch as they have probably been due to defective hardening methods or faulty observation.

From these descriptions it can be seen that the theory of paralytic dementia being due to an interstitial encephalitis, as was maintained by the older authors, is not warrantable. Nor is there any confirmation of the views of Bevan Lewis, who lays great stress upon the rôle played by the connective tissue; for he would have us regard the connective-tissue (or spider or Deiters') cells as playing the part of depurative agents, being, as he expresses it, true scavenger cells. He maintains that these bodies are auxiliary to the capillaries—that they feed upon the degenerated nerve cells and fibres, both of which are converted into granules or oily droplets; and he even presents many alterations of the condition of these scavenger cells at different stages of the degeneration of the cortex, figuring them laden with the products of the disintegration of nerve cells and nerve fibres, and actually at times drawing upon the perivascular sheath with such force as to distort it. Nor, in view of these later investigations, need we attach more

than a relative importance to certain gross changes which have been observed in the brains of paralytic dements. Thus, meningeal hemorrhage may occur on the outer surface of the arachnoid into the subdural spaces, and it may be a mere streak of fluid staining, a coagulation, or a rust-stained pellicle that may be peeled off, forming one of the varieties of the so-called arachnoid cysts. Fuscous degeneration of the cortical nerve cells may sometimes occur. Spitzka has described a cystic degeneration of the cortex consisting of minute cavities, varying in size from a pin-point to a millet-seed, found either in the gray matter or in both the gray and white.

CLINICAL HISTORY.—There are undoubtedly included under the name of paralytic dementia many diseases which the future will differentiate, but at present a very general consensus of opinion prevails as to what we shall call by this name. In considering the pathology we shall ascertain that paralytic dementia consists of molecular alterations affecting the cerebrum as a mass, and this should make it clear to us at the outset that the symptoms, both mental and physical, are almost coextensive with the cerebral functions, so that there is scarcely one symptom to be found in other diseases of the brain which may not be present in this, differing only in the manner of grouping. The stages may be divided into the prodromal, maniacal or melancholic, and the demented.

The COURSE of the disease varies, however, and only the first and second stages may be present in some instances.

The Prodromal Stage.—Both the mental and physical symptoms of this stage are remittent, insidious, and may be protracted.

Those relating to the mental condition consist of vague and transitory alterations of character or of intensifications of pre-existing mental traits, but the essential feature is an element of stupidity. Eccentricities of behavior are at first observed in the patient, and he is found performing causeless and unexpected acts which greatly puzzle his friends or associates. For example, a patient of mine who had been newly married brought a rose home to his wife, gave it to her affectionately, seated himself before his mirror to brush his hair, and then suddenly arose, slapped her face, and nonchalantly went about his dressing as though nothing had occurred, without a word of explanation, seemingly unaware that he had done anything out of the way. In another instance a cautious business-man engaged recklessly in speculation. Another, who had hitherto been of a very jovial and kind disposition, suddenly and without cause became very suspicious of his family. On the other hand, a generous man becomes extravagant, a naturally frugal man becomes penurious, a talkative individual is converted into a loquacious one, and one who is reticent becomes still more silent; and thus we witness a deepening of all the innumerable shades of character. At the same time—often, indeed, at the very first—there is a change in the finer ethical sentiments which lie in most civilized human characters like the down upon the peach, indicative of the highest culture, and varying in each individual with the sex, age, race, social station, and nationality; so that a kind husband or father becomes brutal or seeks the society of loose women, or a chaste and modest woman gives rise to remark, or an affectionate son or daughter becomes disobedient, disrespectful, or thoughtless.

Physical alterations are observed contemporaneously with these mental ones. Tremor and speech and pupillary alterations are among the earliest of these, though they are often so slight as to escape the trained observer. The tremor generally affects the facial muscles and the tongue, sometimes also the extremities. In the former the tremor is best seen by causing the patient to speak, when the facial muscles of the lower part of the face, especially at the angles of the mouth, will twitch with slight irregular contractions that are intermingled with tremor. When the tongue is protruded slowly the lingual muscles are seen to be agitated by a series of slight fibrillary movements. The speech defects are made evident either by a slurring and indistinctness in the pronunciation of certain words or consonants, particularly the labials, or by a combination of these two peculiarities. Frequently, however, a patient will slur over words in the context of a sentence or in ordinary conversation when he can pronounce them separately with distinctness. The letter *r* is apt to present especial difficulties, particularly to a German or a Frenchman, with whom the *r* is either guttural or prolonged. A good test of this is to get the patient to pronounce such a sentence as "riding cavalry brigade," or, better still, the same words in German, "reitende Cavallerie-Brigade," with the full German guttural intonation, or the sentence, "Round about the rough and rugged rocks the ragged rascal ran." The tremor in the extremities when slight may be best detected in the hand by causing the patient to extend the arm at full length at right angles from the body, and then lightly supporting the fingers upon the palm of the examiner's hand, when a delicate muscular tremor will be made evident, like the throbbing of a small engine. In the lower extremity tremor may be detected by having the patient sit down and extend the lower extremity at right angles from the body, and then lightly supporting the heel with the examiner's palm, when the foot and toes will be seen trembling with fibrillary tremor that is usually coarser and more distinct than is observed in the fingers. The pupillary alterations are to be noted in the size of the pupils, the regularity of their contour, and the reaction of the iris to light, to consensual or cutaneous stimulation, and to accommodation. In about one half of my cases the pupils have been dilated, my observations thus agreeing with those of Bevan Lewis; and, whilst the moderate-sized pupil is next in frequency, the contracted pupil is rare. One pupil was larger than the other in about one half of my patients, which also confirmed the experience of Bevan Lewis, but whilst he found the right pupil was generally the larger, this has not been a constant observation in my experience, as in the same case sometimes the one pupil and sometimes the other would be more enlarged. This pupillary inequality may be temporary, lasting only a few days or weeks at a time, and is therefore apt to escape observation. The pupil may be perfectly immobile or very slowly reactive when tested by a strong light and focal illumination of the eye by a convex lens, and in some cases this sluggish reaction may be followed by wide dilatation; or the accommodative movements of the eye may be attended by no pupillary response or only a sluggish one, while the want of the normal reaction may be likewise observed upon stimulating the skin by the electric brush, by pinching, or by pricking. It is usually the smaller pupil which fails to react consen-

sually or with the light reflex, and the consensual movements generally occur when there is impairment of the light reflex, although the light reflex may be impaired whilst the consensual reflex persists. In this prodromal stage the knee jerk is abnormal in a majority of cases, being sometimes exaggerated, sometimes slight, sluggish, or absent. Transient urinary incontinence is also an occasional symptom of the prodromal stage.

Whilst all the symptoms that have been enumerated are more or less constant, varying slightly in intensity perhaps, there are apt to be paroxysmal exacerbations, when the mental defects, the tremor, the speech alterations, and the pupillary abnormalities are increased, and the face, and less frequently the extremities, become congested or pale. These symptoms increase in frequency and intensity as time goes on, and the ataxia which has constituted the speech defects invades the upper extremities as well. This extension is readily detected by getting the patient to write his name, or by having him close his eyes, swing his arm from his side, and quickly touch the end of his nose with the tip of his index finger. At this time the muscular strength is usually unimpaired, as the strength of the facial muscles or the grasp of the hand will demonstrate even in this prodromal stage, but some few cases will evince the paresis in certain of the muscles of the face. For example, there may be detected a loss of power in one eyebrow or one side of the brow, or even an imperfect action of one upper eyelid, or a flattening or sluggish movement of the muscles making the naso-labial fold, or a slight deviation of the tongue.

From a diagnostic as well as a medico-legal standpoint the prodromal symptoms are of vast importance, for it is by means of them that an early diagnosis can be made, and thereby much misfortune to the patient, his relatives, and his business interests averted. Gradually or suddenly patients in this condition pass into the second stage.

In the maniacal or melancholic stage the patient has superadded to the other symptoms a mania or a melancholia, each with certain peculiarities distinguishing it from the corresponding phase of other forms of insanity. The delusions and the hallucinations in the mania of paralytic dementia are almost invariably pervaded by a certain stupidity, so that the former are illogical; and with this stupidity is mingled an expansiveness or exaggeration of idea giving rise to the so-called delirium of grandeur. For example, the patient may say that he owns all the railroads in the United States, or all the hotels, that he is the wealthiest man in the world, that he will make his physician a present of a million, and so ramble on in this extravagant strain; but when asked how it is possible that he can be worth all this when his circumstances are known to be far inferior to what he claims them to be, he reiterates his assertion without attempting to reason about it. In this respect he affords a marked contrast to the paranoiac, who may have the same grand conceptions, but will adroitly lend them plausibility by any number of settled and fine-spun reasons. It is this tinge of stupidity or dementia coloring the delusions and hallucinations that is so especially characteristic of the paralytic dement and gives one of the names to the disease. The mania usually becomes violent, and in occasional instances may lead to a fatal termination by exhaustion. While obstinate insomnia

and post-cervical ache do not accompany the melancholia of the paralytic dement, there are often suicidal tendencies, and the delusions and hallucinations are the depressed ones characteristic of ordinary melancholia. Illusions have been observed in paretic dements in some few instances, but their occurrence is very difficult to determine in such cases. Paroxysms of terror, coming on frequently without external cause, are characteristic in both the melancholia and the mania.

As has been already said, this second stage of mania or melancholia may be wanting in certain cases in which there is a simple dementia throughout, but if present it usually merges into the third stage of dementia.

Although a certain element of stupidity prevails throughout all three stages of the disease, this stage of dementia proper is characterized by real lack of mental faculty to a greater or less degree, so that the patient becomes stupid or childish, talking and acting foolishly, at the same time that he may be difficult to restrain temporarily.

Epileptiform or apoplectiform attacks may occur throughout all the stages of paralytic dementia—*i. e.* there may appear at any time slight loss of consciousness, with or without convulsions, localized or generalized, or losses of consciousness varying from mere syncope to prolonged coma, with paralysis that may be hemiplegic or monoplegic in distribution or affecting speech. Paresis affecting at first the lower extremities usually begins to be apparent toward the end of the first stage or during the second, showing itself at first only in the paroxysmal exacerbations, but gradually increasing in degree until eventually it may result in complete paralysis of both the upper and lower extremities. Occasionally, however, the paretic symptoms are among the earliest, and it is by no means rare to find an initial tremor, pupillary alterations, and defects in speech accompanied by a paresis of one side of the face and tongue, or to note that the patient, even at this early stage, has a distinct paresis superadded to his ataxia.

An important aid to diagnosis may at times be found in the peculiarity of certain symptoms, such as affection of sight, smell, hearing, tactile, pain, muscular sense, and temperature, which should, therefore, be considered in detail.

That the sense of smell is always defective in the initial stage of general paresis was claimed by Voisin, but in this he has not been borne out by the later writers, and my experience leads me to state unqualifiedly that he is mistaken, although I have occasionally seen it affected in cases of cerebral syphilis simulating general paresis. Most writers are agreed in stating that it is certainly impaired in the later stages, but I regard this to be rather due to the mental impairment than to any organic lesion of the olfactory apparatus itself.

A peculiar variety of mental blindness, first described by Fürstner, is sometimes observed. Usually the defect is in one eye, especially the right. The patient can name and recognize single letters, but when combined in a word they are not recognized, and it is the same with simple objects. The ophthalmoscopic appearances are usually of little diagnostic value, as comparatively few cases furnish any lesion of the eye, but when present it is generally of the nature of a primary optic-nerve atrophy. Even this condition is not usually observed until the

disease is well established, and therefore has but slight value in the question of differential diagnosis.

Word-deafness is observed at times, but at the onset hearing is occasionally over-acute.

In the first stage of general paresis the sense of pain is usually acute, and hyperæsthesia and neuralgia are apt to occur. I believe Sander is right when he calls attention to the fact that an onset of migraine without a precedent history, either personal or hereditary, in an individual in the fourth decade of life is of great diagnostic value. When the disease is well marked anæsthesia is a usual accompaniment of general paresis, and it may occur even in the early stage. In six cases reported by Mendel in which there was marked anæsthesia there was also a spinal lesion; but the former has occurred in patients of mine in which there were no spinal symptoms whatsoever, and it is not necessary that there should be such. This anæsthesia in the latter stages may become very complete. Mickle relates an instance of a patient chewing his right forefinger as if it had been a piece of tough food, so that it became gangrenous and was amputated without any general or local anæsthesia, the patient gazing stupidly at the operation without the least concern or evidence of pain. A similar case of amputation is recorded by Baillarger, and Lines tells how a patient seized a live coal and kept it in his hand long enough to produce a severe burn. I have seen anæsthetic phenomena almost similar to this in several instances, and in one case that had just passed out of the initial stage into an attack of mania the anæsthesia was so complete that a large jagged wound caused by the patient thrusting his hand through a plate-glass window was dressed without the slightest sensibility being manifested. It is always well to remember this tendency to anæsthesia when general paretics are made to bathe in warm weather, as it is often the explanation of the so-called parboiling accidents occurring in asylums which create so great a furor in the columns of sensational newspapers. In cases complicated with tabes fulgurant and stabbing pains may be observed.

Diagnosis.—It should be borne in mind that general paresis is in reality a gradual dementia, complicated by stupid delusions, often expansive in nature, by motor symptoms that are ataxic in the initial stage and paralytic in the later, by pupillary alterations, by general tremor that causes a peculiar pronunciation, and, in many instances, by the supervention of a mania or melancholia upon the initial symptoms. It therefore is really a chronic dementia of a peculiar type, with physical symptoms. The pupillary changes, the tremor of the tongue and facial muscles, and the peculiar pronunciation, associated with mental alteration that is stupidly eccentric and vague, the tendency to paroxysmal exacerbations, lasting several days or longer, during which time all the symptoms are exaggerated, and flushing or pallor is superadded,—all these make a picture in the early stage that is seen only in this disease, although many others resemble it to a certain extent. When to these symptoms are joined delusions of grandeur, the diagnosis becomes very easy; but even when the dementia has not been interrupted by any markedly expansive delusions the consideration of the foregoing facts is amply sufficient for a diagnosis. When a melancholia or mania of sudden or gradual onset is added to these symptoms of the initial stage, the diag-

nosis is still more easy, and one should be careful to obtain in every case of mania or melancholia the precedent history.

It is necessary to differentiate general paresis from cerebral syphilis, melancholia or mania, primary dementia, secondary dementia, katatonia, paranoia, disseminated sclerosis, paralysis agitans, lepto-meningitis cerebri, alcoholism, bromism.

Although I am aware that I hold my opinion alone against most authors, I feel quite positive that cerebral syphilis can be diagnosed in most cases by means of the symptoms to which I have called attention —namely, quasi-periodical headache and insomnia, both headacke and insomnia ceasing upon the supervention of any paralytic or convulsive symptoms. When a cerebral disease characterized by these symptoms, preceded or not by a history of specific infection, passes into dementia resembling general paresis, in the larger number of instances a fairly positive diagnosis can be made. In some, however, in which the history of these initial symptoms is absent, the diagnosis is impossible.

As has been already emphasized, every case of mania or melancholia should have the previous history carefully reviewed, so that the melancholiac or maniacal stage of a general paresis may not be mistaken for an initial mania or melancholia.

Primary dementia occurs in individuals who are younger than general paretics, and the character of the dementia is entirely different, the patient acting in a silly manner and talking foolishly and incoherently, while the onset of dementia is sudden in comparison with the very gradual and insidious onset of the first stage of general paresis. It must also be remembered that primary dementia comes on fully developed, whilst the dementia of general paresis is only manifested vaguely in the earlier stage. The ataxia, the pupillary alterations, the apoplectiform and epileptiform attacks are absent in primary dementia.

Secondary dementias are invariably subsequent and terminal conditions of different forms of insanity, as implied by the term; but the history should make it evident in every instance that the previous insanity has not been of the nature of general paresis.

A case of katatonia may at times be mistaken for general paresis, but a careful history and examination of the patient should remove all difficulties of differential diagnosis. The disease is a cyclical one, interrupted by epileptiform, choreiform, and cataleptoid attacks as it passes through its various mental phases, and is characterized by verbigeration. General paresis is, on the other hand, a chronic dementia of a remittent and insidious type, occasionally varied by demented agitation or demented depression without verbigeration, sometimes with epileptiform and apoplectiform attacks, but seldom or never with cataleptoid symptoms. The reflex pupillary alterations and inequality are wanting in katatonia, as are also the tremor, the ataxia, the peculiar speech, and the eventual paresis.

The expansive delusions of some paranoiacs may be mistaken for general paresis, but the resemblance between the two diseases is too slight to subject to a prolonged error of judgment. On the part of the former the delusions are always reasoning and logical within certain limits, while those of the general paretic are illogical and stupid. Occasional tremor of tongue or facial muscles may accompany paranoia, but the patient is abnormally keen and acute, instead of evincing the de-

mentia of the general paretic; and the ataxia, the characteristic pupillary alterations, the tremor, the tremulous speech, the apoplectiform or epileptiform attacks, and the terminal paresis are lacking in him.

It is not always easy to differentiate paralytic dementia from disseminated sclerosis, and from a meningitis of the pia mater the diagnosis is sometimes equally difficult. The mental symptoms in disseminated sclerosis are not usually so pronounced at the beginning. There is not the same degree of ataxia; there are more apt to be nystagmus and the pupillary alterations characterizing general paresis; and the tremor precedes the mental symptoms by a period of months or years. In disseminated sclerosis the tendency to paroxysmal exacerbations which is seen in most cases of general paresis is absent, whilst the intention type of tremor observed in the early stage of disseminated sclerosis is rare in general paresis.

From paralysis agitans the diagnosis is easy because of the peculiar bent attitude of the patients, the slow, deliberate speech, instead of the stuttering, scanning enunciation of the general paretic; by the lack of mental symptoms, except some dulness in the later stage; by the lack of marked pupillary alterations except those belonging to old age; and by the absence of any paroxysmal exacerbations, although it should be always remembered that in the aged apoplectiform attacks may occur at any time.

Alcoholism at first sight may bear a striking resemblance to general paresis, especially in those cases where the pathological findings consist of opacities of the pia mater and congestion of the cerebral substance. The diagnosis can be made by means of the alcoholic history, the relatively acute onset of the symptoms, the tendency of the delusions to be more keen and logical than those of general paresis, and to partake of the character of suspicion, especially of marital infidelity. The fact that prolonged alcoholism will of itself produce a genuine general paresis must not be lost sight of, however, and that these cases which we are now considering are those of acute alcoholism.

The acne, the fetid breath, the excessive stupidity without delusions characteristic of bromism, together with the history, should be sufficient to indicate its difference from so serious a malady as paretic dementia. Bromide intoxication should be suspected by the lack of other pupillary alterations than dilatation, by the lack of tremor and ataxia, by thick, muffled speech rather than a tremulously scanning one, and by the early onset of general weakness that may simulate paresis.

Prognosis.—In general paresis the prognosis is an exceedingly serious one, although competent observers, such as Minot, have recorded cases of cure. It should always be borne in mind that remissions of considerable duration may occur in any case. These I have witnessed, but never a permanent cure.

Treatment.—In any case of general paresis the first point to determine is whether there has been a history of cerebral syphilis, and then, if it is possible, to cause improvement of the symptoms by large and increasing doses of the iodide, either alone or in combination with mercury. Should there be no cerebral syphilis, the use of either iodide or mercury will be of slight value, simply diminishing the tendency to convulsive and apoplectiform attacks and to agitation; but for the latter effect the

dose need not exceed fifteen or twenty grains three times a day. Large doses of ergot have sometimes seemed to me to have a favorable effect. Commencing with half a drachm of the fluid extract three times a day, the doses may be increased to half an ounce if the patient's stomach will endure such a quantity. Sulphate of quinine, five to ten grains at bedtime occasionally, with twenty grains of bromide of potash, has also seemed to me to be of considerable use. The attack of mania can be quieted by means of warm baths, by hyoscyamine, by the bromides, and by tincture of veratrum viride. In using warm baths the patient's sensations should never be relied upon; the temperature should not exceed 100° or 105° F., as there may possibly be the anæsthesia which has been spoken of, while carelessness may easily lead to vesication. The crystallized form of hyoscyamine should be used—Merck's preparation being the best in my opinion—and the doses should vary from $\frac{1}{300}$ to $\frac{1}{100}$ grain once or twice a day. The large doses, $\frac{1}{50}$ to $\frac{1}{25}$ grain, that are sometimes recommended, have not gained my approval. The sedative effect of hyoscyamine will be increased without adding to its depressing influence by combining each dose with ten or fifteen grains of the bromide of potassium. The tincture of veratrum viride is sometimes of excellent use in quieting maniacal excitation in doses of five to ten drops, but it should always be carefully watched, and should not be given patients with a cardiac lesion. No form of electricity has proven of the smallest value to me in the treatment of cases of general paresis.

IDIOCY AND IMBECILITY.

By PEARCE BAILEY, M. D.

Idiocy is not in itself an entity. It includes clinical conditions which arise from many causes, and which depend upon anomalies of cerebral structure of diverse character. No definition, therefore, can satisfactorily describe the various manifestations of the infirmity, together with their causes and their morbid anatomy.

The study of idiocy is a study of the pathology of the infantile brain rather than that of any individual disease. It has to do with mental deficiencies so pronounced that the life of the individual is purely vegetative, as well as with those which are so inconspicuous that they only become evident upon close observation. It includes also any and all of the multifarious causes and lesions which interfere with the development of the encephalon. In spite, however, of the heterogeneous character of the pathology, causes, and symptoms of idiocy, the clinical forms have certain characteristics in common, and it has been found more convenient to describe the various conditions as a whole, rather than to consider them separately.

In the following pages the term idiocy will be used as a generic designation for the intellectual deficiencies which occur in infancy or childhood as the results of congenital or acquired cerebral lesions. The slighter degrees of mental incapacity will be called feeble-mindedness, and the degrees between it and profound idiocy, imbecility.

Etiology.—The causation of idiocy is concerned with the processes which interfere with the normal development of the brain. The chief anatomical evidence at present available for the determination of the time required for the gross development of the cerebrum, and to a certain extent, consequently, of intellectual capacity, is derived from the weight of the brains of persons at different ages. From a table arranged by Vierordt,[1] which is based upon the examination of 417 cases, it appears that the most rapid accessions in brain weight occur during the first two years of life. Thus the mean brain-weight is, at birth, 381 grammes, but at the end of the first year of life it has reached 945 grammes; at the end of the second year it is 1025 grammes, or nearly three times its weight at birth. The increase in succeeding years is less rapid, but, with slight variations, is constant until the fifteenth year, when the maximum is attained. These facts have an important bearing on idiocy, and furnish a means of differentiating that condition from dementia, since dementia consists in loss of intellectual power due to lesions affecting the developed brain. As Voisin[2] has aptly put it, "The idiot is poor, the dement is bankrupt." Inasmuch as the brain has attained

[1] *Arch. für Anat. u. Phys.*, 1890.

[2] *L'Idiotie*, Paris, 1893.

its greatest growth, as far as can be determined by weight, at the fifteenth year, mental enfeeblement occurring after that age should be called dementia, and not idiocy.

By far the larger number of idiots are born with brains which are either already imperfectly developed, or which receive the check to development in the first few years of life. Over 60 per cent. are congenital. Of 286 acquired cases quoted by Piper,[1] in 216, or 75 per cent., the defects in intellectual calibre became apparent in the first four years of life. In the first and second years it was 30 per cent. and 24 per cent. respectively. It is very unusual for idiocy to appear after the seventh year.

Boys are affected twice as frequently as girls. In cases due to difficult labor this proportion increases to three to one—a fact which is explained by the relatively larger size of the male child.

Determining Causes.—The determining causes of idiocy are divided into three classes :

1. Those occurring before birth ;
2. Those occurring at birth ;
3. Those occurring in infancy and childhood.

1. *Prenatal Causes.*—As has been said, more than one half of all cases of idiocy are congenital ; it is in them that the agency of heredity is particularly conspicuous. The transmission from parent to offspring of the tendency to defective or abnormal development follows such peculiar and seemingly irregular laws that a thorough history of a family is often necessary for the demonstration of morbid hereditary factors. Such a history being frequently unobtainable, we cannot always prove in idiocy, any more than in other degenerative conditions, the morbid heredity to which all analogy would point as a cause. In a certain proportion of cases,[2] however, this factor is demonstrable. It exists as an organic proclivity in persons whose nervous systems are exhausted, diseased or defective, to transmit to their descendants nervous systems which, in their turn, are either incapable of proper development or which are abnormally liable to succumb to the action of disease-inducing agents. It is the manifestation of a deterioration of the vital forces. Under the name of degeneration, it has, especially in recent years, been the subject of much discussion and speculation. To discuss degeneration in its relations to morbid heredity would be far beyond the limits of the present article. Nevertheless, mention of a few of the well-established facts in regard to it is essential for an understanding of the causation of idiocy.

The causes of degeneration are numerous and varied. Anything which exhausts or invalidates the nervous system of an individual renders him prone to transmit to his offspring an imperfect physical legacy. The diseases which are especially undesirable testators are insanity, hysteria, epilepsy, or any chronic degenerative disease of the nervous system ; syphilis, tuberculosis, gout, and rheumatism also figure in this class. The nervous system need not be, however, actively diseased. Conditions of exhaustion, such as neurasthenia, or of chronic intoxica-

[1] *Zur Aetiologie der Idiotie*, Berlin, 1893.

[2] See Shuttleworth and Beach : "Idiocy and Imbecility," *Tuke's Dictionary of Psychological Medicine*, Philada., 1892.

tion, such as alcoholism, are of themselves sufficient to endanger either perfect development or the acquisition of normal resisting force in the nervous systems of descendants. Similar results may occur from consanguineous marriages, especially if the family history is not perfect. Senility of the father, temporary exhaustion, intoxication, or perverted mental states in either parent at the time of conception, must also be regarded as causes. "Young man," said Diogenes to a feeble-minded boy, "thy father must have been very drunk when thy mother conceived thee."[1]

Finally, physical or psychical evidences of degeneration may appear in children whose forbears presented no more pronounced degenerative evidences than eccentricity or criminal tendencies. Although these causes are most prominent when on the maternal side, they act in various ways. The hereditary taint may come from one or both parents or from one or both grandparents. It may affect only one of a family of children, leaving the others apparently normal or at most eccentric.

Morbid nervous heredity is not necessarily direct. It is an unstable or imperfect nervous system, rather than any individual disease, which is transmitted, and the clinical manifestations of such instability or imperfections depend largely upon the environment of the individual. Thus the child of an alcoholic father may be epileptic, or the child of an epileptic may be insane or idiotic without ever giving any evidences of epilepsy.

Degeneration is progressive. The larger the number of degenerate members in a family the greater is the likelihood of more widespread and more pronounced deterioration in the offspring of its individual members.

Idiocy is the last stage of degeneration, and eventually results in extinction of race.

Among other prenatal causes of idiocy are to be reckoned any maternal disease or any accident of pregnancy by which the nutrition, and consequently the development, of the embryo may be impaired. In this category, consequently, fall infections (notably those of tuberculosis and syphilis), fright, anxiety, and similar psychic influences which prove injurious to the mother, and traumatisms, which may injure either mother or child.

2. *Parturitional Causes.*—The factors which are active at the time of delivery in the causation of idiocy are mainly of traumatic character, and are such as may bring about direct injury of the infant brain or its membranes, or such as may obstruct the circulation for too long a time. Thus prolonged parturition by causing protracted pressure on the ununited bones of the skull may seriously compress the brain or may induce asphyxia. Compression of the head by forceps figures in a certain proportion of cases. Premature birth can only be regarded as a cause when it can be shown that it and the idiocy are not both results of a common underlying condition.

3. *Causes Acting After Birth.*—The most important of these, and the ones which are susceptible of the most reasonable proof, are diseases of the brain and its membranes. Epilepsy, infantile convulsions, and meningitis are especially prominent in this connection. Hemorrhage, particularly from the meninges, is probably the most fertile cause of

[1] Voisin: *Op. cit.*

paralytic idiocy. To the infective fevers, especially scarlatina and enteric fever, a causal influence is frequently traceable. In rare cases the mental deterioration appears to be the immediate consequence of a fall or a blow on the head (traumatic idiocy). Proof of the other factors, such as fright, overwork at school, etc., which are not infrequently cited as causes of idiocy, is generally difficult to establish. These agencies are often advanced by the family of the patient in explanation of the mental condition, but, inasmuch as idiocy is so frequently congenital, and since it may exist for some time unsuspected, these and similar indefinite causes are only to be accepted with caution.

Pathology.—Idiocy depends upon developmental defects of the encephalon. While it is generally true that the more imperfect the mental power, the more probable it is that the brain defects are serious and conspicuous, and that gross encephalic deficiencies prevent the elaboration of the intellect, the truth of these statements is not universal. A brain which to the unaided eye presents only trifling variations from the normal may never have become the seat of the higher intellectual faculties; and, conversely, a congenital absence of cerebral tissue may be found after death in persons who during life were not remarked as lacking in mental capacity. Although brain defects vary with their causes, we are unable to describe the pathology of idiocy according to causation, because the inhibiting influence is most frequently exerted during intrauterine life, and defies our attempts to accurately determine either its character or the time of its action. Accordingly, the most that can be done here, in speaking of pathological anatomy, is to mention the more important varieties of brain defects found after death in persons who presented during life the symptoms of intellectual retardation.

Such defects exist in varying degrees. They may be associated with failure of development of the bones and membranes, and, when complete, constitute the condition known as anencephaly; when partial, encephalocele or encephalo-meningocele. In such cases the infants are either born dead or survive their birth but a short time. Or the failure in development may be limited chiefly to the cortex, while the basal ganglia are normal in size. The cortical anomalies consist principally in rudimentary development of the convolutions, shown by imperfect markings or by an aberrant development (microgyria), or one hemisphere or certain lobes only may be rudimentary. Certain tracts and certain of the basal ganglia, notably the corpus callosum, the corpora albicantia, and the optic thalami, may be totally deficient. Bourneville[1] has found bilateral absence of the lobes of the cerebellum.

Sachs[2] calls attention to the fact that a congenital absence of the cranial nerve nuclei, causing oculo-motor and facial paralysis, is not unusual.

Some of the above-mentioned anomalies are the results of agenesis, others are due to circulatory disturbances. Meningeal hemorrhage causes extensive destruction, and, when its immediate results have disappeared, leaves cysts and deficiencies in the cerebral structure.

In a small proportion of cases tumors, especially solitary tubercles,

[1] *Récherches sur l'Épilepsie et l'Idiotie*, Paris.

[2] *A Treatise on the Nervous Diseases of Children*, N. Y., 1895, p. 603.

are found after death. In all cases adherence of the membranes to the skull or to each other are generally met with.

Porencephaly.—A condition originally described by Heschl,[1] consisting in a funnel-shaped cavity in the brain, whose base, directed toward the surface, is covered by the dura and pia, and whose apex goes inward, where it may or may not communicate with the lateral ventricles. The most frequent situation of porencephaly is in the inferior portions of the precentral and post-central convolutions, in the adjacent part of the frontal convolution, and in the island of Reil. It may be bilateral, although it is more frequently confined to one side. The convolutions surrounding the *porus*, or hole, are usually irregular or smaller than normal, and, as a rule, there are associated anomalies in other parts of the brain.

The causes of this condition are obscure. Kundrat[2] refers it to an anæmic necrosis resulting from disturbances of fœtal circulation, while in an elaborate and recent study Von Kahlden[3] explains it by agenesis. The microscope may some day throw more light on this question.

Contrasted with these more or less circumscribed defects are those which affect the brain in its entirety. They may be most conveniently considered as hypertrophy of the brain, microcephalia, and internal or chronic hydrocephalus.

Hypertrophy of the brain is a rare condition, and is supposed to be congenital. It is due to an increase in the neuroglial elements in the cortex and in the white matter. This hypertrophy may cause a great increase in size. Ziegler[4] reports a case of a girl of 20 years whose brain weighed 1857 grammes. The enlargement of the encephalon may interfere with the closure of the fontanelles, or, if the bony union takes place, the cranial vault may be thinned from the effects of pressure. Consequently, hypertrophy of the brain may be easily mistaken clinically for hydrocephalus.

Microcephalia.—In its broadest meaning microcephalia is a condition in which the brain fails to attain its full development either in weight or size, and is consequently one of the most frequent brain anomalies of idiocy. In its more restricted and usual meaning the term applies only to brains which are uniformly small, without presenting localized lesion. As such, microcephalia is the result of a generalized arrest of development and is not common.

When it occurs it may be so marked that many of the fissures are rudimentary or are absent, and that the whole brain is less than half its normal weight; or the gross defects may be apparent in one or two convolutions only, although the encephalon as a whole is somewhat smaller and lighter than usual. The cranial capacity is also diminished. This condition is probably not induced by a premature union of the bones of the skull, but by pathological causes which act on the brain itself. It is frequently associated with internal hydrocephalus.

Hydrocephalus internus may complicate any of the anomalies already described, or it may exist as a condition *sui generis*. It consists in an abnormal increase of secretion into the ventricles of the brain of a fluid

[1] *Gehirndefect und Hydrocephalus. Vierteljhs. für d. prak. Heilkund*, 1859, Bd. 61, p. 61.
[2] *Die Porencephalie*, Graz, 1882.
[3] *Ziegler's Beiträge*, 1895, xviii. p. 231.
[4] *Lehrbuch der Path. Anat.*

closely allied in chemical constitution with the normal cerebro-spinal fluid. As a result the ventricular cavities of the brain, with the exception of the fourth, are dilated, and the brain substance is compressed and flattened. In extreme cases the cranial cavity is almost wholly occupied by fluid, and the brain is reduced to a mere shell. The immediate cause is attributable to some disturbances in the ependyma.

In the *microscopical anatomy* of idiocy are naturally to be sought the explanations of the psychical conditions, yet contributions to this branch of the subject are conspicuously few. Most of our knowledge is founded upon the masterly work of Hammarberg.[1] From the study of a series of carefully chosen cases, controlled by sections from normal brains, Hammarberg concluded that "the psychic defects of idiocy depend upon an absence of functionally capable cortical cells—an absence which is caused by the greater part of the cerebral cortex remaining in the developmental stage corresponding to that of the normal of embryonal life or of early infancy, and as a result of which only a small number of cells become developed during the growth of the cortex." This is, of course, nothing more than one would be led to expect from analogy, but by means of the microscope Hammarberg was able to demonstrate conspicuous cellular deficiencies in brains whose gross appearances were normal.

Further into the pathology of idiocy it is needless to enter here. The conditions of the cranium, as regards size and shape, and the various deformities of the body, will be considered on a later page. The anomalies of the spinal cord and peripheral nerves have never been investigated with minute care. Certain portions of the spinal cord may be defective, or the cerebral lesions may cause descending degenerations. The viscera are frequent seats of tubercular and degenerative lesions.

Symptoms.—The symptoms of idiocy, inasmuch as they are the clinical expressions of defective cerebral development, are modified by the character, extent, and time of appearance of their causes. Gross lesions which affect large areas of the brain cause not only profound idiocy, but conspicuous physical deformities as well. Hydrocephalus, microcephalia, paralytic and myxœdematous idiocy have individual and striking peculiarities. The time of the arrest of brain-growth, although difficult to ascertain, exerts an important influence upon the degree of the intellectual enfeeblement. It affects prognosis from the point of view of education and of vitality, and it affects diagnosis by assisting in the discovery of the cause.

The beginning of congenital or early acquired idiocy is, however, usually difficult to determine. From the time of its birth it may be observed that the child does not take the breast properly or does not laugh. A mother who has borne normal children may notice that the last one is different from the others, but it is usually six months or more before she becomes alarmed at her child's indifference to the ordinary diversions of the nursery. Indeed, the child often is not brought for medical examination until he has passed the walking age without learning to use his legs, or has reached his second or third year without

[1] *Studien über Klinik und Path. der Idiotie*, von Dr. Carl Hammarberg; *Herausgegeben* von Prof. Dr. S. S. Henschen, Upsala, 1895.

acquiring any words. Examination then reveals the true condition. In acquired cases, on the other hand, in which intellectual progress, previously normal, ceases after some well-recognized causes, such as fever or convulsions, the symptoms, by contrast, are usually apparent at once.

Although the symptoms of idiocy which are physical and mental differ widely in character, degree, and association, it is most convenient to describe them together, reserving for a summary the characteristics of the more common clinical types.

Physical Symptoms.—Cranial anomalies are the most constant of the physical symptoms of idiocy. The skull is too large or too small, is asymmetrical on one side only, or presents general inequalities in its measurements.

The following measurements, compiled by Peterson from the examinations of 500 new-born children at the Sloan Maternity Hospital, may be accepted as representing the normal average for the infant skull:

Occipito-frontal diameter	11.13
Horizontal circumference	34.50
Greatest transverse diameter	9.10

While some deviations are physiological, marked differences from these measurements involve a pathological significance. In microcephalia the circumference and all diameters are decreased; in hydrocephalus, increased. The dolichocephalic skull is long and flat, the antero-posterior diameter being proportionately larger than the transverse; in the brachycephalic the transverse diameter is too great and the skull seems flattened from before backward.

The nose is frequently malformed; the ears present various degenerative anomalies. The lips are thick, and imperfections in the teeth and prominence of the lower jaw are particularly constant. In short, congenital idiocy furnishes a rich collection of the various physical

FIG. 88.

Growth of beard in a female: stigma of degeneration (Incurables' Hospital).

stigmata of degeneration. Deformities, asymmetries, and anomalies of the face (Fig. 88), palate, eyes, body, limbs, and genital organs are present in profusion.

In addition to these inherited defects, the individual may be further deformed by the addition of localized palsies, with resulting shortening and contractures in the limbs.

The general appearance of the patient depends partly upon the physical symptoms and partly upon the degree of the intellectual defect. General body-growth is almost unexceptionally interfered with. Nearly all the patients are undersized, ill-proportioned, and clumsy in their movements. Nearly all are homely, and profound idiots are extremely repulsive in appearance. They lie in bed motionless or performing rhythmical movements, mute or giving utterance to meaningless sounds. The attention cannot be attracted. Saliva runs from the open mouth; the fæces and urine are passed in bed. In milder grades the general appearance varies with the type of the infirmity, some of the patients being constantly in action, running to and fro in the wards, others sitting quietly by themselves, others timid and shrinking, and others presenting no immediately noticeable differences from the appearance of normal people.

Mental State.—The psychology of idiocy has to do with defects in sense perceptions and in the acquisition and association of ideas, brought about by arrested development of the brain.

It is by the proper appreciation and association of sensations that as children we become acquainted with our environment. As the impressions of sight, hearing, taste, smell, and touch are repeated and multiplied, they begin to acquire for us individual significance, until from associated memories we gain ideas regarding the objects by which we are surrounded.

Those who are born with, or who early acquire, defects in specialized brain areas or in sense organs are more or less cut off from the chain of ideas normally obtained through these parts. The congenitally blind can never acquire conceptions of color; the child born with bilateral deficiencies in the temporo-sphenoidal lobes is not only deaf, but is also, unless systematically educated, deprived of the possibility of speech. In the larger number of idiots, however, the lesions are distributed rather than selective, and the mental condition must be explained by lack of interpretation of sensorial messages, rather than by fault in their transmission. It is psychic rather than sensorial. Sensations pass to the receiving centres and are perceived, but through lack of attention fail to acquire an intellectual value. As Seguin[1] aptly says, "The idiot sees, but he does not look." We may add that he hears, but he does not listen, and that he tastes, smells, and feels without noticing these sensations. He is consequently shut off from his normal fellow-creatures, and leads a life of vegetative isolation which is sad to look upon, but of the sadness of which he is himself unaware.

Although in a large majority of idiots the visual sense organ is unimpaired, anomalies of sight are the most frequent symptoms of idiocy. The child is not attracted by lights or colors or moving objects, and, through failure to observe them, does not acquire ideas of form or distance or color. In profound idiots this sense may be so useless that the patients pay no attention whatsoever to their environment. Threatening movements, such as shaking a stick before the eyes, causes the patient neither to wink nor dodge. In imbeciles, on the other hand, association of visual perceptions may be in every way normal, and between idiocy and imbecility there are various degrees of defect.

[1] *Idiocy and its Treatment by the Physiological Method*, N. Y., 1866.

Vision is the most important of the senses from an educational point of view, and its defect, either peripheral or central, seriously interferes with normal development.

Defects in the ear or auditory nerve are even less frequent than defects in the peripheral visual apparatus. Nevertheless, many idiots do not notice sounds at all. In some profound idiots a sudden and unexpected noise may make them jump or give other evidence of hearing. Others pay no attention whatsoever to noises of any kind, and in such cases it is impossible to tell whether the patient is really deaf or whether the apparent deafness is due to lack of attention.

In less profound idiots the attention can be usually attracted by various auditory stimuli, and especially by music; in imbecility hearing is in all respects normal.

Taste and smell are frequently affected in idiocy. The patients do not distinguish between different odors and different flavors. In rare cases the sense of smell is abnormally acute. Most idiots are gluttons. They will often eat anything, even the most repulsive substances, that they can lay their hands on. Their voracity is only limited by the quantity of food within reach. Many idiots, who cannot be attracted by any other means, show signs of excitement and pleasure at the prospect of a meal.

General sensibility appears diminished. In profound idiocy there is not only an apparent absence of the perception of tactile sensations, but the patients also seem to suffer no pain even from severe wounds and mutilation.

The muscular sense appears late in all idiots, and in profound idiots it may never develop. One of the patients at the almshouse, thirty years of age, though not paralyzed, has never been able to sit up or walk or to make any co-ordinated use of the hands. When the development of the muscular sense is only retarded, the child does not begin to walk till he is four or five years of age or even older. He is slow in learning how to use the hands; he does not learn how to grasp objects properly.

When some power of co-ordinated use of the muscles is finally acquired, it is clumsy and grotesque. The gait, when not interfered with, is slovenly. The patients let things fall from their hands; their movements are ataxic and irregular; upon attempting to execute some intended movement the body and limbs are thrown into contortions; on attempting to speak the face is thrown into grimaces. The automatic movements, or the *tics* of the French writers, are very common, especially in microcephalics. The patients seem to derive pleasure from rhythmical oscillation; they wag their heads from side to side or from before backward or in a rotatory way; or the movement may be chiefly at the hips and back, the patient slowly rocking himself to and fro or from side to side. There is often also a rhythmic play of the features or of the arms and hands.

The organic sensations may be almost entirely wanting in idiots. Profound idiots pass their urine and faeces apparently unconsciously, and show but little or no desire for food or drink unless it is put before them. In milder degrees these defects may be less marked, and in imbeciles they are usually normal, or, as regards food and drink, at least

exaggerated. The instinct of self-preservation, which underlies the struggle for existence throughout the animal kingdom, may be entirely wanting in idiots. From lack of interpretation of the messages sent by the sense organs, from absence of memory, from want of correlation of cause and effect, the idiot is afraid of nothing, shrinks from nothing, and exposes himself to constant danger. Where the mental enfeeblement is less pronounced the patient may know enough to neither lean against the stove nor cut his fingers with a knife, but is indifferent to such dangers as of being run over in the street or of poisoning himself by eating or drinking anything he may lay his hands on. Many of the applications for admission to idiot asylums are made for the purpose of securing protection for these unfortunate creatures.

Sleep is usually undisturbed in idiocy. Insomnia and agitation result occasionally, however, from various causes.

The desire for movement is often well developed. The patient may be constantly walking to and fro or making the automatic movements or ties of which mention has already been made. Sollier[1] describes the case of an epileptic idiot who took a particular delight in climbing trees, and who (although he never learned to feed himself) could do so without falling.

The sexual instinct is absent in profound idiots. Although nearly all idiots masturbate, in the profounder degrees of idiocy at least this is to be interpreted as an automatic movement rather as a gratification of sexual desire. Puberty is usually late in appearing. Sexual perversion of all kinds is frequently observed among imbeciles, and these latter also often take pleasure in obscene language and in personal indecencies. The instinct of imitation is never perfectly developed. In the patients who pay no attention whatsoever to their surroundings it is, of course, entirely absent; in the feeble-minded, on the other hand, it is present in greater degrees. Many idiots and imbeciles possess individual faculties or aptitudes disproportionate to their general intellectual powers. Robbie, a diplegic idiot at the Incurables' Hospital, New York, who cannot talk or feed himself, draws recognizable likenesses of the attendants and nurses. All idiots are fond of music, and some, extremely defective in other ways, show remarkable powers of playing on musical instruments and of remembering tunes. It is only in exceptional cases, however, that these aptitudes are in themselves remarkable.

Destructiveness and self-mutilation are also prominent characteristics in some idiots.

In profound idiocy the sentiments are absent. In lesser grades they are present, and often are very well developed. Imbeciles and the feeble-minded are susceptible to physical pleasure and pain, less so when these sentiments have a moral or intellectual basis. They often become attached to one another and to their attendants, and some of them evince great satisfaction at visits from the members of their families. All idiots are timid, and those who may seem courageous are in reality cowards, exposing themselves to danger from ignorance rather than from bravery. In the lesser grades of intellectual deficiency irascibility and attacks of sudden and apparent causeless anger are sometimes prominent features, during which the patients break everything within reach, and even attack

[1] *Psychologie de l'Idiote et de l'Imbecile*, Paris, 1891.

innocent individuals. The sentiments of right and wrong are never fully developed, and are absent altogether in profound idiots. Vanity is a prominent feature in imbeciles, and even in idiots in whom intelligence is almost totally lacking love of attention and personal adornment is often conspicuous.

It would be impossible to enter here into a discussion as to the part played by language in the elaboration of the intellect. That a certain degree of intelligence can exist independently of spoken or written language there can be no doubt. On the other hand, the range of ideas is necessarily limited in persons in whom no association of ideas is produced by symbols or sounds. In idiocy, inasmuch as the cerebral defects are usually generalized, the inferences to be drawn as to the educating power of written or spoken speech are not particularly valuable. The facts are briefly as follows: Profound idiots are practically mutes, as the sounds they utter are inarticulate and almost meaningless, neither do they understand what is said to them. In the paralytic forms the speech disturbances may be due to localized lesion of the centres of language. When infantile cerebral palsy occurs after these centres have developed and are functionally active, there may be aphasia similar to that of adults, except that it is associated with left hemiplegia more frequently than is aphasia in adults.

In most imbeciles and feeble-minded speech is retarded in appearing—a fact which is often the first sign-post of the mental condition. From the absolute mutism and lack of understanding of spoken or written language in profound idiocy to the cases in which these functions are in many ways normal there are various gradations. Disturbances of articulation, such as jerky utterances, lisping, "baby talk," etc., are frequent. Many imbeciles are very loquacious. Echolalia sometimes occurs in a remarkable degree in idiots who never talk voluntarily. It is entirely automatic, as the patient does not understand the words he repeats, thus being analogous to the long recitations and readings of which many imbeciles are capable, although unable to explain a word of what they have spoken or read. Reading, writing, drawing, and speaking in the lesser grades of idiocy may be at first sight normal. When, however, the patients are asked the meaning of what they say or write or draw, their apparent proficiency is shown to be largely mechanical, and the result of habit rather than of the outcome of higher intellectual activity.

From the defects in the faculty of language and from imperfect appreciation of special sensations it naturally follows that the intelligence in idiocy, even when these sensori-motor defects are not pronounced, is of a low order. The higher kind of memory, which is formed by sensorial impressions, and which, through the association and comparison of ideas, is the basis of judgment and will, is never well developed in these patients. If it were, they would not be idiots. This acquired memory implies comprehension—a faculty which is absent, in a great degree at least, even in those idiots and imbeciles who have special aptitudes for recollection. The judgment is always imperfect. Consciousness is absent in profound idiots; in the lesser grades it is impossible to determine exactly the degree of its existence. All volition is wanting in profound idiots. In some idiots and in imbeciles there are

volitional impulses which find their expression in action rather than in inhibition.

There can be no legal responsibility for the lower grades of idiots. They are unable to manage their affairs or to enjoy political or civil rights. Imbeciles and the feeble-minded who are at liberty may retain these rights, although they are in many respects irresponsible. The sudden, violent, and unprovoked outbursts of destructive or homicidal fury to which these patients are subject are in reality insane impulses, for which they are not legally accountable. Under the present lunacy laws of the State of New York patients with such tendencies may be confined in the State hospitals for the insane.

Diagnosis.—The diagnosis of idiocy merits more careful consideration than is commonly bestowed upon it. It is too frequently forgotten that an early appreciation by the physician of the degree of intellectual enfeeblement, the probable nature of the underlying lesion, and the special clinical class to which the patient belongs is associated with significant importance for prognosis and treatment. By being able to foretell what the outcome of the infirmity is probably to be the physician adds to his own reputation. But a more cogent reason for the closer study of these cases is the weighty consideration to the patient and to society of early diagnosis. With certain notable exceptions the chief hope for mentally deficient children lies in the timely institution of pedagogic measures. Education, which is totally useless in profound idiots only, can, in the lesser degrees of the condition, materially widen the intellectual horizon of those who, without it, would be little better than the lower animals. To be successful, however, it must be begun in infancy or early childhood.

The diagnosis of the intellectual defect depends chiefly upon an appreciation of what has been said concerning the psychology of idiocy. In pronounced and advanced cases no diagnosis is simpler, but in the early months of infancy, when gross defects are absent, the solution of the problem is difficult or impossible. It demands on the part of the physician not only a knowledge of infant psychology, such as may be found in Preyer's work,[1] in which the normal development for the first forty months of life is portrayed, but also that he be familiar with such deviations from the normal standard as are not inconsistent with ultimate mental and physical health. When physical abnormalities are added to the psychical defects the diagnosis is much easier. Paralysis, deformities, or any of the stigmata of degeneration are valuable diagnostic aids, and often call attention to peculiarities in the mental state which otherwise might have, for a time at least, escaped observation ; similarly, an arrest of intellectual progress after some well-recognized cause may prevent any possibility of doubt.

The diagnosis of the cause is frequently impossible and only occasionally of practical service, yet its importance may be very great. The conditions with which the commoner forms of idiocy are most likely to be confused are cerebral tumors and cretinism. Tumors, especially solitary tubercles, in the cerebrum or cerebellum may cause the enlargement of the head and other signs of hydroceph-

[1] *The Mind of the Child:* "1. The Senses and Will; 2. The Development of the Intellect," trans. by H. W. Brown, N. Y., 1888.

alus internus; cretinism, in common with most of the other forms of idiocy, is usually observed in the early months of life. Until 1891 it was regarded as one of the more hopeless forms of idiocy. The diagnosis of these affections is so fully described elsewhere (Vol. III., p. 693) that it is only necessary to allude to it here. But that the possibility of their existence should be ever present in the mind of the physician who has to do with mentally deficient children has been amply proved by the history of cerebral surgery and of the therapeutic uses of the thyroid gland.

Diagnosis as to Clinical Type.—In preceding pages no attempt has been made to classify the various brain defects which may give rise to idiocy. While many such classifications have been elaborated by eminent authorities, and have been based successively upon causation, pathological anatomy, time of appearance, and clinical type, none of them is in all respects satisfactory. It has seemed more advisable in this article to insert in this place descriptions of the more important clinical forms, instead of attempting any systematic classification.

Hydrocephalic Idiocy.[1]—Hydrocephalic idiocy includes the cases in

FIG. 89.

Hydrocephalic imbecile (Incurables' Hospital).

which the mental enfeeblement is due to pressure on the cortex brought about by over-distention of the lateral ventricles. It sometimes complicates other varieties of brain anomalies, but frequently exists as an independent condition. It is especially prone to appear in the children

[1] For a fuller discussion of hydrocephalus see Peterson, *N. Y. Med. Journ.*, July 25, 1896.

of persons who were drunkards or were themselves branded with some of the stigmata of degeneration.

Enlargement of the head is its most striking symptom (Fig. 89). If the onset of hydrocephalus is delayed until the sutures and fontanelles are closed, the increase in the size of the cranium is often not particularly noticeable; when, however, the condition is congenital or appears in the earlier months of life, this symptom is very conspicuous. The head is usually rounded in shape, it may be increased by one-third of its normal circumference, and offers a great contrast to the small face below it. The frontal eminences are unusually prominent. The fontanelles are open in early cases only. The teeth are defective as a rule. The face is wrinkled and old looking. Hydrocephalic children are the "little men" and "little women" of the idiot asylums. The increase in size and weight of the cranial contents may be more than the muscles of the child can support, and he is consequently obliged to remain lying down or to have the head supported when he sits up.

The general symptoms consist in anæmia and emaciation, which frequently pass into marasmus. Rachitic deformities, variously distributed palsies, and epileptiform convulsions are frequent complications. The children do not learn to walk early even when there is no paralysis. The mental deficiency is variable in degree. As a rule, the patients are serious, quiet, and not inclined to movement, offering in this respect a contrast to microcephalics of a similar degree of general intelligence. The power of speech is fairly well acquired in many cases. Vanity is a conspicuous characteristic, especially in females. The course of hydrocephalus is uncertain. In perhaps the larger number of cases the encephalic affection is progressive, and the child dies in infancy or early childhood as a result of complicating disease, of which pneumonia is the most frequent. In other cases the process is checked and the patient continues to live as a quiet and inoffensive imbecile, amenable to a certain degree of education.

Microcephalic Idiocy.—In nearly all paralytic idiots the head, in addition to being asymmetrical, is smaller than normal; the patients are consequently microcephalic. Microcephalia and microcephalic idiocy, however, are terms which are reserved for the cases in which the brain and its case are uniformly diminished in size, without offering evidence, such as paralysis, of a circumscribed lesion. Considered as such, microcephalia is much less frequent than hydrocephalus. As clinical types the patients offer in many ways direct contrasts to hydrocephalics (Fig. 90, p. 873). In many microcephalic skulls the sutures have been found closed at an early age, but there are also so many recorded cases of abnormally small brains in which the sutures had remained open that early closure of the sutures cannot be regarded as an important cause of this condition. Indeed, very little is known concerning the causes of microcephaly, other than those which are generally active for the various phases of idiocy.

The general appearance of microcephalic skulls consists in a cone-shaped or oxycephalic skull, which is diminished in size. The forehead is low and slanting, the eyes seem very high up, and the unusual prominence of the face gives the whole head a bird-like or animal-like appearance. General bodily development is usually much repressed, and

many of the patients are absolute dwarfs. When the intellectual defects are very pronounced, the general characteristics of microchepalics are the same as those of profound idiocy, whatever its cause. But in lesser degrees microcephalia has a certain individuality. In such cases the

FIG. 90.

Microcephalic idiot (Incurables' Hospital).

patients are active and energetic, although their movements are purposeless. The different varieties of tics already mentioned are particularly frequent. The senses are active, but lack of systematic attention prevents sense-impressions from gaining any intellectual value. Few microcephalics can talk coherently. The sexual instinct is not well developed. Physical stigmata of degeneration are frequent. The course of these cases is, in a way, better and worse than that in hydrocephalic idiocy, for while the nutrition and general health are often good, the patients sometimes living to an advanced age, the prospects for intellectual evolution are more gloomy than for hydrocephalus, and are in direct ratio to the size of the head.

Paralytic Idiocy.—Under this heading are grouped the cases in which some gross cerebral lesion arrests the evolution of the mental faculties, and at the same time, by destroying more or less completely the upper segments of the motor path, causes paralysis of the limbs. It includes the important chapter of the infantile cerebral palsies in which intellectual defects are combined with the physical disabilities. The clinical types of paralytic idiocy vary with the distribution of the palsy, with the age at onset, and with the character and causes of the lesions. The distribution of the palsy is (1) hemiplegic, (2) diplegic, and (3) para-

plegic. Nearly all of the diplegic and paraplegic cases are congenital or are observed shortly after delivery. The hemiplegias occur most frequently in the first three years of life. The most reasonable explanation for the diplegias and paraplegias is an extravasation of blood in the longitudinal fissure between the hemispheres, brought on by traumatisms or pressure during delivery, which compresses the motor tracts from within outward on both sides. In the causation of hemiplegia, circulatory disturbances, such as hemorrhage (especially from the meninges), embolism, or thrombosis, are probably active. Meningitis is also an occasional cause.

According to Sachs,[1] acute polioencephalitis (a term proposed by Strümpell[2] as the cerebral counterpart of poliomyelitis) should not be considered as a cause at all unless every other morbid state can be excluded.

(1) *Hemiplegic Idiocy.*—In hemiplegic idiocy mental enfeeblement is associated with paralysis of one side of the body. Occasionally the condition is either congenital or is observed within a short time after birth : for the explanation of the mode of origin of such cases we are obliged to invoke the general etiological factors, such as have been described under parturitional causes. But by far the greater proportion of hemiplegic idiots have a clinical history identical with the ordinary one of infantile cerebral palsies—namely, that of an acute paralysis occurring in infancy or early childhood. The child is usually healthy until, during or after some infectious disease such as scarlet fever, measles, pneumonia, or, rarely, diphtheria, he has one or more convulsions, at the subsidence of which power is lost in one side. While this is the usual history, in some cases there are no evidences of infection or of severe cerebral disturbances, the paralysis appearing as a result of a fit of coughing or of vaccinia or without any cause being discoverable.

Although mental defects are very frequently associated with the paralysis, many patients escape intellectual impairment. Of 160 cases of infantile cerebral palsies examined by Wallenberg,[3] 50 presented mental defects, and in 15 imbecility followed the complicating epilepsy ; of 80 cases of Gandard[4] 15 were feeble-minded and 19 were idotic. Age is an important element in causation. Of 110 cases observed by Osler,[5] 15 were congenital, 67 occurred in the first two years of life, 14 in the third year, the others being distributed through the other years up to the tenth. In the congenital and in the early cases idiocy is apt to be profound ; in later cases, occurring in older children, imbecility and feeble-mindedness are more frequent results if the mind is impaired at all.

The symptoms of hemiplegic idiocy, after the disturbances of onset have passed away, are mental and physical. The mental symptoms present all the varieties from those of children who are merely backward, to those of the helpless institution inmates who are absolutely devoid of all intelligence, and who, cut off from understanding of their environment, lead purely vegetative lives. The intellectual impairment is, how-

[1] *Op cit.* [2] "Ueber die Acute Encephalitis der Kinder.," *Jahrb. der Kinderklinik*, 1884.
[3] "Ein Beitrag zur Lehre von den Cerebralen Kinderlähmungen," *Jahrb. der Kinderheilk.*, 1886.
[4] *Contribution a l'Étude de l'Hémiplegie cerebrale infantile*, Geneva, 1884.
[5] *The Cerebral Palsies of Children*, Phila., 1889.

ever, less constant and less pronounced in the hemiplegic than in the other paralytic forms. Of 52 cases examined by Sachs and Peterson,[1] the mental impairment was classified as feeble-mindedness in 16, imbecility in 31, idiocy in 7, and insanity (epileptic) in 1.[2]

The physical symptoms are those of hemiplegia, and differ in slight respects only from the hemiplegia of adults. The loss of power is rarely absolute, the tendon reflxes are almost always exaggerated, foot clonus is often present, rigidity and contractures are pronounced. When the rigidity is extreme it may be impossible to elicit the reflexes. The leg and face usually recover more than the hand. The sensation and electrical reactions are unchanged. Infantile cerebral palsy differs from the adult in certain respects. Squint is more common. There is a flattening on the side of the head corresponding to the intracranial lesion, and athetoid, choreiform, and associated movements are especially prominent in children. Also, interferences with growth cause shortening of the limbs. Epilepsy, which is only occasionally seen in the cerebral palsies coming on in later life, occurs in at least one half of the infantile cases. It is one of the gravest factors of the condition, for recurring convulsions often lead to imbecility and idiocy even when these symptoms had not followed the original palsy. It also predisposes its victim to the development of psychoses.

(2) *Diplegic* (*Double Hemiplegic*) *Idiocy.*—In diplegic idiocy the paralysis, instead of being on one side, involves all four extremities (Little's disease). The condition is almost always congenital, and is supposed to be due to mechanical violence to the head during parturition. Of Sachs's and Peterson's cases of diplegia 71 per cent. presented mental anomalies. This proportion is consequently higher than in hemiplegia, and the intellectual involvement is also more serious. Of 17 cases 15 were idiotic or imbeciles and 2 only were feeble-minded. The physical symptoms are those of bilateral hemiplegia. The spasticity is extreme (Fig. 91). Most of the patients cannot walk at all on account of the contractures of the muscles of the lower extremities (especially the adductors), and those who are able to walk do so by the "crossed-leg progression." The adductor spasm is often so pronounced that the thighs are constantly held crossed, and the patients are almost entirely deprived of any use of the lower extremities. The hands are either useless or else extremely clumsy in their movements. The head is small and asymmetrical. Athetoid and similar morbid movements are particularly frequent. Speech is rarely acquired. Curiously enough, epilepsy is less frequent than in the hemiplegic variety.

(3) *Paraplegic Idiocy.*—In paraplegic idiocy the palsy is identical in its general characters with the diplegic form, except that it is limited to the legs. The separation of these two types will probably be some day shown to be unjustifiable. Both are of bilateral cerebral origin, and the differences between them are probably only of degree. From the frequency of athetosis, of exaggeration of tendon activity, and of other indications of irritation in the motor tracts to the upper extremities, it

[1] "A Study of Cerebral Palsies of Early Life, based upon an analysis of 140 cases," *Journ. of Nerv. and Ment. Dis.*, May, 1890.

[2] These were chosen from a series of 105 cases, of which the others were intellectually normal.

is entirely reasonable to assume that in many cases of so-called cerebral paraplegia the hands and arms were at one time involved, but recovered sufficiently to give especial prominence to the affection in the legs. As in diplegia, so in these cases, the condition is usually congenital and the

FIG. 91.

Diplegic idiot (Incurables' Hospital).

mental symptoms are frequent and pronounced. As regards physical symptoms, there are asymmetries and deformities of the skull, retardation of growth in the limbs and various *stigmata degenerationis*. The contractures in the legs are extreme, spasmodic crossing of the legs and talipes equinus being especially conspicuous. As in diplegia, epilepsy is less frequent than when the paralysis is limited to one side.

Epileptic Idiocy.—Inasmuch as the cases of idiocy, imbecility, or feeble-mindedness which are complicated by epilepsy differ in essential particulars from those in which recurring convulsions are absent, some mention must be made of them as a class, even at the risk of repeating what has already been said concerning the other forms.

Epilepsy occurs in idiocy in three principal ways :

1. As a complication of some gross focal lesion which at the same time produces paralysis. This is the form observed in paralytic idiocy.

2. As a complication of some gross lesion which does not press sufficiently on the motor tract to bring about paralysis. Hydrocephalus, meningitis, etc. may be the causes of this form.

3. Idiopathic epilepsy, for which there are no gross lesions sufficient to explain the convulsions. Idiopathic epilepsy may be the direct cause of the mental decadence in that the patient is normal until the fits

appear, but that after their appearance intellectual progress diminishes or ceases or such mental capacity as had been obtained becomes weakened. In other cases the appearance of epilepsy and feeble-mindedness is simultaneous, and both are to be referred to a common cause.

Whatever its origin, epilepsy renders the prognosis of any case much more serious. Its tendency is almost always progressive, and even when it begins as petit mal or as Jacksonian epilepsy the convulsions eventually become generalized, the intervals between them shorter, and the psychical conditions more and more hopeless.

The wide distribution of epilepsy among cases of idiocy, and the frequency with which it is the precursor or the accompaniment of intellectual weakness, make epileptic idiocy the most desperate, as it is the most frequent, or of any individual form.

Traumatic idiocy includes the various degrees of mental enfeeblement resulting from *trauma capitis* in the child. It requires no special mention, except to state that blows or falls upon the head, with or without fracture of the skull, may be followed by intellectual conditions similar to those already mentioned under other headings.

Myxœdematous Idiocy (*Cretinism*).—This condition, a familiarity with which has become so essential to every physician, is fully described on page 693, Vol. III., together with the other diseases of the thyroid gland, to which the reader is referred.

Sensorial Idiocy.—Deprivation of sight or hearing, either as a congenital condition or acquired in infancy or childhood, is a serious obstacle to intellectual perfection, and probably no children thus afflicted, even when the original lesions are entirely peripheral, ever attain complete mental development.

The best known example of this class was Laura Bridgman,[1] who, a blind deaf-mute, and also lacking in the senses of smell and taste, was a normal child up to the age of two years. Then, as a result of scarlet fever, both eyes and both ears suppurated and the child became blind and deaf. She eventually learned to write, and was intelligent in many ways. "She lacked certain data of thought, but not, in a very marked way, the power to use what data she had."

Further observations of this character are necessary before it will be possible to formulate the laws of sensorial idiocy. It is certain, however, that proper training, while it cannot provide conceptions of sensorial impressions which have never been perceived by the individual, can do much to render these unfortunate people intelligent and useful members of society.

Amaurotic Family Idiocy.—Sachs[2] has given this name to a rare form of idiocy of familiar type. The mental defects are first observed between the second and eighth months; they may progress to profound idiocy. Paralysis of the limbs may be flaccid or spastic. The ocular symptoms are impairment of vision, which may lead to total blindness. The opthalmoscope shows characteristic changes in the neighborhood of the macula, originally described as "a white speck, more or less

[1] See Donaldson: "Anatomical Observations on the Brain and several Sense Organs of the Blind Deaf-mute Laura Dewey Bridgman," *Am. Journ. of Psychology*, 1890 and 1891.

[2] *N. Y. Med. Journ.*, May 30, 1896; *Deutsch. med. Woch.*, Jan. 20, 1898.

circular, in the centre of which is a brown point, offering a sharp contrast to the white." In late cases there is optic atrophy. Few of these patients outlive the second year.

Idiots Savants.—In the description of the psychology of idiocy mention has been made of the special aptitude of some idiots, more or less striking examples of which are to be found in every institution devoted to the care of these unfortunates. In most cases the aptitude stands out in contrast to the poverty of the intellect in other ways, and is not in itself remarkable. Some individuals, however, although conspicuously deficient in general intellectual qualities, possess special faculties much more highly developed than those of ordinary normal people. They are called *idiots savants*. They constitute a small but interesting chapter in psychology, and present to the student problems analogous to to those encountered in the study of genius.

The aptitudes, which include arithmetical, musical, artistic, imitative, and similar faculties, are most frequently encountered in profound and congenital idiots, are developed early, and are prone to disappear in adult life. They have been carefully studied by Peterson,[1] who has collected many examples of the condition, some of which may be briefly referred to.

Tom Fuller, a native African, never knew how to read or write, but had phenomenal powers in arithmetic. Asked how many seconds a man had lived who was seventy years, seventeen days, twelve hours old, he replied in a minute and a half, 2,210,500,800.

Blind Tom, the celebrated negro musician, showed intelligence only in regard to sound. He could repeat whole conversations, but without comprehension, and his own speech was little more than inarticulate sounds. He could, however, play on the piano from memory any piece of music, no matter how intricate, after hearing it but once. He is said to have retained as many as five thousand musical compositions in his memory.

Gottfried Mind was an imbecile who died in 1814. He was so skilful in the drawing and painting of cats that he achieved the distinction of being known as "the cats' Raphael." Many examples of his work are to be seen in European galleries.

Special memories and aptitudes for buffoonery are especially noticeable. Winslow records the case of a man who remembered the day of burial of every person who had died in the parish for thirty-five years, and who could repeat with perfect accuracy the names and ages of the deceased and of the mourners at the funeral. He was a profound idiot, and could not reply intelligibly to a single question beyond this, nor be trusted even to feed himself.

Many of the court-fools of former times were imbeciles. Areim describes one who was a fat fool, a trifle over three feet high, two yards in circumference, at the age of forty years.

As Peterson says, aptitudes of this kind are of a low grade, and are imitative rather than creative. He explains them by assuming an increased number of cellular elements and sensori-motor associations in definite parts of the brain.

Prognosis.—The prognosis of idiocy as regards the establishment

[1] "Idiots Savants," *Appleton's Pop. Sci. Monthly*, Dec., 1896.

of full intellectual capacity is unqualifiedly bad. Those who are born defective or who in early childhood suffer an arrest of mental development can never be brought up to the normal standard. But much can be done. The doctrine of "once an idiot, always an idiot," has passed away. As was long ago maintained by Seguin, of whom Americans may be justly proud, many of these unfortunates can be made by proper instruction more appreciative of their surroundings and less burdensome to society. Even the profound idiot can sometimes be made less repulsive; the imbecile may be taught to talk, to learn various occupations, to check his impulsive tendencies, to be cleanly. The improvement in the feeble-minded consequent upon the skilful application of pedagogical methods is often astonishing. Many children who on entering an institution can do little or nothing for themselves, after a few years' training learn useful trades and become capable of supporting themselves. The statistics of the twenty-seventh *Annual Report* of the Royal Albert Asylum at Lancaster, England,[1] showed, with regard to the after-career of pupils discharged on completion of their seven years' training, that 10 per cent. were or had been earning wages; that 5 per cent. were remuneratively employed at home; and that 3.5 per cent. were capable of earning wages.

The amount of improvement is, however, limited, and cannot be brought beyond a certain point. As Fernald says,[2] "idiots cannot be taught common sense." Low grades of idiots continue to advance intellectually till the seventh or eighth year; higher grades, till puberty or a little later. The mental state then remains stationary, or, if not properly cared for, retrogresses till it terminates in dementia.

The prognosis as to life is serious. In many congenital or early acquired cases death occurs in the first few months or years of life. These patients also are more susceptible than normal children to disease-inducing agents, and are prone to succumb to any of the diseases of childhood, especially diarrhœa and pneumonia.

TREATMENT.—The treatment of idiocy is medical and pedagogical. Medical treatment, while it is controlled by the ordinary principles of pædiatric therapeutics and general hygiene, is beset by many difficulties. The patients are often too stupid to call attention to the fact that they are ill, and the reaction to sensorial irritation is so imperfect that they may be seriously or even mortally affected without appearing any differently than usual. Sollier[3] relates the case of a thirteen-year-old idiot who was sent to the hospital suffering from pneumonia. The only symptoms observed were an increase of the customary physical and mental languor, but the autopsy showed the lungs consolidated in gray hepatization—and a suppurative cerebral meningitis.

In the treatment of patients who do not complain themselves, and in whom the symptoms of acute disease are not revealed by general and conspicuous evidence, the physician is thrown upon his own resources, and upon his watchfulness and care in the examination for special symptoms depends in large part the welfare of the patient.

The treatment of individual conditions has no particular differences in idiotic than in other children. Diarrhœa, affections of the skin and

[1] Quoted by Shuttleworth: *Mentally Deficient Children*, London, 1895.
[2] *The History of the Treatment of the Feeble-minded.* [3] *Op. cit.*

mucous membranes, pneumonia, and phthisis, all of which are frequent complications of idiocy, are cared for by the usual means. With the exception that in the mentally deficient more care is necessary, the usual requirements as to food, clothing, bathing, exercise, etc. are maintained. The two specific procedures, whose object is cure, are thyroid therapy in myxœdematous idiocy (Vol. III., p. 697) and craniectomy in premature ossification of the fontanelles. While in the larger number of cases of this latter condition, as shown by Bourneville, the too-early union of the bones is expressive of a general developmental defect, and consequently not amenable to surgical treatment, we are not yet in a position to deny that in some rare cases it is of service. It is only permissible, however, when undertaken before the end of the second year, and in cases in which the clinical course indicates that the mental arrest is in some way dependent upon the symptoms.

In hydrocephalus little is to be gained by surgery. These patients are weak and sickly, and do not bear cranial operations well. When the pressure symptoms are extreme the fluid may be partially drawn off by lumbar puncture, although this procedure is palliative rather than curative.

Into a discussion of pedagogical methods it is impossible to enter here. While much can be done by an intelligent mother in the way of promoting sensorial development, the power of co-ordinated movement, and habits of cleanliness, feeble-minded children are best cared for and trained in institutions, whose teachers devote their lives to this work. The successful education of the idiot requires upon the part of the instructor patience, skill, and, above all, experience. In the institutions the children are taught regular exercises and purposeful occupations. Various mechanical devices are employed to train the touch and the eye; music by appealing to the ear improves attention; speech is either taught by the "oral method" or perfected by the constant and methodical use of that faculty. Every backward child should have the benefit of this systematic instruction. Few are so hopeless as to derive no benefit from it, and by this means many, if they do not become ornaments of society, cease to burden it.

The writer desires to express his obligations to Dr. Francis C. Wood, who kindly took the photographs which illustrate this article.

SEXUAL PSYCHOSES.

By MORTON PRINCE, M. D.

SEXUAL PERVERSION; CONTRARY SEXUAL INSTINCT; SADISM; MASOCHISM; FETICHISM.

Sexual Perversion.

The subject of sexual perversion is important medically, socially, and forensically—medically, because it may form part of a group of symptoms manifested by the insane or degenerate, or, as some writers hold, may be a psychopathy in itself; socially, because it may in its consequences deleteriously affect social decency and order; forensically, because it sometimes leads to murder, theft, and other crimes, the motive for which cannot be understood without a knowledge of the perverted instinct and of the underlying psychopathy when this is present, as is usually the case.

So far as this perversion is the expression of pathological conditions of the nervous system, it is important that it should be considered in this work. The consideration of the subject here is still further made desirable by the fact that even when the deformed instinct can be looked upon as only a cultivated vice, nevertheless in many such instances the individuals who practise it are affected with some form of insanity or imbecility, so that they are properly the subjects of medical study.

Then, too, even if we may take the ground that it is almost always through perversity or cultivation that psychopathic individuals develop a perversion of the sexual instinct, nevertheless it is difficult to deny that the final result may be a true psychosis or perversion which may have the force of imperative feelings. Thus, though vice may be the road traversed, the last stage may be disease.

The sexual instinct may be associated with and excited by thoughts, feelings, and acts which to a normal individual are repulsive or without any sexual association; in other words, the sexual instinct is perverted. It may be excited by (and therefore lead to) acts of cruelty or violence inflicted upon the opposite sex (sadism), or by the opposite state, the passive suffering of pain which has been inflicted by the opposite sex (masochism); or it may be excited by certain objects, whether a part of the female body or dress or other objects (fetichism). Perversion may further take the form of homo-sexuality; that is, the substitution or coexistence of sexual feeling for the same sex in place of, or by the side of, that for the opposite sex. This is also known as *contrary sexual instinct* or *sexual inversion*. These different forms of perversion have also been classed as varieties of sexual paræsthesia. Before entering

into a further analysis of these conditions it will be well to briefly consider the pathological groundwork upon which they rest. We shall then understand better the relation of the perverted instinct to the individual.

The first important question is, How far are these perversions the necessary expression of a disordered nervous system, and how far do they represent merely indulgences in vice and cultivated habits? So far as they are simply vicious habits, they can only be regarded as *perversity*, not *perversion;* that is, as vice, not disease. This view is not altered even in the case of individuals who have degenerated or in other ways diseased nervous systems, provided that they have cultivated the habits, and that the habits are the direct result of such cultivation, as may occur in normal individuals. A paranoiac or an imbecile may cultivate vice as well as a sound-minded person. Degenerate people may not be morally or legally responsible, but this is not a question of responsibility, but of *genesis*. What is the origin, and what influences have developed the sexual aberrations?

If these aberrations are the manifestation of a diseased nervous system in the same sense as hysteria is the manifestation of a neuropathic condition, then these sexual phenomena are true perversions and pathological. On the other hand, as far as these perverted instincts are merely cultivated habits of feeling or acts—that is, are modes of perversity—they do not properly belong to the subject matter of a medical work, unless, perhaps, the individuals otherwise exhibit pathological defects. But if they are the symptomatic expression of disease, they properly constitute a part of medicine.

There are two views regarding the nature of perversion which are radically opposed, and which from a social and therapeutic point of view have respectively important consequences. The one leads to therapeutic nihilism and social hopelessness, the other offers hope and possibilities.

The theory that has been most widely accepted by writers on the subject is that sexual perversion has its basis in a diseased nervous system, which in most cases is the result of inheritance. A psychopathic or neuropathic groundwork is in almost all cases essential, but the perverse phenomenon arises spontaneously without external cause. Its origin is therefore entirely independent of cultivation by vicious habits, education, or seduction. In some instances, it is equally maintained, these perversions are *acquired* as the result of cultivation, with or without the co-operation of an inherited neuropathic condition. But it would seem that with the exception of fetichism, which is always acquired, the acquired cases are a distinct minority. In most cases *nascitur non fit.*

"This perverse sexuality," says von Krafft-Ebing, speaking of the contrary sexual instinct, "appears spontaneously, without external cause, with the development of sexual life, as an individual manifestation of an abnormal form of the vita sexualis, and then has the form of *a congenital* phenomenon; or it develops upon a sexuality the beginning of which was normal, as a result of any definite injurious influences, and then appears as an acquired anomaly. Careful examination of the so-called acquired cases make it probable that the predisposition, also present here, consists of a latent homo-sexuality, or at least bi-sexuality, which for its manifestation requires the influence of accidental causes to

rouse it from its slumber.[1] While objections may be made to this theory when applied to homo-sexuality, the theory has considerable strength when we seek for an explanation of sadism and masochism. Between the homo-sexual influences and the sadistic influences which lead to murder and mutilation of the victim's body there is a wide gulf, and we should not necessarily expect a similar pathological condition as a basis of both. As to sadism, von Krafft-Ebing expresses the opinion that, "as a rule, it may be safely assumed that the psychopathic state (perverse instinct) exists *ab origine*."

Von Krafft-Ebing's[2] work being almost the first to treat systematically the subject of sexual perversion, and presenting the matter with great erudition, has been very widely drawn upon by subsequent writers. The interpretation of these aberrations given by this author has very profoundly influenced medical opinion, and has been quite extensively accepted. This work was soon followed by a publication on the contrary sexual instinct by A. Moll,[3] who also adopted the congenital theory originally proposed for this anomaly, it is true, by Casper,[4] in 1852. In America, Kiernan (1888), Chaddock, and Lydston (1889) have advocated the congenital theory. Quite a large number of contributors to the subject, with reports of numerous cases of different kinds of perversion, have appeared since von Krafft-Ebing's work. More lately a strong protest against these views has appeared in the work of von Schrenck-Notzing.[5] This author, in opposition to the opinion of the writers just cited and of others, has urged with great force that sexual perversion, instead of being an original psychopathy, is a cultivated instinct. Heredity and a neuropathic constitution play an important part, but this part is only that of weakened power of resistance to external influences. The contrary sexual instinct is, as such, not inherited, nor is it congenital any more than are the majority of psychoses, but only that tainted or degenerated nervous system in consequence of which the individual offers a mental weakness, a lack of resistive power to external influences, and a lack of control over desires, however excited. By a process of cultivation the neuropath develops feelings and gives them expression in outward acts over which he sooner or later may lose all control. The first awakening of the perverse instinct may be entirely fortuitous or by auto-suggestion, or it may be by seduction or other accidental external circumstances; from this time on it is a process of education. Von Schrenck-Notzing would explain in this way the origin of all forms of sexual perversion, although in the exposition of his theory his argument is devoted almost entirely to the contrary sexual instinct.

The influence of von Krafft-Ebing's able exposition of the subject, as just said, has colored much of the writings of others, but I think the

[1] *Psychopathia Sexualis*, translated by Charles Gilbert Chaddock, M. D., 1893. See, also, "Zur Erklärung der Conträren Sexual Empfindung," *Jahrbücher für Psychiatrie und Neurologie*, 1895.

[2] According to von Krafft-Ebing, the most important previous writings were those of Moreau (*Des aberrations du sens génésique*) and Tarnowski (*Die Krankhaften erscheinungen des Geschlechts-Sinnes*).

[3] *Die Conträre Sexual Empfindung*, Berlin, 1891.

[4] Westphal adopted the congenital theory for contrary sexuality.

[5] *Suggestive Therapeutics in Psychopathia Sexualis*, translated by Charles Gilbert Chaddock, M. D., 1895.

conviction must be forced upon the careful student of these writings that the attempt to make vicious habits the result of congenital anomalies has been based upon evidence that from its very nature must be incomplete and unreliable. Rather, the cultivation theory, modified perhaps, is that which must commend itself to the intelligent and common-sense mind. As von Schrenck-Notzing has pointed out in his careful study of von Krafft-Ebing's published cases of contrary sexuality, very few of them will stand analysis. The autobiographies of such individuals are untrustworthy, and probably there is no class of people whose statements will less stand the test of a searching cross-examination than the moral pervert. The historical evidence further tells in favor of the cultivation theory. It would appear that homosexuality has prevailed in different times and amongst different peoples to an extent as to almost make of it a social custom. It was, for example, extensively practised amongst the ancient Greeks and Romans. To assume that ancient society was made up of degenerates is to reduce the theory to an absurdity. But while the congenital theory seems farfetched in connection with contrary sexuality, it is not so easy to put it aside, even though we may not wholly accept it, when we seek an explanation of sadism and masochism. There are certain physiological facts which would seem to indicate that at times, at least, the association of lust and cruelty may be a sort of freak of development—*i. e.* of the association of feelings—although cultivation must play a tremendous part in the final evolution of the freak-like association. Von Krafft-Ebing explains the origin of this, the worst of the perversions, as follows: "In an attempt to explain the association of lust and cruelty, it is necessary to return to a consideration of the quasi-physiological cases in which, at the moment of most intense lust, very excitable individuals, who are otherwise normal, commit such acts as biting and scratching, which are usually the result of anger. It must further be remembered that love and anger are not only the most intense emotions, but also the only two forms of active (sthenic) emotion. Both seek their object, try to possess themselves of it, and naturally exhaust themselves in a physical effect on it; both throw the psychomotor sphere into the most intense excitement, and then, by means of this excitation, reach their normal expression. From this standpoint it is clear how lust impels to acts that otherwise are expressions of anger. The one, like the other, is a state of exaltation, an intense excitation of the whole psychomotor sphere." [1]

"Sadism is, then, nothing else than an excessive and monstrous pathological intensification of phenomena—possible, too, in normal conditions in rudimentary forms—which accompany the psychical vita sexualis, particularly in males." [2]

The same writer lays stress on the weakness or absence of all normal restraining ideas in the psychopath, while free hand is given to the development and expression of the congenital perversion. But he neglects the influence which a deliberate cultivation may have upon a mild impulse or sensory association at the beginning. If sadism is an "excessive and monstrous intensification of phenomena 'existing' in a rudimentary form" in *normal* individuals, then the perversion is the *intensification*, and the question is, To what is this intensification due?

[1] *Psychopathia Sexualis*, p. 58. [2] *Ibid.*, p. 60.

Does it exist *ab origine* in its intense form as a result of pathological development, or is the intensification due to cultivation by a normally depraved and mentally weakened individual? or may it be due to both? The autobiographies and histories of cases found in the literature do not allow of the first interpretation. It is possible that certain anomalous sensory associations may be the starting-point of such perversion, and cultivation does the rest. For example, the case was brought to my attention of a perfectly healthy, mentally and physically, medical man who was sexually excited by the sight of a surgical operation. This person is a typically strong and healthy-minded man. Suppose him to have been a mental degenerate, how easy it would have been for him to cultivate sadistic impulses! The origin of sadistic impulses is of less practical importance than is that of contrary sexuality, as most of the individuals who exhibit the former are otherwise psychopaths (*e. g.* imbeciles, degenerates, or insane), though the question is of some importance, forensically, as bearing on the question of responsibility.

It is obvious that forensically it is of great importance to determine the origin of these perversions, especially contrary sexuality, for upon the view taken must largely—not entirely—depend the matter of responsibility. If this condition is congenital, responsibility must hinge upon the resisting power present in any individual case; but if it be the result of cultivation, the matter assumes a different aspect, for then we are dealing not with a perversion, but a perversity, a vice rather than a disease.

From one standpoint this view may be modified. It is well recognized that symptoms may by constant repetition become organized into independent habit neuroses or psychoses, which persist long after the original disease condition which gave rise to them has subsided. In the same way, nervous processes which originally were the expression of physiological stimulation of the nervous system may become so intensely cultivated as to become in time true psychoses and independent of volitional control in weak-minded subjects. Thus it is conceivable that sexual feelings and actions may by constant excitation (cultivation) become associated together and developed into a sort of quasi-independent neural activity which may thus become practically independent of the will, or, in other words, a psychosis. Thus, what was originally an accidental association may by cultivation become a true pathological condition. This is exemplified by other neuroses and psychoses. Morphinism, alcoholism, and various habit neuroses may originate in this way. Thus it may happen that through perversity a true perversion may become developed. Such a perversion may acquire all the force of imperative ideas or feelings, as von Schrenck-Notzing thinks. If the aberration of sexual paræsthesiæ are to be regarded as pathological (psychoses), this is unquestionably their true mode of origin and their true relation to perverse habits. It must still remain an open question, perhaps one of definition, whether mental habits thus formed are not to be still regarded as vice, and it must always be difficult to decide in individual cases whether or not cultivation has resulted in a psychosis. Perhaps the answer will depend upon whether the perverted feelings are really imperative or not—a matter not easy to determine. Finally, the important point, clinically, socially, and forensically, is the

recognition of the fact that many perverts, mostly sadists, are insane, hopeless degenerates, and that their acts, even if cultivated, are the result of a lack of the power of the use of self-restraint.

Sadism and Masochism.

Sadism and masochism are the association of cruelty and suffering with lust; but in the former the expression of this instinct takes an active form in the infliction of the suffering on another; in the latter the opposite occurs—namely, the lustful feeling is excited by the passive endurance of suffering. The term *algolagny* (*αλγος*, pain, and *λαγνος*, sexually-excited lust) has been suggested by von Schrenck-Notzing to include both these perversions, *active algolagnia* signifying sadism, and *passive algolagny* masochism. There are several forms in which each of these perversions finds expression.

Sadism.—The desire to satisfy the instinct may lead to murder—so-called lust-murder. It is probable that many murders, the motives for which have seemed enigmatical or which have been overlooked, have been of this kind. In true sadistic murders the victim is killed, not for the sake of concealing crime or accomplishing rape, but because the act of killing excites intensely lustful sensations. In the most monstrous development of this perversion the sadist may commit the most bestial acts, such as cutting up and mutilating the body of his victim. Sometimes portions of the body, especially the genitalia, are carried off. The Whitechapel murderer is probably a sadist.

Sadism may be confined to an ideational form, without actual commission of violence. This is illustrated by a case of my own: A boy aged 22, with bad heredity, was in the habit of lying on the bed and indulging in sort of day dreams of the most vivid kind. He would then imagine himself killing young girls, tearing them to pieces, and eating them. This gave him great sexual excitement—in fact, was a method of practising onanism. His ideas grew until he imagined himself living in towns and countries where, it being the custom for the men to destroy all the women in this way, great slaughter and cannibalistic feasts were held. The boy in other respects was hopelessly insane, but he had managed to conceal his morbid condition for years. He was sent to an asylum and his mind has since become still more degenerated.[1] Tardieu reports much the same sort of a case. Other forms which this perversion takes are—the mutilation of corpses; cutting or stabbing (without killing), whipping,[2] and defilement of women; whipping of boys; torturing of animals, etc. It is hardly necessary to narrate here instances of these different acts, many of which are disgustingly repulsive. Details of cases may be found in monographs on the subject.

A symbolic form of sadism has been described in which the perverse inclination expends itself in what are senseless and silly acts. Von Krafft-Ebing cites, amongst others (Case 35), the instance of "a man in Vienna who regularly visits several prostitutes only to lather their faces, and

[1] Reported in full in *Boston Med. and Surg. Journ.*, Aug. 20, 1896.

[2] The writer is cognizant of the case of a man who regularly visits a prostitute, paying her for the privilege of spanking her with a shingle, at the rate of a dollar a blow.

then to remove the lather with a razor, as if he were shaving them. He never hurts the girls," etc.

Masochism is the excitation of sexual feelings by the passive suffering of pain or abuse. A common mode of having the pain inflicted is by flagellation, but it may take almost any form—*e. g.* being trodden upon, cut, beaten, etc. But often this perversion consists of more than this: then the pleasurable sensations are excited by the idea of subjection to a woman, by whom the masochist is humiliated and made to feel that he is under her absolute power. In such cases the flagellation, or whatever be the mode of infliction of suffering, is only a symbol or evidence of subjection; it is merely an expression of the relationship. In fact, some masochists assert that when they have tried to realize their fancies by subjecting themselves to corporal punishment, the result has been a failure. It has been inferred from the statements of masochists that this idea of subjection is always the essence of this perversion. But this is hardly correct. It is not possible to recognize this idea in the accounts given in many cases, or if it is to be made out, it is only by a psychological subtlety that is hardly worth the analysis. Undoubtedly, many sexually enjoy the brute suffering of pain; on the other hand, with those who revel in the feeling of subjection the perversion may take a purely ideational form, without any attempt at realization of masochistic fancies. In this it is analogous to one form of sadism.

It has been thought that a distinction should be made between true masochism and the reflex stimulation of weakened powers by flagellation of the nates. But it can scarcely be necessary or possible to make this distinction practically, as it is hard to believe that any one would have himself painfully whipped for such a purpose unless the pain is accompanied by pleasurable feelings, in which case it becomes masochism. A masochist may or may not be psychically impotent for natural coitus.

As an example of the pure fancies of which this perversion sometimes consists, and which are made use of to excite sexual feelings, the following from the autobiography of a masochist is typical: "'She' is a peasant-woman, a rough, tall, large-boned woman of forty or fifty years. She is the possessor of a small remote farm, which she works with the help of her slave alone. The work begins before sunrise. At four o'clock in the morning she opens the shed where she has kept me shut up over night, and wakens me, as I lie on the ground, with a kick; then she leads me out and harnesses me to a milk-cart bound for town. She leads me by a halter, and urges me along. On the road she gets on the heavily-loaded wagon and sleeps until the destination is reached. Then in the open market-place of the town, still harnessed to the wagon, I lie down on the bare ground to rest. Those passing knock against me or step on me, without giving me any attention. After the stock is sold we start homeward. After a short rest the work begins again, always under the direction of the mistress, who holds me by the halter and urges me on. At seven or eight o'clock at night I am put up to rest, and sleep until the next morning, when the same thing begins again. Work and blows, blows and work—no pleasure, no recreation day in and day out.

"Another time I fancy myself in the rôle of a paid lover of an elderly female roué, who makes use of me sexually in the most reckless

manner, and in this direction makes the most shameful demands on me. If I do not submit to these willingly I am beaten and punished, and at the same time she despises me unspeakably, gives me the lowest household work to do, and on every occasion shows me how low an opinion she has of my manhood." [1]

Masochists who enjoy the actual infliction of pain usually employ prostitutes to abuse them, and for the purpose devise all sorts of schemes, sometimes curious comedies.

Like sadism, there is a symbolic form of masochism, consisting of the various devices to represent subjection, as where a man has himself thrown out of the house or shaved by a woman.

With this perversion there may be united greater or less tendencies to sadism and fetichism and contrary sexual instinct. Some cases seem to show a transitional state between masochism and fetichism, as when along with the association of pleasurable feelings with women's shoes there is the desire to be trodden upon.

Masochism seems to be a very common perversion if the statements of those who are subject to it and the evidence of prostitutes can be believed. In the form of flagellation it is common in this city (Boston), as in the large capitals of Europe, if the statements of those who should know can be believed. It certainly appears as a most extraordinary psychical phenomenon when it is considered in all its phases.

Mode of Origin of Masochism and Sadism.—In seeking for an explanation of masochism we must recognize certain facts and phases of its development. In the first place, an analysis of the cases shows there is almost always a neuropathic basis, usually the result of a tainted heredity. The depth of this degeneracy does not, however, seem to be as great as it is in the extreme forms of sadism. In the second place, the perversion begins at an early period of life with certain unusual associations of sexual, or other pleasurable feelings, with the idea of mental or physical suffering, subjection or pain at the hands of a person of the opposite sex (a woman). Starting with this primitive association of feelings, in process of time there becomes developed, on the one hand, most complicated mental states consisting of fancies, mental pictures, recreations, imaginary actions, etc., and, on the other hand, various forms of corporal punishment. Both may exist together or each separately. We have to explain both the primitive feelings and its later development. Further, I think it must be admitted that, practically, masochism consists not in the primitive association, but in the final development. The sexual perversion consists in the often monstrous expression of the original association of feelings, rather than in the rudimentary association. So long as this association does not find expression in active mental fancies and physical acts, or at least so long as these fancies and acts which are used by the masochists to sexually excite themselves have not been created, masochism can hardly, with strictness, be said to exist otherwise than potentially. In other words, the perversion consists in the sexual excitation, and is a form of masturbation. Now, a study of the autobiographies of masochists shows plainly that the development and expression of the original primitive

[1] Case 50, von Krafft-Ebing, trans. by Chaddock.

association have always been brought about by *cultivation*. The accounts plainly indicate that the masochist has wilfully and deliberately used all his endeavors to cultivate to the acutest form his imagination, to generate sensual images and dreams that would by association excite his sexual feelings, and when physical pain has been of service he has invented all sorts of devices for this purpose. In this way an association which primarily was loose has become so rigid, while the feelings which are united have become so acute, that an overmastering passion has become created that finally overwhelms the individual. This passion may then perhaps be called a psychosis, and possibly imperative, but, given the original association, the mode by which this development is brought about is *perversity* or vice.

As to the primitive association of lustful feeling with passive suffering it is not so easy to speak of its origin. It may well be that it may often, if not always, be congenital, something that exists *ab ovo*. But it does not follow from this that it is to be looked upon as necessarily pathological. There are a great many curious anomalous associations between customarily unrelated mental states which are experienced by healthy individuals, and which therefore can scarcely be regarded as more pathological than physiological. For instance, I have already mentioned the otherwise healthy medical man who was sexually affected by surgical operations. This connection between the sight of blood and the sexual sense is apparently tolerably common. In Case 54 of von Krafft-Ebing it was the primitive association, and gave rise to the masochism, which in this case, it is interesting to notice, was the idea of being killed, the counterpart of sadistic murder. It is highly possible that such associations are analogous to anomalous physiological associations between the functions of other parts of the nervous system. For instance, colored hearing is an unusual but physiological phenomenon. Galton has shown that the power of visualization exists in a curious way in some people who see figures before them when thinking of numbers. I know of an individual who has a queer and exceedingly disagreeable sensation in the testicles whenever he hears of any violent accident that involves mutilation of the body. The sight of ugly wounds causes the same sensation. It is exceedingly probable that if a census were taken similar to that made by Galton, it would be found that anomalous associations between the sexual instinct and other sensations and ordinarily unrelated ideas are fairly common and quite within the field of physiology. In the absence of definite knowledge on this point any attempt to explain the sexual perversion must be largely speculative. But it is easy to see that if, for instance, a person who was endowed with colored hearing should derive great and pleasurable excitement therefrom as from the sexual instinct, he might by cultivation develop it into what would be called a perversion. Some writers might then feel justified in speaking of it as a congenital psychosis. In other instances the starting-point of primitive association may be in reflex stimulation of the sexual centres by spanking, as in Case 49 (K.-E.). A physiological connection of this kind is admitted. Binet would explain the perversion in this way. Again, it is not impossible that the sexual instinct may be awakened indirectly through the general emotional state that is common to the sexual instinct on the one hand,

and pain, fear, anger, etc. on the other. Kiernan's atavistic theory, by which it is a survival of the cannibalistic tendency of lower animals, should be mentioned, though hardly acceptable. Krafft-Ebing has offered a theory which is extremely ingenious, though somewhat far-fetched. The chief element in this theory is "sexual bondage." By "sexual bondage" is meant that dependence of one person upon another of the opposite sex which in normal individuals may occur in a very extraordinary and remarkable manner, even to the loss of all independent will—a dependence which forces the party in subjection to acts and suffering which greatly prejudice personal interest, and often enough to offences against morality and law. A dependence of this kind is abnormal, but not perversion. This "*abnormality is hereditarily transferred to a psychopathic individual in such a way that it becomes transformed into a perversion.* The agent perfecting this transformation is the tendency of sexually hyperæsthetic natures to associate all impressions coming from the beloved person with the sexual impression." This theory, although ingenious, is hardly intellectually satisfying, nor are the different steps in the process made clear. This author, whose writings have given such prominence to sexual psychoses, also has overlooked the fact that, whatever the origin of the early association, the evolution of the "psychosis" is due to pure cultivation. According to von Krafft-Ebing's views, the whole completely developed psychosis (excepting in a small minority of cases) is congenital and the result of pathological conditions. If this were the case, there would be no way of accounting for the growth, both in diversity and intensity, of the psychosis, excepting by increase of degeneration of the nervous system. But this increasing degeneration is not the rule, but, on the contrary, recovery may take place. For the determination of the beginning of the masochistic feelings reliance has been placed upon the statements and autobiographies of perverts. To rely upon the memory of a person for the feelings that he had under particular circumstances in his childhood, to trust to any one's introspective memory in such matters, is risky business. Every one has forgotten much that is essential, and few, if any, can say what and when was the first beginning of masochism, or even of the sexual instinct, which he had as a child. Therefore, not knowing the exact circumstances, it is difficult to decide in individual cases on the question of origin.

Much that has been said applies to sadism. A primitive association of sexual feeling with pain arises either as the result of an accidental event, anomalous physiological condition, auto-suggestion or external suggestion, or a normal physiological state. By cultivation the final condition of sadism results.

Fetichism.

Fetichism is the association and excitement of lust with certain articles of female attire or certain portions of the female body.

Thus far, fetichism has only been observed in men. When the fetich is an article of dress, the perversity is seen in its purest forms, for then the excitation of sexual feelings may occur when the object is isolated from and not connected in idea with any particular person. When the

object is a part of the human body, excepting in the case of hair, it is not, of course, possible to separate the fetich from the individual to whom it belongs, and hence the associations are complex. But with articles of dress the fetich becomes a sexual excitant in itself. The most common objects of this kind are handkerchiefs, shoes, under-garments, petticoats, aprons, etc. Handkerchiefs and shoes are said to be the most common objects. The fondling, and even the sight, of the fetich is capable of causing great erotic excitement, and the fetichist seeks to possess these objects for this purpose. Thus it happens that fetichism sometimes has forensic importance because individuals are sometimes driven to thefts for the purpose of acquiring their favorite objects and the satisfaction of their desires. One man was found to have stolen three hundred articles of female apparel, including chemises, drawers, garters, etc.; when arrested he was wearing a chemise (Passow and Krauss[1]). Most commonly the desire is for a single object. A handkerchief-fetichist is excited by and steals only handkerchiefs. One man was found, when his house was searched, to have four hundred and forty-six ladies' handkerchiefs; he had also stolen many others (von Krafft-Ebing). A shoe-fetichist may spend much of his time trying to catch a glimpse of women's shoes or gazing into the windows of shoeshops. Another class of objects is some particular material, commonly fur, velvet, and silk, which at first sight seems to have the power in some people to tactilely excite erotic feelings, entirely aside from any relations to the human body. These materials may have this effect even when not made up into garments, but from the statements of some fetichists it would appear as if the effect were the stronger when they are worn as garments by women. Von Krafft-Ebing thinks that this fetichism cannot be due to original accidental association, as are the others, but that "it must be presumed that certain tactile sensations (a kind of tickling which stands in some distant relation to lustful sensations (?)) in hyperæsthetic individuals furnish the occasion for the origin of this fetichism." An analogy for this idea may be found in the curious sensations felt by some people from eating the skin of a peach, or in those similarly associated with the scratching of a slate-pencil. Perhaps the sexual feelings may be a like anomalous association. But it is not possible to disprove associations in early youth, for the circumstance of erotic feelings caused by a female clad in fur or velvet or other material might well be forgotten.

Less pure forms of fetichism are those where sexual feelings are excited by a woman only when completely dressed or clad in a particular costume. The most common parts of the female body that may serve as objects of this perversion are the hand, foot, and hair, less frequently the eyes, ears, and mouth. In such cases the hand-fetichist, for example, is excited by the touch of beautiful hands irrespective of the owner, and he seeks in every way to see and press them. According to Binet, hand-fetichists are very common. Hair is more like dress or fur in that it can be cut off and cherished as a thing apart. In consequence of this certain perverts are known as "hair-despoilers." These people are impelled by their erotic feelings to forcibly cut off and steal the hair of women. One man when arrested was found to have sixty-five switches

[1] Quoted by von Krafft-Ebing.

and tresses of hair.[1] Individuals with fetichism may also be tainted with sadism, masochism, or contrary sexuality.

The practical importance of fetichism is threefold: it may cause psychical impotence—that is, the pervert may be impotent for normal sexual relations or unless his fetich can in some way be brought into association with these relations; secondly, it may lead to theft; and thirdly, the mental suffering that may be indirectly caused may be intense. The psychical impotence in all marriage relations, the attacks of erotic excitement under the influence of the fetich, the induced onanism, the feeling of after-mortification, the self-recrimination, may in certain persons of a sensitive temperament cause a mental suffering which may be truly pitiable.

As to the *origin* of fetichism, Binet's explanation has been generally accepted—namely, that it is always due to some circumstance which in early youth excited the sexual instinct in association with the presence of the object that afterward became the fetich. This association, being once formed, persists, so that the object always excites the instinct. Fetichism is thus *acquired*, and is not congenital. The intensity which this association attains, so that it becomes a sort of imperative feeling or idea, must not let us forget that the histories of fetichists show that this *intensity* has been reached by deliberate cultivation or perversity.

In the great majority, if not all cases, there is a neuropathic base, usually through heredity, for the perversity, so that the fetichist has less resistive power than normal people. In such a substratum associations are easily formed, feelings and ideas acquire great intensity and fixedness, and the whole forms a quasi-psychosis of a more or less imperative nature.

Contrary Sexual Instinct (Homo-sexuality, Sexual Inversion, Hermaphroditism).

This aberration consists in the existence of sexual feeling for the same sex, coexisting in its fully developed form with entire absence of sexual feeling for the opposite sex. In the more moderate form there may still be inclination toward the opposite sex, but in the higher degrees of the perversion there may be a feeling of actual repulsion for the opposite sex, while the whole psychical personality, the tastes, feelings, and modes of thought of the individual may become changed to correspond with the sexual perversion; that is, the character of the male becomes feminine, and *vice versâ*. The justification for this aberration to be considered as a true psychosis depends upon the thesis that in a certain proportion (great majority) of cases it is *congenital* and a "partial manifestation of a neuro-psychopathic state, in most cases hereditary." It is therefore a functional sign of degeneration. According to this view, this manifestation in these cases is not acquired and is in no sense a perversity or vice, but a true anomaly or perversion of instinct in the sense that it is the product of *arrest* development, in the same way that any of the normal instincts, tastes, or sensory functions are the product of normal development. In other words, with "a normal anatomical and physiological state of the (genital) organs a sexual in-

[1] Voisin, Socquet, Motet, quoted by von Krafft-Ebing.

stinct may be developed which is the exact opposite of that characteristic of the sex to which the individual belongs." It appears spontaneously, without external cause, with the development of sexual life.

Various theories, many of them fanciful, have been proposed to account for the origin of this (according to this view) anomalous condition. Ulrich, himself a pervert, thought a female mind was enclosed in a male body. This condition he considered due to atavism. This fanciful notion, which reminds one of some of the early legends of human beings appearing in the form of animals, is maintained even by later medical writers (Magnan, Gley). This same idea appears in a new form in the hypothesis of Kiernan,[1] adopted by Lydston,[2] that contrary sexuality is a reversion to the primitive type of the lowest forms of life, which are bisexual. This bisexuality appears in a rudimentary form in adult human beings, as shown by the rudimentary female organs in the male. In contrary sexuality, while there is a differentiation of anatomical form, the nervous system is developed on the female type. One of Krafft-Ebing's patients independently suggested this explanation, the inadequacy of which is apparent when one considers that there can be no reversion, as, at the time when bisexuality existed there was no nervous system worth speaking of—nothing that corresponded with the human psychical sexuality. Atavism can therefore scarcely be accepted.

A modification of this theory has been proposed by Chevalier. The human embryo is bisexual. In its later development one or the other factor, male or female, pushes ahead at the expense of the other, but traces of the undeveloped sexual factor persist. Sometimes both develop, but in different directions, so that while the sexual organs of one sex are formed, the nervous system of the other is developed, and thus contrary sexuality results. All such theories are of course only another way of putting the original idea of the female soul in a male body. Kiernan writes: "It seems certain that a femininely functionating brain can occupy a male body, and *vice versâ*." These theories assume, what is probably not true, that there is a difference in the brain of the two sexes corresponding to the difference in the bodily form. Westphal, who first gave the name of contrary sexuality, thought the condition congenital, but refrained from hypothesis. The most sensible congenital hypothesis is undoubtedly that of von Krafft-Ebing, who thinks that an explanation "may perhaps be found in the fact that it represents a peculiarity bred in descendants, but arising in ancestry. The hereditary factor might be an *acquired* abnormal inclination for the same sex in the ancestors, which, being transmitted, becomes fixed as a congenital abnormal manifestation in the descendants."

Kiernan had also suggested this possibility for certain cases. The absence of proof of the ancestral facts, excepting in particular instances, prevents the acceptance of this hypothesis.

More in accordance with our psychological knowledge is the theory of Binet, although it is, nevertheless, rejected by most writers. By this theory the whole perversion is acquired through the force of association of ideas.

Amongst the names of those contributing to the subject are to be found many of well-known writers in neurology and psychiatry. But

[1] *Med. Standard*, Nov., 1888. [2] *Med. and Surg. Reporter*, Sept., 1889.

the most important contributions are those already mentioned, and especially the works of Moll,[1] von Krafft-Ebing,[2] and von Schrenck-Notzing.[3] The difference in the views of these writers has already been pointed out above when speaking of the pathology of the perversions in general. Besides the fact that the manifestations of contrary sexuality are acquired, von Schrenck-Notzing holds that, nevertheless, these manifestations become in time imperative sensations and imperative ideas, and thus from this point of view may be looked upon as psychoses artificially created, in a neuropathic soil in most instances. This opens a very wide field for discussion, as it is no easy matter to settle what decisive element constitutes an imperative idea. The familiar language of the pervert, which is stereotyped in "irresistible impulse," too often should be written, "I don't want to.[4]" Still, we must allow, as we see in the alcoholic and opium habit, that for weakened resisting powers sensations may be well educated to such an extent as to become imperative.

I have elsewhere[5] stated what appear to me to be the chief objections to the congenital or perversion theory as opposed to perversity, and, as I cannot more briefly express them, I may be permitted to repeat here what was said:

Now, putting aside hypotheses of the How, an examination of the congenital-perversion theory shows that it rests entirely upon the autobiographies of perverts and certain assumptions (to be presently mentioned) regarding the normal development of the vita sexualis, and of the tastes, habits, and modes of thought peculiar to each sex.

It is believed that a person is capable of remembering all the circumstances attending the gradual growth of the sexual functions in early childhood—has a distinct recollection of the causes which first called it forth, and that a failure to remember possible excitants is equivalent to their non-existence. A reliance upon evidence of this kind in any other department of human knowledge, whether medical or non-medical, I am sure, would only excite surprise. Even in taking an ordinary medical history we should hesitate to accept such testimony as final, and I think we should be even more cautious in our examination of autobiographies which attempt to give an analysis founded on introspection of the feelings, passions, and tastes of degenerate individuals who attempt to explain their first beginnings in early childhood and attribute each to its proper excitant. As von Schrenck-Notzing has pointed out in his careful study of the published cases, very few of these autobiographies will stand analysis. Probably there is no class of people whose statements will less stand the test of a searching cross-examination than the moral pervert. One cannot help feeling that if the pervert was thus examined by an independent observer, instead of being allowed to tell his own

[1] *Conträre Sexual Empfindung.*

[2] *Psychopathia Sexualis* and *Zur Erklärung der Conträren Sexual Empfindung.*

[3] *Suggestive Therapeutics in Psychopathia Sexualis.*

[4] "I wish to state expressly that, though I am conscious of the abnormality of my inclinations, I have no desire to change them; I long only for a time when more easily and with less danger of discovery I can give rein to my desires and experience a delight that will harm no one."—*Autobiography*, Case 149, v. K.-E.

[5] "Sexual Perversion or Vice?" *Journ. of Nerv. and Ment. Dis.*, 1898. I have made free use of this contribution for the purposes of this article.

story without interruption, a different tale would be told, or great gaps would be found which are now nicely bridged, or many asserted facts would be resolved into pure inferences.

Taking one point alone, it is extremely doubtful whether any one can remember the first beginnings of the vita sexualis. He may remember certain occasions which, from the special intensity of the excitation or from peculiar associations, persist as vivid mental pictures, just as we remember certain pleasurable experiences of boyhood connected with sports, but not all or the first.

The second error of those who maintain the congenital theory is that they overlook the influence which casual external circumstances have in suggesting feelings and ideas to the mind and in directing thoughts which appear to be spontaneous.[1] These external circumstances may be trivial or not, and may be forgotten. Even when very prominent for the moment in consciousness, they may be forgotten, while the effects may persist. The enlargement of our knowledge of the substrata of consciousness and the after-influence of such subconscious states upon the personality of the individual has made it possible for us to understand the genesis of certain neuroses which before were inexplicable. Janet has demonstrated "this influence in the productions of some of the manifestations of hysteria. With this knowledge it is next to impossible to say that sexual aberrations were not originally suggested by external conditions in individual cases or the product of auto-suggestion.[2] A very suggestive example of the influence of this kind upon the lower strata of consciousness in producing psychoses is the following from the writer's experience: A young girl about sixteen years old was pursued with an uncontrollable fear of vomiting. As a matter of fact, she never did vomit, but the fear was so intense that she was unwilling to leave the house alone, or, for that matter, even when accompanied, go to places like theatres or to such distances from home that she could not quickly reach her house. The fear, although always present, was subject to exacerbations. In such attacks her suffering was very great and the mental state uncontrollable. She would take off her clothes, and run up and down the room crying and begging her mother not to let her vomit. This fear had apparently developed spontaneously during early girlhood, and might easily have been considered congenital if the original history as given by the patient herself and mother had been believed. But from the mother, after persistent inquiry, I obtained the following history, till that moment forgotten. When the patient was a child, say five years old, her sister was taken ill with scarlet fever, the first symptom of which was violent vomiting. In order to prevent the child from catching the disease, she was told that if she went near her

[1] Whether or not a neuropathic taint is necessary, as has been maintained, is a secondary matter. The existence of an hereditary taint has, however, been sometimes accepted on insufficient evidence.

[2] A capital illustration of the influence of forgotten causes of producing psychical phenomena is the following: A lady told me of a dream which she had in which she saw distinctly the face of a person whom she had never seen. Her description of the person being very accurate, I insisted, to test the matter, that she must have seen or heard of the person before. On assuring me the impossibility of this, I told her, as was the fact, that a few days previously I had described this person to her, using the *same language* that she now used for the same description. She had no recollection of it. Sexual suggestions and excitants might be similarly forgotten.

sister she would be taken with vomiting in the same way. This had the desired effect, but when the sister recovered it was with some difficulty that my patient could be induced to come in her presence. She ran away and hid in a closet, exhibiting considerable fear. It is reasonable to suppose that the impression made upon the mind at that time had left a subconscious idea which was the cause of the apparently motiveless fear later exhibited. The patient has no memory of all this. The excitation of abnormal sexual feelings may well have similar external causes long since forgotten.

The third error of this school is that it assumes that normally there is a hard and sharp line drawn by nature between the normal personalities of the sexes. As a matter of fact, sharp lines of demarcation do not occur any more than in the length of the nose or size of the hand. Taking a large number of people, the male personality normally shades into the female, and *vice versâ*. What I mean to say is, that taking a large number of normal males and an equal number of normal females, we might place them in a row so that at one end would come the males, with strong vigorous masculine characters; in the middle, but at the extreme end of the male line, the men with feminine personalities; adjoining these the masculine females, differing but slightly, excepting in anatomical configuration, from the males; while at the extreme end of the female line would come those with strongly marked feminine characteristics.

Fourthly, the effect of education, meaning by this the total environment, intentional education, unconscious mimicry, external suggestion, example, etc., etc.—the effect of this, I repeat, in moulding the tastes and habits of thought and manners of the child, and thus differentiating those of one sex from those of the other, has been overlooked. I think it is extremely probable that if a boy were brought up as a girl and a girl as a boy, and absolutely freed from all counter-influences—such as the unconscious influence of public criticism, etc.—each would have the non-sexual tastes and manners of the other sex.

Fifthly, it is questionable whether only abnormally the vita sexualis of the male is excited by the female, and conversely. There is every reason to believe that in some perfectly healthy individuals some degree of erotic feeling or ideas may be excited by the sight or touch of the form of a person of the same sex, and, at any rate, thoughts (pertaining to anatomy) so excited may very naturally awaken secondarily associated sexual feelings. For instance, the vita sexualis in a boy is at first associated with his own sexual organs; later, the sight of those of another boy awakens this association of ideas by the well-known law, and then, in a degenerate, cultivation does the rest. Von Krafft-Ebing's very first case (106) of a girl with hyperæsthesia sexualis and homo-sexuality is readily explainable in this way. As von Krafft-Ebing points out, in the beginning of sexual development in the child "the psychical relation to persons of the opposite sex is still absolutely wanting, and the sexual acts during this period partake more or less of a reflex spinal nature." "With the inception of anatomical and functional development of the generative organs, and the differentiation of form belonging to each sex which goes hand in hand with it in the boy or girl, rudiments of a mental feeling corresponding with the sex are

developed; and in this, of course, education and external influences in general have a powerful effect upon the individual, who is now all attention." Now, in a person of perfectly healthy mind and body all social customs, habits of thought, unwritten laws, and moral precepts tend to suppress any existing homo-sexual feeling and its gratification, and to encourage hetero-sexual feeling. On the other hand, the person of tainted constitution does everything in his power to foster, indulge, and cultivate the perverse instinct, while in such a soil the feelings themselves acquire monstrous force. That the future development of this perversity is due to cultivation there is no question. We have only to read the autobiographies to be convinced of it. Thus may arise a perversity that had its origin in a normal reflex, but the accidental cause of which is forgotten with much else of the psychical life of childhood, or, if not forgotten, considered abnormal because of its future monstrous development. Such a reflex, it may be said, if normal, is congenital. This much is in strictness true, but an entirely different aspect is given to the congenital theory. What is really pathological in this aberration is the extraordinary intensification of the sexual feelings and the unbridled lack of restraint with which the subject indulges his senses and seeks every opportunity for gratification. These, without doubt, depend upon the neuropathic constitution. The contrast in this respect with normal hetero-sexual persons brings the difference into strong relief.

Finally, the fact must not be lost sight of—it is not questioned—that cultivation is capable of generating this aberration and developing it to its most intense degree, even to the feeling of repulsion for the opposite sex and to the acquisition of contrary tastes and habits. Acquired cases of this kind are recognized and illustrated by Cases 94, 95, 96, 99, etc. of von Krafft-Ebing. It is not, then, a question of the sufficiency of this influence. The only question is, Are all cases due to this influence, or are those cases in which there is no evidence in the histories, *so far as obtained*, of cultivation, and in which there is an apparent spontaneous origin, properly to be regarded as congenital?

One logical consequence of the cultivation theory has been overlooked, as it seems to me, by von Schrenck-Notzing. It follows as a necessary corollary that this so-called perversion is not really a perversion, but a perversity—a vice rather than a disease.

From one standpoint the view may be modified. It has already been said that a habit may be so intensely cultivated as to become in time almost automatic and independent of volitional control. The nervous processes involved may thus become shunted off from the rest of the psychical life as true psychoses. It is tenable that in some persons these aberrations may become by cultivation real imperative sensations and ideas. Though vice may be the road traversed, the final stage may be disease.

Analogy with what takes place in other fields of the nervous system would make it intelligible that sexual feelings and actions may by constant repetition (cultivation) become associated together, and developed into a sort of quasi-independent neural activities which may then become practically independent of the will, or, in other words, a psychosis.

Sexual perversion, then, may, from the point of view of pathogenesis,

be put in the same class with many of the manifestations of hysteria and other psycho-neuropathic states. The constant excitation of various bodily symptoms by the neurasthenic tends to cultivate them into imperative habits which control his organism. The hysteric, dwelling on certain ideas, whether they relate to herself or her environment, tends to nurture and cultivate them till they may acquire such monstrous intensification that they control her psychical life.

From small beginnings it is possible that even most intense doubts and fears may be evolved by this cultivation, culminating perhaps in imperative ideas (insanity of doubt, folie de toucher, etc.). By constant indulgence of her feelings, revelling in morbid retrospection, giving herself up to egotistical debauches, self-pity, and wrong inferences, the degenerate cultivates her body and mind into becoming such a sensitive machine that she can no longer adapt herself to her environment, but must be removed to an institution where her environment can be adapted to her: of course I am drawing an extreme picture, but such extreme pictures exist.

Therapeutically, the point of view which we take of the genesis of these psychoses, whether sexual or non-sexual, is of extreme importance. If they are the manifestations of a diseased nervous system in the sense that they are the necessary expression of a diseased body, whether congenital or not, then there is no escape from therapeutic hopelessness so long as the psychopathic state continues. But if psychoses of this kind are the result of cultivation, whether by the influence of external surroundings or by the subject's own conduct—cultivated into psychoses because the soil is a psychopathic one—then we may fairly hope by *counter*-education in many instances to replace the morbid processes by healthy ones.

The *manifestations* of contrary sexuality need only be briefly considered. In the great majority of instances an hereditary taint will be found. One or more members of the immediate family or ancestors will be found to have been affected with one of the neuroses or psychoses—hysteria, neurasthenia, hypochondriasis, alcoholism, or even some form of insanity. In bad cases several members of the family may have been affected. For example, in one case the father was a drinker and committed suicide; a sister had hysteria; a brother and sister also committed suicide; a maternal aunt was insane and committed suicide; the mother was sickly and died of apoplexy, and the patient had grave hysteria. On the other hand, in some cases no hereditary history can be obtained. The subjects of the perversion may be apparently strong and healthy, exhibiting perhaps only sexual weakness as the result of the perverse indulgence. But more often symptoms of neurasthenia or hysteria will be found. In the former case they pursue their vocation without giving rise, excepting by indiscretion, to any suspicion of their habits. Other individuals, again, may exhibit evidences of insanity, especially paranoia. As paranoiacs they may have hallucinations of actually being of the opposite sex, but then these hallucinations should not be classed as a part of contrary sexuality, but rather like any other hallucination of the insane. The neurasthenic condition may be primary or may appear later secondarily, as a result of perverse habits. As a rule, these people are given to onanism, which plays a part in the development of the aberra-

tion. The modes in which the perverse instinct finds expression are the same as in vice, and do not need description. The subjects give themselves up to the gratification of their sensual feelings without apparently feeling any moral obligation to control or suppress them. For this the presence of a hyperæsthesia sexualis, which commonly is present, and a lack of resisting power, are responsible. The latter is a result of the psychopathic constitution. When the erotic feelings are associated with certain ideas the pervert voluntarily gives himself up to the cultivation of the exciting thoughts, which thus grow by practice: a sort of ideational debauch may thus be indulged in. In this way intense feelings and thoughts may become uncontrollable, and the subject be unable to resist indulgence in them. Then they may be looked upon as *imperative*. They may come on periodically, with intermissions of freedom. In personality the subject of these aberrations may exhibit a tendency to femininity. The male shows a taste for dolls and girls' playthings and games, and later for feminine dress, ornaments, and occupations, such as crocheting, knitting, etc. The female may similarly exhibit masculine tastes. But this is not the rule, and beyond the sexual perversion there may be no change in personality relative to sex. It is open to question whether these tastes are secondary to the perversion or are not accidentally associated. With this perversion there may be psychical impotence for the opposite sex or not, or a person may be potent only by the help of contrary mental images. Impotence may therefore play a part in the domestic drama. Homo-sexuality may coexist with hetero-sexuality (psychical hermaphroditism), but in more highly developed cases only homo-sexuality may exist. In such cases the individual may feel all the longings and passionate feelings for a person of the same sex that normally are, as expressions of love, felt for an individual of the opposite sex. A person of the opposite sex may be sexually repelling and disgusting to such a person. Such a person, having deified the object of his love, feels all the jealousies, pangs of unrequited love, heart-burnings, etc. normally experienced in hetero-sexuality. These people are called urnings.[1] When in such people these feelings coexist with the above-mentioned tastes for the sports, dress, and occupations of the opposite sex, the appearance is created of complete change of sexual character (effemination and viraginity). The common expression of such people is that they feel as if they had a female soul in a male body, or *vice versâ*. These cases lend more support to the theory of congenital origin than ordinary hermaphroditism. But it must not be forgotten that the real perversion is that of the sexual instinct—that is to say, femininity without change of sexual instinct is not a perversion. The development of feminine or masculine tastes in male and female respectively in psychopathic individuals is easily accounted for by the facility with which fixed ideas and feelings take possession of such people. Cultivation is easy in such people. A somewhat fanciful attempt has been made to associate a bodily conformation resembling more or less that of the opposite sex (hips, breasts, deficient or abundant beard, features, voice, etc.) with homo-sexuality.

TREATMENT OF SEXUAL PSYCHOSES.—Far from being hopeless, as

[1] A good illustration will be found in the article on "Psychical Hermaphroditism," by W. L. Howard, M. D., *Alien. and Neurol.*, April, 1897.

the congenital theory would imply, the treatment of sexual paræsthesia is attended in a large proportion of cases with encouraging results, which contradict the congenital theory. When the sexual aberration is only a part of great central degeneration, such as imbecility, dementia, or paranoia, of course any attempt must be hopeless. But where the psychopathic basis is of a minor degree and the intellect is not materially affected, it must appear, if we are to judge by the reports of published cases, that improvement or cure may be accomplished. This of course presupposes that a person desires to be cured. It is highly improbable that a person can be cured against his will, and it is evident that many do not want to be cured. The chief and most effective therapeutic remedy is hypnotic suggestion. In the hands of von Schrenck-Notzing and others this remedy has given decidedly favorable results. The total number of cases collected by von Schrenck-Notzing is 32. The results of treatment were as follows:

Failures	5 = 15.625 per cent.
Slightly improved	4 = 12.5 " "
Essentially improved	11 = 34.375 " "
Cured, with later report, 10; without later report, 2	12 = 37.5 " "
	100 per cent.

Thus, about 70 per cent. were essentially improved or cured. The fact that of the 12 cures later reports were obtained, sometimes after considerable periods of time (four to five years), in 10 makes these statistics of considerable value. Of the 32 patients, 5 were not amenable to hypnosis, 7 were cases of psycho-sexual hermaphroditism, 20 of contrary sexual instinct, 2 of sadism, 3 of masochism.

Treatment must be prolonged. For instance, in 1 case one hundred and fifty-two, and in another, two hundred and four, sittings were necessary. Deep hypnosis is not always necessary. It is also undoubtedly true that in using hypnotic suggestion in this as in other psychoses much depends upon the way and form in which suggestions are given. Considerable judgment is required for this, and the manner and character of the physician count for much. That is, a "suggestion" given by one person will be effective, while from another, perhaps because of lack of confidence, it will be no suggestion at all or suggest the opposite. The earnest and faithful co-operation of the patient also counts for much. Without this it may be questioned whether success is attainable. Besides direct suggestion, other forms of mental therapeutics should be employed for the purpose of strengthening the will power and developing the character of the patient. He should be made to feel that the perverted instinct is one that should not be cultivated, and to wish not to do so. For this a sufficient motive should be given. The mode of doing this must be determined in each case by the physician according to the character of the person with whom he is dealing. The more the patient is under the personal influence of the physician the better. It is a great service if the latter can be a sort of moral confidant to whom the patient can almost daily turn for advice, help, and confession.

Finally, the physical health must be improved when neurasthenia is present. So long as a neurasthenic condition exists, ideas tend to fix themselves, and have an automaticity far in excess of what occurs in

well people. There will probably always remain a considerable proportion of patients who cannot, or do not want to, be cured. When distinct evidences of insanity are present an asylum may be the only and the best resort. When sadistic tendencies are exhibited the safety of the community may render this course imperative. The question of marriage will often be one for decision. Undoubtedly, as a therapeutic measure, marriage is often of the greatest benefit, and perhaps normal coitus may be essential to bring about a cure; but the patient is not the only person to be considered. The responsibility of a physician in recommending marriage is great, as this may mean the wrecking of other and innocent lives. Then, too, as perverts are usually by inheritance psycho- or neuropathic, marriage means the generation of more miserable stock and the probable perpetuation of nervous disease in some form. Although it is true that in some instances persons with sexual perversion have married and lived happily, still the risk is great, and, excepting in individual instances, marriage should not be advised, even though the pervert may gain thereby.

DISEASES OF THE MUSCLES.

DISEASES OF THE MUSCLES.

THOMSEN'S DISEASE; CONGENITAL PARAMYOTONIA; PRIMARY MULTIPLE MYOSITIS; MYOSITIS OSSIFICANS PROGRESSIVA.

By FREDERICK G. FINLEY, M. D.

THOMSEN'S DISEASE (MYOTONIA CONGENITA).

Definition.—A rare affection, usually showing strong heredity. It is marked by stiffness and rigidity of the voluntary muscles when first put into motion, and is named after the physician, himself a sufferer, who gave the first systematic description of the condition.

Etiology.—The disease shows a remarkable tendency to run in families. In Thomsen's[1] family it was present in five generations, and in a case of Erb's[2] it was present in four. Several members are usually affected, and although both sexes suffer, it is commoner in males. The symptoms usually appear in childhood from the fourth to the tenth year, but it may appear in infancy or be postponed until puberty or even later.

Other neuroses have been present in some families, and in a few cases the sufferers themselves have been dull and lacking in intelligence. A history of fright has preceded a few cases.

Pathology and Pathological Anatomy.—Déjerine and Sottas[3] have recently recorded the first autopsy performed in this affection. They found the brain, medulla, spinal cord, and peripheral nerves normal.

The condition of the muscles has been investigated by excising small fragments during life. The individual fibres are much increased in size, and the nuclei more numerous and slightly larger than in health. The fibres in transverse section are rounded or oval; some retain their polygonal shape, but the angles are rounded. When cut longitudinally, the transverse striæ are less distinct, the contours of the fibres are slightly irregular and curved, and vacuoles are often present. Déjerine and Sottas in an exhaustive study of numerous muscles regard the increase in nuclei as the earliest change, and this is followed by hypertrophy of the muscular fibres. Hypertrophy is more marked in muscles

[1] Thomsen: *Arch. v. Psychiatrie*, Bd. 6; *C. blatt für Nerven Krankheiten*, 1886.
[2] Erb: *Deut. Arch. fur. klin. Med.*, xlv.; *Die Thomsen sche Krankheit.*, Leipzig, 1886.
[3] Déjerine and Sottas: *Rév. de Méd.*, 1895.

like those of the calf, on which most work is thrown, and may, therefore, be regarded as of functional origin. Not only are the individual fibres enlarged, but the bulk of many of the muscles is considerably increased. Some of the fibres undergo a granular degeneration and disintegrate, leaving only a sarcolemma sheath. Other fibres atrophy and are represented by small rounded bundles. The intermuscular connective tissue in the more advanced stages is increased in thickness. Its participation is, however, secondary to changes in the muscle fibres, and no proliferation of nuclei or bloodvessels takes place. The affection is thus proved to be a primary myopathy. It resembles the other myopathies in its hereditary tendency and in the pseudo-hypertrophy of the muscles. There is, however, no tendency to steatosis.

Symptoms.—Although appearing in childhood, the symptoms often become prominent at puberty, when they are apt to undergo exacerbation.

A history is often obtained of the patients being unable to join in the usual games in childhood. The most characteristic feature of the disease is tonic spasm of the muscles when they are first put into motion. There is inability to overcome this spasm for a few seconds by voluntary exertion; relaxation then gradually occurs, and, after the movement has been repeated two or three times, the spasm passes off and only returns after a rest. The muscles of the limbs, trunk, neck, and rarely the face, tongue, and eyeballs may all suffer. The legs are most frequently affected, often with the trunk and arms. Difficulty is experienced as soon as the patient begins to walk, but after he is fairly started he can continue without special fatigue. A sudden change of gait or direction may, however, be again accompanied by spasm. Falls are not uncommon, owing to loss of muscular control when movement is first attempted. In one instance the sufferer could readily swing himself from a moving tram-car provided that he could walk from the far end of the car, but without these preliminary steps he invariably fell. When the jaw muscles are affected, the mouth may remain open until the spasm of the depressors relaxes and allows the internal pterygoid and masseter to act. In the face the eyelids sometimes become fixed, and strabismus has occurred from spasm of the muscles of the eyeballs. Contractions of the muscles of the face have been compared by Buzzard to the sardonic expression of trismus. If the abdominal muscles are affected, they are thrown into spasm on bending forward, but are unaffected in the movements involved by parturition or defecation. The degree of spasm is sometimes lessened by alcohol, digestion, or warmth. In one case it was only present in winter, passing off in summer. Excitement is apt to increase it. Thomsen believes that a life of active exertion rather tends to lessen the spasm. The muscles are well developed, sometimes even much enlarged, but the motor power is somewhat diminished, being rather less than in health. Erb has carefully investigated the mechanical and electrical irritability of the muscles and regards them as characteristic. The muscles when struck with the hand or by a percussion hammer contract, and the spasm may continue from fifteen to thirty seconds. With the faradic current the irritability of the nerves is normal, but with strong currents the muscular contraction is much prolonged and relaxes slowly. Applied directly to the

muscles, a shock with a weak current gives the usual lightning-like contraction, but a strong current produces a slow contraction with slow relaxation.

With the galvanic current the nerve irritability is qualitatively normal. With strong labial currents, the kathode being placed on the nerve, contraction is kept up and relaxes slowly. The muscles with a weak current react equally to C. C. C. or A. C. C. The contraction lasts longer than in health and relaxes slowly, and this phenomenon is more marked the stronger the current. In addition to the above "myotonic reaction" a peculiar wave-like contraction may be produced in the muscles, passing from the positive to the negative pole with an unbroken current. This phenomenon, however, is often difficult to obtain, and requires much perseverance and alteration in the strength of the current. Although abundantly confirmed, many observers have failed to obtain it. Sensation is unaffected, the reflexes are normal, and fibrillary twitchings are absent. Myographic tracings show the marked duration of the muscular contractions. The latent period, however, is unaffected.

Diagnosis.—The diagnosis rests on the character of the spasm, the myotonic reaction, and the hereditary character. It can hardly be confused with any other condition.

Course and Prognosis.—Once developed, the disease lasts throughout life, but is subject to occasional remissions. It is not known to shorten life, although producing much discomfort.

Treatment.—Erb recommends general faradization or galvanization and electric baths. Improvement is sometimes observed with these measures, but no case of cure is recorded. As already mentioned, active exertion seems to ameliorate the condition.

CONGENITAL PARAMYOTONIA.

Definition.—Under this name Eulenburg has described a peculiar hereditary and congenital condition characterized by tonic contraction of the muscles followed by temporary paralysis. It is somewhat allied to Thomsen's disease, although differing from it in its special features.

Etiology and Symptoms.—In the family described by Eulenburg the affection was traced through six generations. It sometimes declared itself at or shortly after birth, and was recognized by the mothers in a tight closure of the eyes. The muscles of the face and neck, and especially the orbicularis palpebrarum are affected. The hands and legs are subject to attacks of tonic contraction lasting from a few minutes to several hours, and are followed by weakness lasting for hours or for half a day. The attacks, which last throughout life, are induced by cold and relieved by warmth, a full meal, or a warm drink. They are not, however, induced by movement. The condition is transmitted by father or mother to son or daughter. In a case of Bernhardt's there had been

intermarriage of blood-relatives. The muscular waves and slow relaxation following electrical stimulation, which form a marked feature of Thomsen's disease, are here absent. The faradic and galvanic irritability of the muscles are diminished at the time of the attack, and there is a tendency to closing tetanus during the passage of the current. There is no change in the nerve irritability.

PRIMARY MULTIPLE MYOSITIS.

Synonyms.—Dermato-myositis; Pseudo-trichinosis.

Definition.—An inflammatory affection of the muscles and skin accompanied by fever, and probably of toxic origin.

Etiology.—The disease attacks both sexes, and no special age is exempt. The real cause of the disease is obscure. In one of Senator's cases it developed after eating stale shrimps, and he regards the condition as an auto-intoxication from the alimentary canal. Unlike multiple neuritis, it does not seem to be induced by alcohol, nor has cold or overexertion any influence in its production. In the lower animals certain gregarines produce a myositis, but these parasites have not been found in any of the recorded cases in man.

Pathological Anatomy.—Macroscopically the muscles may present no changes, but are sometimes rather paler than usual. Microscopically the essential feature consists in infiltration of leucocytes between the muscle fibres and frequently small hemorrhages. The muscle fibres are involved secondarily and vary in appearance. In Senator's case there was no trace of degeneration. In others granular, hyaline, and waxy degeneration has been present. The muscle nuclei are increased and the striæ obscured or lost. Pus formation has not been observed in the muscles, but in the subcutaneous tissue purulent foci may occur in addition to the constantly-present œdema.

The spinal cord and peripheral nerves are normal.

Symptoms.—The onset is usually rather gradual, and is ushered in by febrile disturbance, headache, and occasionally vomiting. At the same time or shortly afterward, pains appear in the muscles of the limbs and back, and they become hard, firm, and extremely tender on pressure. Other muscles may, however, be soft or almost fluctuating, and in chronic cases circumscribed nodular swellings have been noted. Movements are at first limited and later completely lost.

The arms are usually more severely and earlier affected than the legs; the thighs and arms suffer more than the peripheral portions of the limbs. The muscles of the neck and back are usually involved somewhat later. More important, however, is interference with the muscles of deglutition and respiration, interfering with their functions and producing a marked tendency to a fatal issue.

The muscles of the face, tongue, and eyeballs are occasionally attacked. In Strümpell's case ptosis, and in Hepp's paralysis of the soft palate, occurred.

In advanced cases muscular irritability to the galvanic and faradic

currents is lost, but an electrical examination is often unsatisfactory, owing to the pain produced and to the swelling of the subcutaneous tissues.

Œdema of the skin in the neighborhood of the affected muscles is a prominent symptom, and is similar to that observed in trichinosis. Inflammatory redness is also often present, and has been mistaken for erysipelas. Urticaria, labial herpes, roseola-like eruptions, and subcutaneous hemorrhages also occur, and there is commonly profuse sweating. Angina, stomatitis, and profuse salivation are occasionally seen, and slight splenic enlargement is often present. The urine sometimes contains a trace of albumin, and in one of Senator's cases there was acute nephritis. The disease runs an acute or chronic course. The former is accompanied by higher fever, and the constitutional disturbance is consequently more severe. Most cases terminate fatally, but there are a few instances of recovery in both acute and chronic forms. Death has occurred as early as the twelfth day, and again the affection has lasted for over a year.

Complication.—Broncho-pneumonia, induced by inhalation of bacteria from the mouth, and by paralysis of the respiratory muscles, is not infrequent as a terminal event.

Diagnosis.—Clinically the disease bears a close resemblance to trichinosis in the presence of swelling and tenderness of the muscles, in the subcutaneous œdema, in the cutaneous eruptions and sweating. An absolute distinction can only be made by examination of a piece of excised muscle. Marked gastro-intestinal disturbance and a history of exposure are points in favor of trichinosis. Multiple peripheral neuritis also presents some features closely resembling the disease in question. Pain, tenderness of the muscles, paralysis, sweating, and occasionally œdema may be present in both. In myositis, however, the œdema is more marked, and is often accompanied by inflammatory redness, and there is no disturbance of sensation. A history of alcoholism or the presence of anæsthesia would suggest neuritis. The reaction of degeneration, if present, would also favor neuritis.

Treatment.—The treatment is symptomatic and directed to alleviating the pain and tenderness in the muscles. If there is any reason to believe that an intestinal toxin is being absorbed, it might be advisable to use intestinal antiseptics and purgatives.

MYOSITIS OSSIFICANS PROGRESSIVA.

Etiology.—The etiology is obscure. More than half the cases begin before puberty, and of these the majority occur in early childhood. The onset, however, may be postponed until after middle life. It is four times more common in males than females. Some of the cases have followed injuries, others have been attributed to cold or overexertion. The disease is not hereditary, and does not attack more than one member of a family.

Pathological Anatomy.—Although usually referred to as a mus-

cular disease, the process seems to start from the bones (Virchow) or muscle sheaths (Birsch-Hirschwald), involving the muscles secondarily. Many of the bones are thickened by an overgrowth of bone which may involve the skull and the bones of the face and extremities. Irregular deposits in the form of exostoses are also present on the ribs, pelvis, scapulæ, vertebræ, humerus, and femur. Bridges of bone frequently unite the vertebræ, rendering them rigid and immobile, and even the scapula and ilium have been connected in this way. The bones of the lower leg and forearm commonly escape or are but slightly affected, and the hands and feet remain free. Many of the muscles are replaced by bone, sometimes lying free in irregular plates or again attached to the skeleton. The muscles of the neck, back, arm, and thigh are especially apt to suffer. The muscular fibres themselves are much atrophied or fatty, whilst the muscle sheaths may be thickened. The joint surfaces remain free, although movement is much curtailed by overlapping masses of bone. Microscopically the ossified parts consist of true bone.

Symptoms.—The affection commonly begins about the scapula, where hard bony masses develop in the muscles. Some are free and movable plates, others fixed and attached to the skeleton. These gradually involve the various muscles of the back and neck, forming prominent bony arches and bosses, and frequently those of the axillary folds, the serratus magnus, the intercostals, the thigh, and arm. Movements are much interfered with by the rigidity of the muscles. The jaw, which is affected in a considerable number of cases, may be closed so as to require the removal of the teeth for the taking of food. The spine and head are also more or less fixed. Exceptionally there may be pain and tenderness at the onset of the bony growths. In some instances soft doughy swellings have preceded the bony formations.

The disease is progressive, gradually advancing with periods of intermission. It lasts many years and has no influence on the duration of life.

In quite a number of instances the great toe and thumb have presented the conditions of either microdactylia or hallux valgus, but their relationship to the disease is unknown.

Diagnosis.—Localized bony growths sometimes occur in muscles from irritation, but these have no tendency to progress and involve other muscles. Ossification in the adductors of cavalry soldiers and the bony plates which occurred in the deltoids of the old Prussian infantry are well-known examples.

Prognosis and Treatment.—The disease is progressive and incurable. No form of treatment has hitherto proved of service.

MISCELLANEOUS.

OSTEOMALACIA.

By FREDERICK G. FINLEY.

Definition.—The essential feature of the disease consists in softening of the bones and consequent deformity.

Etiology.—The real cause of this remarkable affection is unknown. It occurs most commonly in those parts of Germany bordering on the Rhine and in Northern Italy. It is found, however, in every other country in Europe, but in North America Dock was able to collect records of only ten cases. Its endemic occurrence has suggested an origin in some peculiarity of climate or soil, a theory which is scarcely borne out by its widespread distribution. The disease occurs almost exclusively in females during the childbearing period. Isolated cases occur in men, but are extremely rare. In the great majority of cases the disease first develops during pregnancy or lactation, and exacerbations usually occur during these periods. Most cases begin between the ages of thirty and forty. Its occurrence in children is questionable, and it seems not improbable that in them rickets has been mistaken for the affection in question. Allison has, however, recently recorded a case commencing in a girl of thirteen. A senile form of the disease has been described, differing somewhat, however, from the puerperal form in its anatomical characters. Individuals in all classes of society may suffer from the disease, but it appears to be favored by damp and unsanitary surroundings.

Kleinwachter found that in Bukowina the disease was almost exclusively confined to the Hebrew race, and attributes this to their filthy and unhealthy surroundings, as well as to the fact that lactation is prolonged for one and a half to two years. In Japan, however, where lactation is kept up for three or four years, the disease is unknown. Various theories have been put forward to account for the disease, but none of them are satisfactory. Lactic acid has been found in the bones, and solution of the lime salts has been attributed to this substance. It has, however, been conclusively shown that the acid may be in excess without producing the disease, and efforts to produce the malady in the lower animals by feeding them with lactic acid have signally failed. Micro-organisms again have been carefully searched for, but with no constant result, and there is no ground for believing that bacteria are instrumental in producing this condition. The disease has been attributed to affections of the nerve centres. It has been observed that it is more common in lunatic asylums than elsewhere. Anatomical observations, however, on the nerve centres are very scanty and inconclusive.

The beneficial results following removal of the ovaries have suggested

that the condition is due to a reflex irritation of the centres presiding over nutrition.

Pathological Anatomy.—In advanced cases the bones are almost completely decalcified. They bend readily and are easily cut with a knife. In the long bones the medullary cavity is expanded and the wall thinned, ultimately being converted to a mere shell of bone. The periosteum is thickened, and when stripped off, the underlying bone is rough and often perforated by openings from which the marrow escapes. Irregular thickening of the bones is also often seen. The bone marrow is either yellow or red, the latter being probably connected with more active changes in the bone. Hemorrhages in the marrow with the formation of cysts are not uncommon. Fractures are of common occurrence, especially in the ribs and femur, and that an attempt at regeneration is present is shown by the formation of a soft porous callus. In the short and flat bones the medullary spaces coalesce and form larger cavities.

Microscopically, the bone salts are removed from the neighborhood of the Haversian canals, and osteoclasts are observed as in normal bone when absorption is going on. Inflammation, so far as it is evidenced by infiltration in the bone or marrow, is absent. Marked deformities result from softening of the bones. The acetabula are pressed in, and the projecting pubic arch gives the pelvis a beaked appearance. The sacrum also sinks in between the ilia and approaches the pubes. The femurs are arched outward, the neck shortened, and the head often is below the trochanter. Various forms of spinal curvature occur; the chest is flattened laterally, its antero-posterior diameter increased, and the ribs and sternum are much distorted. The bones of the arm are free from marked deformity, owing to the absence of pressure, but softening of the clavicle allows the weight of the limb to rest on the thorax, and a corresponding depression on the wall of the chest results. The height of the skeleton is considerably diminished, owing to the curvatures in the spine and legs and the depression of the head of the femur. Although the muscles have been found healthy, yet increase of the nuclei and other changes resembling those of muscular atrophy have been noted. Fatty changes have also been found.

Symptoms.—The earliest symptom is pain in the pelvis, spine, and thighs. It is of a more or less constant character, often of much severity, and is increased by movements. An important feature of the pain is that it usually starts in the latter part of pregnancy, ceases after delivery, and recurs with subsequent pregnancies. The bones themselves are tender to the touch. Weakness in the muscles of the thighs and pelvis is often an early symptom, and may be present before any bony deformity sets in. Attacks of painful spasm are sometimes present, particularly in the adductors of the thighs, coming on spontaneously or induced by slight irritation. Owing to weakness of the ilio-psoas muscle, the trunk is thrown from side to side, to enable the foot to clear the ground in walking, and a peculiar waddling gait results. With the advance of the disease deformities of the bones occur, and the patient ultimately becomes bedridden. The bones in the advanced stages of the disease become yielding, elastic, and capable of being bent in almost any direction. A feature very noticeable to the patient is diminution in

stature, a point on which Strümpell lays considerable stress in the diagnosis of the condition. In a case of Trousseau's the stature diminished from 1 metre 78 cm. to 1 metre, but the patient afterwards regained 43 cm.

Increased knee jerks are almost constantly present and often ankle clonus: occurring early and associated with muscular weakness or spasm the condition may simulate, and has been mistaken for, disease of the spinal cord. Women affected by the disease are stated to be more fruitful than others. Eisenhart found the average number of children born in Germany to be 3.9, whereas it was 6.4 in the sufferers from this disease. Abortion is also more frequent. Neusser has found myelocytes and an increase of eosinophilous cells in the blood, and Von Jaksch has noted a diminished alkalinity. The course of the disease is essentially a chronic one; most cases last for years and undergo remission and exacerbation. Pregnancy has a most deleterious influence, always lighting up a fresh attack.

The urine has been subjected to frequent examinations, but no very constant changes have been found.

Diagnosis.—Previous to the occurrence of deformity the diagnosis of the disease may be obscure. A history of recurring attacks of pain in the pelvis during pregnancy, with tenderness of the bones, is suggestive, and a diminution in stature is again very characteristic. Mistakes in diagnosis occur usually more from a want of familiarity than from any obscurity in the nature of the disease. It has most frequently been mistaken for rheumatism, an error which, by careful consideration of the history and features of the case, is usually avoidable. From spinal disease the distinguishing features must be sought in the bone tenderness and deformity. The increased reflexes usually serve to distinguish the condition from peripheral neuritis. There are instances of diffuse infiltration of bones with malignant growths simulating osteomalacia. According to Köhler, these growths may be distinguished by occurring only in the bones of the trunk, whilst the increased elasticity of the bones and the muscular weakness are absent. A history of a primary tumor is sometimes obtained and points to secondary infection of the bones.

Prognosis.—Most cases gradually progress, although frequent instances of improvement under various forms of treatment are recorded. Much depends upon the possibility of preventing conception.

Treatment.—Of late years the treatment of this disease has become much more hopeful.

Sternburg strongly recommends phosphorus; four cases treated by him showed marked improvement. The dose should begin at $\frac{1}{60}$ gr. twice daily, and may be gradually increased.

Trousseau strongly advocated cod-liver oil, and there is a reason to believe that improvement may take place under a purely expectant plan with good hygienic surroundings.

Removal of the ovaries or Porro's operation has been performed in a number of cases with very encouraging results. Either of these operations fulfils two conditions, preventing further pregnancies and apparently often arresting the disease. Improvement sets in sometimes surprisingly early, the pains being relieved within forty-eight hours.

Many cases from being bedridden recover so far as to walk and perform their ordinary duties. Of 44 cases collected by Baumann in which Porro's operation was performed, 18 died and 26 recovered. Of the latter, 3 died from other diseases and 2 were lost sight of. Of the remaining 21 cases, 17 were cured or markedly improved.

Winckel reports the case of a woman bedridden for years, who after castration recovered and performed the arduous duties of a nurse in a German hospital. Of 40 cases collected by this writer, the after history of 16 was traced, 12 being cured and 4 improved.

RHACHITIS.

By A. JACOBI, M. D.

Definition and Pathology.—Rhachitis is best understood when considered as a general affection of all parts of the organism. It is true that it is usually recognized by alterations in the osseous tissue, but in many cases it is not even the bones that exhibit the first morbid changes. The general condition of the body, the muscles, the viscera, to a certain extent the blood also, is suffering, not equally it is true, not always simultaneously, not to the same degree in all cases, but there are very few that do not show alterations beyond those in the bones. That is why so many different theories have been advanced with a view of explaining the nature of the disease, and why pathological physiology, chemistry, neurology, even bacteriology, have been searched for the explanation of all its symptoms.

The normal bone grows in length and in thickness. The former depends on the formation of new tissue in the "zone of proliferation" between the epiphysis and the diaphysis, the latter on the same process on the inside of the periosteum; absorption takes place in the interior with a tendency to widen the medullary canal. In the newly deposited tissue ossification takes place readily as long as there is no disease. In the rhachitical bone the formation of some new tissue and the absorption of the old take place normally; it is ossification that is defective. Excessive absorption, which at one time was considered sufficient for an explanation of the rhachitical process, is not so any longer. The anomalous process is mostly taken to be a parenchymatous inflammation; some, like Pommer[1] and Tedeschi, look upon it as a nutritive disorder lit up by neurotic influences originating outside the skeleton. According to Stern, the rhachitical bone grows like other bones. The disease, however, manifests itself in the absence of calcium as long as the nutritive disorder is kept up by disorders of the nervous system; and the irritation in certain parts of the skeleton, the cartilage proliferation near the joints, and the periosteal thickenings are but secondary consequences of pressure, muscular traction, and other external or traumatic influences.

Guerin, Virchow, Kassowitz, Baginsky, and others favor the theory of a *parenchymatous inflammation*. It is particularly Virchow (1853) and Kassowitz (1881–84) who insisted upon the necessity of assuming the presence of disturbances in the circulating blood, whose effect would be mostly noticed wherever the physiological functions of blood and tissues were most active; that is, where growth happened to be most intense. Such parts are, besides the fœtal and infant brain, the bones,

[1] *Researches upon Osteitis and Rhachitis*, Leipzig, 1885.

and mainly the zones of proliferation between the epiphyses and the shafts, and under the periostea. It is here that intense hyperæmia will set in, with the result of rendering the tissues red, soft, and succulent, and of giving rise to intense and irregular cell proliferation and deficient —in the later periods of impeded circulation superabundant—calcium deposit. That increased cell proliferation extends into and beyond the ossification line, which is straight and narrow in the normal and becomes diffuse and broad in the rhachitical bone. At the same time, the medullary spaces filled with medullary cells extend into the conglomerates of cartilage cells. In all of these, after a while, calcium is deposited in a short time and irregularly, and the newly-formed cells are not, as is normally required, mostly first transformed into cartilage before becoming bone, but the transformation of cartilage cells into bone corpuscles is a direct one. This "metaplastic ossification," which is a limited one in the normal bone, is universal in the rhachitical.

Which are the causes of this parenchymatous inflammation, or rather, what is the source of the circulating irritation? Wegner could produce it by feeding animals on minute doses of phosphorus. But clinical observation cannot be so positive as the experimental proof.

I shall consider in some of the following pages to what extent bad air, improper food, infectious diseases, chemical changes of the blood, or the presence of microbes may have their irritating and deteriorating effects.

The *physical* and *chemical* composition of rhachitical bones is considerably altered. Friedleben found an increase of water and of fat, the latter mainly in cases in which the rarefaction of the tissue persisted. The proportion of organic and inorganic parts is reversed: while the latter are 65 per cent. in the normal, the former are 65 in the rhachitical bone. Still, in places there may be found a good deal of calcium in the rhachitical bones, mainly in the epiphyses. In such it is not so much the deposit that is at fault, but its adaptation for normal ossification.

What is it that results in the *diminution of phosphate of lime* in the rhachitical bone? Is it its relative absence from the system by either insufficient ingestion or insufficient absorption? or is there a local disorder in the growing bone which prevents deposit of calcium? or are both at fault? And if proliferation and dilatation of bloodvessels, which is so manifest about the affected parts, have anything to do with defective ossification, are they primary (Kassowitz) or secondary?

Ingesta are certainly not at fault unless we have to deal with downright cases of starvation, and these do not become rhachitical. Woman's milk, and still more cow's milk, also farinacea, contain a great deal of calcium, the milk of the mothers of rhachitical children as much as that of others (Seemann); and, according to Voit, the ingested lime exceeds always that which is deposited on or required by the bones. The experimental findings of Guerin, Chossat, and Roloff, who deprived animals of phosphate of lime in their food, do not prove much, for what they accomplished was mere fragility of bones, but not rhachitis. Still, it has been suggested that this and forced abstinence may be able to create, though not rhachitis, a disposition to rhachitis. If that be so in some cases, it cannot be the rule, for, as I said, starving babies do not generally become rhachitical.

Nor can deficiency of absorption be at fault. When babies, healthy or rhachitical, are overfed on phosphate of lime a single day, there is at once more elimination of it, both through the kidneys and the intestines. In the latter it is not merely an overflow, for more is found in the lower than in the upper part of the alimentary tract, exactly like iron, which behaves in the same way; that is, it is first absorbed, and then eliminated again. To digest milk very little hydrochloric acid is required. If the latter (or chloride of sodium) be present in fair quantities, a goodly amount of the salt of the food is easily dissolved and absorbed in the upper part of the intestines, and just as quickly eliminated in the lower. The circulating blood does not carry overflow material any length of time. If it did, it would soon become decomposed, indeed would be destroyed sooner than this floating strange material could be expected to exert a lasting influence on solid tissues. It has been claimed that the acid condition of the blood explained the absence of calcium from the tissues. It was principally Charles Heitzmann in 1873 and Baginsky in 1879 who claimed lactic acid to be the solvent of lime in the bones, and thus to become the cause of rhachitis. But fever, anæmia, diabetes, with their acidulated blood, produced no rhachitis. Circulating acids would immediately be counteracted by the circulating alkalies. Indeed, Siedamgrotzki found loss of salts, but no rhachitis, under such circumstances, and if tolerated at all, any quantity of lactic acid flowing in the blood sufficiently to wash out lime from the tissues would first destroy life. It was suggested that possibly from a cause hitherto unknown some nascent acid of local origin could exert a local effect (lately again by Vierordt), but no proof has been forthcoming. It is not impossible that the many causes actually known to produce rhachitis act primarily on some organ with "internal secretion," which may still be found the anatomical and physiological source of rhachitis. What we know, for instance, of the connection of osteomalacia with the ovary makes our thoughts run in some such direction.

As long as the actual cause (or causes) of rhachitis is not understood we have to admit that in regard to the etiology of rhachitis we have not advanced beyond the knowledge possessed by our predecessors. The syphilis theory of Parrot and the nerve theory of Pommer, who took rhachitis to be the result of some trophic disorder depending on nerve derangements, are undoubtedly incorrect. The microbic theory of Mircoli and others is certainly far from being proved. The broader theory of a general nutritive disorder resulting from many sources will be the subject of our studies in the course of these pages.

The MACROSCOPICAL CHANGES of the rhachitical bones are very characteristic. The bones are red, vascular, though not uniformly so; their peri-epiphyseal (ossification) cartilage is bluish and very copious and broad, with irregular and indefinite outlines; their consistency is altered; they are soft; the periosteum is thickened, red, and œdematous, closely attached to the loosened bone, so that when pulled off it may tear pieces of bone with it; periosteal bloodvessels are seen to enter the bone directly; the epiphyses are thick, soft, and compressible; the frontal and parietal bones are thickened, still more so the tibia. The new deposits, besides the periosteum, are quite thick, from one to ten millimetres and more, and so soft as not to resist the knife; the bones when

cut bleed. In the parietal and occipital bones there are open spaces of ½ to 1½ or more centimetres in diameter, sometimes in, sometimes adjoining, the soft deposits; transparent when held to the light. The medullary canals of the long bones are deformed, compressed in places, and narrow. The long bones are deformed in different ways—by deposits on their surface, by curvatures, by subperiosteal infractions, and by fractures. After recovery the bones are short, hard, sclerotic, thick, and more or less curved. In a few cases in which the process of softening is very intense and fat is deposited in the dilated areoli, porosis takes the place of sclerosis. In later life such an unsubstantial and light bone, contrary to the general condition of recovered rhachitical bone, is very liable to fracture on the slightest provocation.

ETIOLOGY.—*Heredity.*—A single observation of a rhachitical fœtus born of a mother with rhachitical pelvis, ought not to militate against the fact that hereditary transmission through semen or ovum has not been proven. If, as some few claim, rhachitis were a bacteric affection, the microbe would have to be carried through the placental circulation like syphilis. Charrin and Gley, who placed small animals under the influence of tuberculosis, diphtheria, and bacillus pyocyaneus, think they saw rhachitical anomalies in the offspring. It is, however, more probable that the circumstances under which the two generations of parents and infants live are the cause of rhachitis in all. Thoroughly rhachitical parents who can raise their infants in favorable surroundings do not transmit their own rhachitis. General debility, poverty, alcoholism, syphilis of the parents, are predisposing causes. Besides, a debilitated, ill-fed, tubercular, or syphilitic infant is apt to contract rhachitis. Boerhaave claimed rhachitis to be almost exclusively the result of hereditary syphilis. This view was strongly opposed by Van Swieten. In our time Kassowitz has taken up Boerhaave's position, and Parrot still more so. Being connected with a service teeming both with syphilis and rhachitis, he asserted the actual identity of rhachitis and hereditary syphilis. That identity does not exist. Cazin and Icovesco[1] studied carefully 109 rhachitic infants. In them syphilis was not more frequent than in the average infants observed. The influence of any and every constitutional disease of the parents or the general condition of the offspring is liable to cause a disposition to rhachitis. Syphilis is very frequent in Africa, China, and Japan, all of which countries are notorious for the almost entire absence of rhachitis.

Geographical Distribution.—Rhachitis is pre-eminently a disease of the zone of Central Europe, and also, at present, of the United States and Canada. In Europe, Holland, France, Germany, Hungary, Russia, and Northern Italy it is very common. In Switzerland it is found everywhere (Feer), more in the industrial than in the rural districts, and, parallel to the density of the population in general, mainly in the valleys. But the highest Alps have no immunity. In these higher altitudes the children of immigrants from the valleys appear to suffer more than those whose parents were native to the soil.

In the far North rhachitis is rare. In Norway, Iceland, Finland, it is said to be scarce (though in Riga, according to Mey, it is universal). In the cold climates more oxygen is used, more carbonic acid exhaled,

[1] *Archiv. gen. de Med.*, Sept. and Oct., 1887.

the blood contains more cells and more hæmoglobin. At an altitude of six thousand feet Egger found 7,000,000 blood cells in the c.mm. In high altitudes and on frozen soil the direct influence of the sun is stronger. On Mont Blanc the atmosphere absorbs 6 per cent. of the solar warmth only; in the plains, 30 or 40 per cent. Besides, there are more sunny days above than below.

Tropical climates have no rhachitis. It is unknown or rare in Africa, Central America, Central Asia, Australia, China, Japan. I was astonished by the absence of curvatures and rhachitis in general amongst the half-naked, dirty, and neglected children of Naples and Greece. It appears their constant exposure to air and sun protects them. Negroes in Africa have no rhachitis, in the United States, a great deal—in Washington, D. C., according to Acker, it is frequent and often complicated with tuberculosis and syphilis.

Age.—As a rule, rhachitical infants are presented for observation when one or more symptoms are quite prominent. That is why the statements as to the age in which rhachitis takes its origin varies so much. There are those who take it for granted that all cases (Unruh), others that from 11.5 (Quislong) to 80.6 per cent. (Schwarz) of all cases, are congenital. They claim the rosary swellings of the costo-cartilaginous junctures and craniotabes for them at birth. In the next few months these anomalies are said to develop more and more. If that be so, all the alleged or actual causes of rhachitis, such as foul air, improper food, could not exist unless as concomitants. Ritter von Rittershain, one of the most honest and observing pedologists that ever lived, coincides but partly with the above statements. Still, he asserts that many cases date from intra-uterine life. Kassowitz placed the origin of 50 per cent. of rhachitis in the first two months. There are many fœtal cases, partly amongst stillbirths. Vierordt extends the time from the fourth month to the end of the second year. On the other hand, V. A. Hoffmann declares rhachitis to be rare in the first half year of life. Pini charges 2974 of his 4176 cases to the second year, but does not take account of either cranial bones or muscles. There can be no doubt that all of these statements are made conscientiously. They vary according to opportunities, seasons, climates, walks of life, and many other circumstances. I find rhachitis sometimes congenital, frequently before the end of the second month, very often before the end of the fifth or sixth, and the first symptoms rarely after the completion of the first year. It is most frequent during the time of rapid development of the bones. Thus it is that the cranium is liable to be affected first when rhachitis develops early.

Air.—Bad air and darkness are amongst the most important causes of rhachitis. Moleschott proved an increase in the elimination of carbonic acid with the amount of light striking the skin and retina. Deep-sea animals, though they belong to the bone fish varieties, have a cartilaginous skeleton. After respiratory disorders rhachitis is often observed. It is true, however, that impeded respiration may be accompanied or followed by all sorts of constitutional anomalies. Animals imprisoned in badly ventilated stables become rhachitical. Soldiers' children locked up in their bad barracks in the East Indies are rhachitical (Spencer Watson), though those of the poor outside the barracks

are not. Large industrial cities have more rhachitis than the country. Still, J. P. West[1] denies the absence of rhachitis from the country districts, and refers to the fact that mild cases are but rarely mentioned at all. In every village of his neighborhood he found signs of rhachitis evident. In nine counties surrounding his residence there were, to his knowledge, eight rhachitical dwarfs, three of whom were members of well-to-do Scotch families. The factory population is mainly affected. Nor are the well-to-do immune. In Riga the well-to-do suffer like the poor: 86 per cent. of the infants are rhachitical because of the long duration of the winter—eight months—during which the infants are kept at home. Most cases are, with us, observed in the spring, the babies having been housed all winter. There are many less, even after the prevalence of summer diarrhœa, in the autumn or toward the end of the year. It has often been observed that, not only in Naples and Athens, those badly fed will thrive when constantly in the open air.

Hygiene and Nutrition.—Hygiene and nutrition are of great influence. There is less rhachitis in rich or well-to-do families than among the poor. There are very many exceptions to this rule from different causes. Bad foods (with, according to Comby and D'Espine, dilatation of the stomach and toxic absorption), such as large percentages of amylum or undiluted cow's milk, are dangerous; but when babies are fed on the same faulty diet in the valleys and on the high Alps, those on the latter suffer less from rhachitis: it is particularly undiluted milk that is better tolerated on the high mountains than in the valleys. In the favorable climate and outdoor life of Athens, where the babies are mostly weaned after the second month and farinacea given, rhachitis, as I said, is not frequent. Breast children suffer less from rhachitis than those raised on artificial food; still, there are plenty of babies whose breast milk contains an undue degree of casein that require weaning, and will require a well-selected artificial food in order to get well of their rhachitis. Marked absence of fat from the food is also liable to produce rhachitis. Cheadle refers to the fact that rhachitical animals of zoological gardens get well when fed on fat meat. The small amount of sodium chloride compared with potassium in vegetables is the cause of salt hunger of animals and of the necessity of adding salt to cereals, potatoes, and cow's milk. It is required to counteract the superabundance of potassium and furnish a sufficient amount of hydrochloric acid in the stomach, which is required for digestion in general, and mainly for the solution of the calcium of the ingesta. Potatoes require a good deal of chloride of sodium. Wagner attributes the rhachitis of poor people, Stockfleth that of the swine in Sweden, to the influence of potato feeding. Still, there was no potato feeding at the time when Glisson wrote on rhachitis (1650).

The charge that amylaceous foods in general, no matter how given or in what qualities, must needs be the cause of rhachitis, is exaggerated. This belief is the result of a fanatical insistence on the dogma that no young infant can digest any starch, no matter in what combination. The teachings of Zweifel and Korowin, proving the very contrary, have been assiduously neglected this quarter of a century, and my addition of cereal decoctions to cow's milk has found as many loud or silent

[1] *University Med. Mag.*, Oct., 1895.

adversaries as disciples. They feed the babies on rice in China and Japan, on vegetables of many kinds in Central Africa. In China breast feeding is rare.[1] There they have no or very little rhachitis.

Uncleanliness has been accused of being the cause of rhachitis, but the poor peasant and the Chinaman are very unclean, and the cases of rhachitis amongst them do not increase with their dirtiness. But this much is true: that uncleanliness, bad dwellings, the foul air of tenements and factories, and improper food go very often together, and, combined with the frequency of respiratory and digestive disorders, will result in rhachitis.

Pulmonary catarrh, narrow dwellings, bad seasons, and bad air generally have been charged with acting through the accumulation of carbonic acid in the blood. But cyanotic children are probably less subject to rhachitis than those not suffering from congenital cardiac anomalies.

Sex does not appear to influence the prevalence of rhachitis: I find as many rhachitical girls as boys.

Twin children are liable to be rhachitical because of insufficiency of food and incompetent attention to cleanliness, air, and so on.

Organic infection has been either suggested or assumed to be the cause of rhachitis. Boerhaave and Ritter von Rittershain (1863) believed the majority of cases to be of syphilitic origin; Parrot (1879), all of them. Friedleben (1860) claimed intestinal ferment, Oppenheimer malaria, to be important etiological factors; Kassowitz believed in the activity of microbes; Volland, in that of a specific virus in causing rhachitis; Chaumier,[2] in epidemic contagious germs that remained in dwellings. Hagenbach-Burckhardt[3] refers to the facility with which tuberculosis and syphilis locate in bones, and to the swelling of the spleen so common in all infectious diseases, as also in rhachitis. Mircoli[4] found in the bones of two rhachitical children staphylococcus pyogenes, and in four cases the same and streptococcus without other pathological lesions, and therefore concludes that they are the causes of the diseases. This conclusion appears premature in the light of other experience. Marfan and Marot[5] found bacterium coli commune and streptococcus in the blood during enteritis; Czerny and Moser,[6] under similar circumstances, staphylococcus aureus and albus, the bacterium coli commune, and the bacillus lactis aërogenes; and Smaniotto Ettore[7] bacillus coli commune and other microbes in septicæmia; the latter were found in the bones also. Indeed, the intense hyperæmia of the epiphyses and of the adjoining cartilage explains why microbes may be found in these tissues, though they need not be anything but accidental. Nor are the experiments of Charrin and Gley more conclusive. On February 22, 1896, they presented before the Biological Society of Paris rabbits which became rhachitical after the parents had been fed on toxins of diphtheria and bacillus pyocyaneus, but were not prepared to claim a direct connection with rhachitis—perhaps an indirect one with enteritis only. Lange suggests that some infecting agent may be attached to certain regions or climates, similarly to cretinism (which is mostly—by no

[1] Lange: *Verhandl. d. 12, Vers. d. Gesellsch. f. Kinderh. in Lübeck*, 1895, p. 144.
[2] *Med. Infantile*, 1894.
[3] *Berl. klin. Woch.*, 1895.
[4] *Gaz. d. Osped.*, 1891.
[5] *Revue mens.*, 1893.
[6] *Jahrb. f. Kind.*, vol. 38.
[7] *Revue mens.*, Mar., 1897.

means always—found in circumscribed territories with mineralogical anomalies), because of the fact that in Iceland, Finland, Northern China, and Japan, and Northeastern Siberia, where dwellings are very inferior and circumstances by no means favorable, rhachitis is scarce.

Symptoms and Complications.—*The Bones.*—The principal causes of *rhachitical changes in the bones* are numerous—viz. the rapid growth, the thick epiphyses, the soft diaphyses, the condition of the ossification cartilage, the traction of the muscles, the debility of the muscles, and the pressure of the atmosphere. The locality where anomalies are found depends largely upon the intensity of growth. This is most active in the young child—first in the cranium, second in the chest, and lastly in the extremities. In the normal infant the two lateral (temporal) anterior fontanelles, and the two lateral (lambdoid) posterior fontanelles disappear within two or three, the small (parieto-lambdoid) fontanelle within four or five, the large (parieto-frontal) within fifteen, months. In rhachitis they remain open many months, the large sometimes for two, three, four, or even nine years, as I have seen it. As a rule, they are larger than normal. Very frequently it is easy to hear over them a systolic murmur, which, however, is not positively pathognomonic of rhachitis. It is probably the result of the increased width of the artery, which is compressed in the stenosed carotic canal. The edges of the sutures are irregular. Such a head is frequently actually larger than the normal head. Relatively, it is very much larger when compared with the usually small body. This is so, though both the longitudinal and the transverse diameters are but slightly increased. In rare instances the rhachitical head is so large that it resembles the hydrocephalic head. Indeed, some of these heads are to a certain degree hydrocephalic; some are entirely so. Still, it must be remembered that there are many rhachitical heads of normal size. Most of them are brachycephalic, quadrangular, rather flattened on top. In a peculiar class of cases, first studied by Virchow, viz. that of the cretins and semi-cretins, rhachitis, which in these cases begins in fœtal life, is combined with a premature ossification of the occipito-sphenoidal synchondrosis. In this condition the base of the skull is shortened. At the same time there is a deep grooving of the root of the nose, the eyes are widely separated from each other, there are shortening of the vomer, and a flat palate. Not infrequently the occiput is slightly flattened, and the oblique diameters are sometimes not equal, so that one side may appear to be entirely flattened. This is particularly so in the rhachitical softening of the cranial bones —*craniotabes.*

In addition to the six fontanelles whose patency was attributed to the retarded formation of osseous tissue at the edges of the cranial bones or to the softening of such as are already calcified, the rhachitical cranium may exhibit any number of more or less marked perforations. These are particularly characteristic of craniotabes. Mostly in the parietal bones, sometimes in the occipital, rarely in the frontal, where I once saw more than a dozen in a prematurely born rhachitical baby of a mother with a thoroughly rhachitical pelvis, there are a number of spots of the diameter of one half to one and a half centimetres, mostly with steep margins, transparent, without any osseous tissue left, and giving way under gentle pressure of the finger like a sheet of paper or thin

cardboard. James Tyson[1] is mistaken when he says: "As craniotabes occurs in connection with syphilis and other wasting diseases in infants a few weeks old exhibiting no other signs of rhachitis, and in newborn infants, it cannot be regarded as pathognomonic," and characterizes craniotabes as "large areas of delayed ossification in the parieto-occipital regions, producing yielding spots." Neither elsewhere nor in the cranium does the rhachitical process consist in delayed ossification. On the contrary, the rhachitical bone is that which was developed normally, and when the child is taken sick is normally absorbed and abnormally reconstituted. The bone thus softened is easily and locally absorbed by pressure, this working both from inside and outside—that is, from brain and pillow. The most characteristic feature of craniotabes for clinical purposes is the presence of soft spots, not along the sutures, but at some distance from them within an area of more or less normal bone. The transparent defects in the bone may have margins of different nature. They may be sloping or quite steep. In this way the rhachitical cranium may be readily diagnosticated from congenital aplasia or from the effects of syphilis.

When craniotabes makes its appearance between the first and third months of life there is much perspiration, particularly of the head, with loss of hair on the occiput. The veins are more dilated, the skin thinner and paler than on the average head, the scalp is very sensitive, the babies cry when laid down, feel better when they are taken up or when they are lying on their faces. In these cases of craniotabes one side may be flattened, usually the one which will be found more softened, and the other bulging. The head may even appear to be triangular, where one side bulges out, and one side is flattened from pressure on the pillow; the forehead is very prominent, the frontal bone sometimes from three to five times its normal thickness, because of the immense amount of new periosteal soft growth between the periosteum and the bones. There is sometimes a groove in front of the coronal suture, which results from this steep thickening of the frontal bone. This is not always a temporary affair. It is true that craniotabes may leave no trace if it gets well speedily. But when there is much deposit under the periosteum it will sometimes remain. When calcification takes place very suddenly, then thickening of the bone will remain unabsorbed for life. As a rule, however, most of such thickenings are absorbed.

The *teeth* appear late or irregularly, often but one instead of two at a time. When they are early the intervals between the first couple and the second or between the second and the third are very long—sometimes six, eight, or ten months. The teeth are frequently discolored, and they decay very easily because of the absence of cement. Sometimes, however, after recovery from rickets the permanent teeth are very hard and rather yellow. Not infrequently do we see in the temporary teeth of rhachitical children what may be compared with the Hutchinson form of syphilitic permanent teeth. Longitudinal groovings and semilunar furrowing of the incisors are quite common. This is one of the reasons why Parrot got the idea of explaining every case of rickets as the result of syphilis.

The *lower jaws* are short, narrow, and very low, flattened in the

[1] *Practice of Medicine*, 1897, p. 780.

middle by the contracture of the muscle on the floor of the mouth. The angles are very sharp; therefore the space for the teeth is cramped and dentition may become difficult. While the angles of the lower jaw are prominent, the alveolar processes turn inward. Thus the teeth of the upper jaw do not correspond with those of the lower jaw, and the space for the teeth becomes still more insufficient.

The *chin* in some cases is very low. In the maxilla superior the alveolar processes of the posterior parts are pushed out; the anterior part becomes triangular and prominent. Thus the upper incisors stand out very much beyond the lower. Sometimes the maxilla is asymmetrical. In many cases there is a depression of the fossa canina. In them the corresponding incisors are still more prominent.

The *trunk* in rhachitical patients is very short. The clavicle shows much periosteal thickening. It is very frequently bent forward by the pulling of the muscles, and there is not infrequently an infraction between the middle and anterior thirds.

The *chest* develops its rhachitis generally later than the skull, but earlier than the extremities. It is frequently triangular, sometimes quadrangular. The dorsum is flat, and the scapula clings to the body. The ribs being soft, form a groove in which the arms are frequently buried. During inspiration the ribs are caving in, giving way under the pressure of the atmosphere. There is a predominance anteriorly on account of the lateral atmospheric pressure. Laterally, above the diaphragm, not corresponding with the diaphragm—for this extends from the ensiform process to the twelfth rib, and both liver and spleen prevent the ribs from moving inward—there is a horizontal depression called *Harrison's groove*. As there is compression by the atmosphere above the diaphragm, the lower ribs, which are thinner and have no direct connection with the sternum, stand outward. As the chest is compressed laterally, the sternum is made to protrude, particularly about the level of the third and fourth ribs, and the antero-posterior diameter of the thorax is lengthened. The ribs are prominent at the ossification point. Frequently there are nodulations on the cartilages. A complete rosary may thus be developed quite early, mostly from the second to the eighth ribs. I have seen it repeatedly at the age of two months, and a case has been published in which there was a complete rosary in a babe of only three weeks. In these extreme cases the sternum is flat and the manubrium stands out. Sometimes it is pressed down above so as to stand out at an angle at its lower end, or the lower end of the sternum may be retracted while the ensiform process protrudes. In accordance with the changed condition of the chest the intrathoracic space is reduced. The softening of the ribs does not permit complete dilatation on inspiration, and thus the danger of suffering from pulmonary affection is very great on the part of rhachitic children.

Kyphosis is very frequently seen in these cases, but it is less angular than in Pott's disease; it is rather more arch-like. In the beginning kyphosis is more functional. By bending the body in the opposite direction it disappears. Scoliosis has mostly its convexity to the right, with compensation above and below. The spinous processes are very frequently directed to the concavity. The intercostal spaces are very

narrow on the left side, because there is less curvature of the ribs, and the ribs are bent out.

The Pelvis.—In the grown-up rhachitical woman the antero-posterior diameter of the pelvis is shortened. This is not seen to the same extent in the babe. In a normal infant the pelvis is small and the sacrum very steep, not concave as in the adult. Therefore, when compression has taken place because of softening, it is still smaller, so that often it is quite difficult to examine the pelvis satisfactorily. The sacrum may be so changed as to exhibit its convexity inward and a contraction of the two sides. This narrowing may be due to the mere fact that the softened bones are compressed on the pillow or by the arms of the nurse —a pressure which is slight, it is true, but quite sufficient. In very mild cases the symphysis is changed but little. In a number of instances, however, it will be found to be bent forward, and thus in very early rhachitis the rhachitic pelvis is very similar to the pelvis deformed by osteomalacia. This is contrary to the usual description in the books on obstetrics.

The *extremities* suffer in different ways in all their parts—the epiphyses and diaphyses, the periosteum, and the epiphyseal cartilages. The epiphysis is frequently thick and painful, particularly on the forearm and tibia. A number of cases of so-called "growing pains" are simply instances of rhachitic epiphysitis. Sometimes the thickening is very considerable; in most cases it is uniform, but in some it is more developed laterally. This is particularly the case on the upper part of the thigh. The diaphysis is usually bent. Semi-fractures take place in the arm, clavicle, and legs from a very trifling application of force. The periosteum, however, being soft, always acts as a shield to the inflamed bone when exposed to the danger of fracturing. In all those cases in which there is much curvature, particularly in the lower extremity, the concavity is inward, and on the forearm and thighs it is very often anteriorly. The difference in the direction of the curvatures depends on the influence of the muscular traction or of the weight of the body. In the very young the concavity of the lower extremity is inward because of the effect of the flexor muscles. When the bones become or remain soft in those who attempt to walk, the weight of the body results in forward curvatures and in lesions of many kinds.

The *ligaments* are very flabby: they may be hyperæmic, inflamed, and painful up to an advanced age, even to adolescence, and give rise to talipes valgus, also to genu valgum, less to varum in children that stand up and attempt walking. The periosteum suffers a great deal and in different ways. It is softened and exhibits a thick layer of rhachitic deposit. Calcification occurs in time, and then the diaphyses will be much thicker and harder than in normal conditions. The bones of rhachitic patients when recovery is completed are solid and able to stand a great deal of hardship.

In the rhachitical periosteum there may be *hemorrhages*. Not infrequently, and in those bad cases which in the course of general ill-nutrition, perhaps with the co-operation of a microbic invasion, develop purpura, they are found under the periosteum in the lower and upper extremities. These anomalies and complications will be treated of below under the heading of Acute Rhachitis and Scurvy.

Deformities consisting of shortening of the whole limb are due to the early calcification of the epiphyseal cartilage. As is known, it is on its physiological function that the length of the diaphysis depends. When calcification is complete both the growth of the bone and that of the limb ceases.

Nervous System.—Symptoms referable to the nervous system are quite frequent in rhachitical infants. The tangible head symptoms of craniotabes, with its hyperæmia and occasional actual effusion, cause cerebral disorders. *Convulsions*, either general or local, about the eye (both *strabismus* and *nystagmus*), or in the fingers, tonic or (mostly) clonic, severe or very mild, are quite common. Even the slight local irritation of dentition may suffice for a convulsion. In most cases of nervous troubles from gastro-intestinal causes the diagnosis may readily be made. After the latter have been excluded rhachitis takes its prominent place in disorders of the nervous system of the very young. *Insomnia* is frequent. Many sleep better when carried about, because cerebral congestion is lessened in the erect posture. As soon as the patient is laid down his brain becomes hyperæmic; the child wakes up, and makes night hideous. Such children have *night-terrors* ("pavor nocturnus"), with which they wake up, or have long *crying spells* with the expression of fright, or are irritable, peevish, or morose and ill-tempered, sometimes to a degree of moral or intellectual insanity. The suspicion of rhachitis ought to be aroused when babies perspire copiously, mainly on the head. They rub their heads on the pillow to such an extent as to cause baldness on the occiput. Not all of these symptoms need be present at the same time to justify or to suspect the diagnosis.

Other disorders of the nervous system are hypertrophy of the brain, hydrocephalus, laryngismus stridulus (crowing inspiration), tetany, Trousseau's symptom, nystagmus, and spasmus nutans, and katalepsy.

Hypertrophy is rare, and far from always being rhachitical. It produces symptoms of pressure. In autopsies the brain bulges out when the cranium is opened; it is pale, rather hard, and the convolutions are flattened.

Hydrocephalus of different degrees is not infrequent in rhachitis. It results from the sluggishness of arterial circulation and from the presence of intense hyperæmia in the cranial bones and the meninges. The former is due to the relatively large size of the arteries and to the feebleness of muscular contraction all over the body; which, therefore, is but an insufficient excitant of the immense peripherous circulation. In craniotabes there is intense hyperæmia, both in the bones and in the meninges and the brain. Effusions, therefore, are frequent. Small ones are easily absorbed; not infrequently will their absorption, after recovery, give rise to renewed growth of the brain. Like the bones, which, after recovery, are more solid and stronger than normal ones, the brains of rhachitical children appear to develop very favorably; the large square heads characterized by their combination with short and thick-set limbs, often belong to the best scholars at school and to the most enduring intellectual and physical workers among adults. Thus, to a certain extent, the intense hyperæmia of rhachitis in some instances is rather propitious than otherwise. But if this hyperæmia

lasts long and be excessive, so as to disturb nutrition permanently, the resulting effusion will persist and actual hydrocephalus follow to a greater or less degree. How to differentiate between this and "genuine" hydrocephalus from other causes will be taught below under the heading of Diagnosis (p. 942).

The first stage of *laryngismus stridulus*, which consists of many very short inspirations or more frequently of complete apnœa, is followed by a long, crowing inspiration. Before the latter takes place, brought on by the accumulation of carbonic acid in, or absence of oxygen from, the nerve centre of the medulla oblongata, death may occur; but that is not a frequent occurrence. Sometimes an attack of laryngismus is accompanied by a general convulsion. The large majority of cases are connected with, or rather dependent on, cranial rhachitis with its hyperæmia and effusion. Still, this almost universal experience is doubted by some. But lately Loos ridicules the idea of the dependence of laryngismus on rhachitis, for the reason that "laryngismus is mostly observed in the early spring, while rhachitis is not limited by any season." Unfortunately for the writer, rhachitis knows better and behaves differently. It is most commonly observed in the spring, after the babies have been housed during the long, dreary winter months, with less oxygen and more carbon oxide than is compatible with their welfare.

There are, however, cases of laryngismus, with or without sudden death, which are not caused by rhachitis. An occasional actual hypertrophy of the thymus does occur without rhachitis; but in rhachitis enlargement of the thymus is not uncommon. I published such cases, my own and some others, in a paper on the anatomy and pathology of the thymus gland, which appeared in the third volume of the *Transactions of the Association of American Physicians*. Besides, there are those in which laryngismus stridulus is observed together with rhachitis, but the former continues after the latter is cured. Such is the case of Kassowitz, who relieved rhachitis with phosphorus, but not the accompanying laryngismus stridulus; also one of Canali, who found that antirhachitical treatment did not relieve laryngismus stridulus. In that case it was complicated with or depended on hydrocephalus. But these cases form a small minority only: 19 out of 20, probably 49 out of 50, are of rhachitical origin. They are met with at the age at which cranial rhachitis is common, mostly before the ninth month. That is the time in which the growth of the cranium and of the brain is most intense: the physiological development is therefore apt to become on slight provocation pathological, and the hyperæmia of the bones and meninges, with its tendency to effusion, predisposes to secondary physical and mental disorders. There is, moreover, a special centre in the frontal lobe which, when irritated, causes bilateral adduction of the vocal cords, with complete obstruction, and another one in close proximity which causes interruption of breathing during expiration.

Many cases of *tetany* are of rhachitical origin: that does not mean, however, that many rhachitical cases exhibit this nervous derangement amongst its consequences or constituents.

In tetany there is flexion in the carpal joints. The thumb is turned in, the forefingers are extended in the interphalangeal, and flexed in the metacarpo-phalangeal, joints. There is talipes equinus, sometimes

flexion of the forearm; the neck is stiff and reclined, the muscles are rigid in the contracted extremities. The attacks are intermittent, seldom last for hours or days; consciousness is intact; the paroxysms last from a few minutes to a few days, and disappear mostly after a few months. Its nervous symptoms share with rhachitis this peculiarity, that they do not interfere with the intellect, and run a limited, though irregular, course, with a strong tendency to recover.

Tetany is credited by many with three symptoms which have been claimed as pathognomonic:[1] 1. That which is called after the name of Erb, and consists of increased electrical (more galvanic than faradic, and more motor than sensitive) excitability. 2. Trousseau's symptom; that is, compression of the vasomotor brachial plexus or pressure in the popliteal space causes an attack of tetany, either through anæmia produced by local pressure, or, rather, by irritation of nerves. 3. Chvostek's so-called facial symptom; that is, pressure on, or tapping or sometimes even gentle friction of, the zygomatic arch in front of the ramus mandibulæ produces a spasm of the upper lid and alæ nasi. Much has been made of the last symptom; perhaps too much.

But a number of authors on the subject of tetany have missed that last symptom in many instances. In a case seen by me but lately it was absent. On the other hand, it may be present sometimes in the healthy, both old and young, mainly the latter, in epileptic, neurasthenic, hysterical, and chlorotic persons. If that be so—or rather as that is so—tetany and the facial symptom are no longer identical. At all events, those rhachitical children who exhibit both Chvostek's and Trousseau's symptoms must not, as it has been done lately, be charged with having had "latent tetany." Indeed, a disease must be very "latent" that has no symptoms except a single one whose significance is very doubtful. The symptoms belong to rhachitis when present, but not to tetany proper. Indeed, cases of complicated rhachitis, particularly such as are developed early and exhibit more or less craniotabes, that yield both Trousseau's and Chvostek's symptoms are not infrequent. Kassowitz recalls 172 rhachitical children suffering from nervous symptoms. In 108 there was laryngismus stridulus, in 120 the facial phenomenon of Chvostek, in 41 the pressure phenomenon of Trousseau, in 19 tetany. Thus, rhachitis, I may add from my own observation, mainly cranial rhachitis, may be found complicated with tetany. That is why one of the disorders depending on cranial rhachitis—viz. laryngismus stridulus—is occasionally met with in, or complicated with, tetany.[2]

Spasmus nutans (*salaam spasm*), a rare enough disease, consists in rhythmical convulsions of the sterno-cleido-mastoid and of the rotatores capitis, and exhibits a nodding and rotatory movement. It is often complicated with nystagmus of one or both eyeballs, and sometimes of the eyelids, also with strabismus and with petit mal. Convulsions are an uncommon complication. The etiology is not always clear. Falls on the head are not infrequently charged with being the cause. Many

[1] S. Sterling: *Arch. f. Kinderheilk.*, vol. xx., 1896, p. 1.

[2] This occasional coexistence of tetany and laryngismus stridulus could not escape as shrewd and consistent an observer as Escherich. What he, however, emphasizes solely is that he found the facial phenomenon in many, and Trousseau's symptoms in some, infants suffering from laryngismus stridulus. This appears to him a sufficient reason for claiming the latter as a symptom of tetany, and not of rhachitis.

cases in which head and eye movements are combined point to a defective equilibrium in the special cerebral centre. Rhachitis has often been accused of being the cause, and has just as often been denied. I know that the vast majority of cases seen by me were rhachitical. In some of the oldest notes I possess I have added the remark: Rhachitical. At least one half of all my cases occurred in the second half of the first year, when rhachitis is very frequent. In exceptional instances only the babies were two years old. Craniotabes was not noticed very often. Still, at that time, the latter diagnosis was frequently not made by me, for I limited it about the time I wrote my first paper on that subject[1] to those cases in which softened spots were distinctly felt in the midst of otherwise ossified bones. That is incorrect, or rather unduly restricted, for craniotabes may exist without those well-marked, circumscribed, softened islets.

Kataleptic symptoms, such as Epstein described,[2] I have observed in a few instances. My cases were those of craniotabic, rather fat, placid, rather sleepy, indolent babies, who, however, exhibited no symptoms of imbecility or idiocy. When they recovered, after many weeks, they had rather an unusual amount of a quiet docility. While they showed their symptoms, these consisted in the characteristic involuntary tonic contraction of the muscles of the fingers and toes and the rest of the upper and lower extremities after having been given a certain position. This symptom is rare, still more defined than what I observed in a few instances in which I am not quite certain of an explanation. A baby may appear faint, ashy pale, sometimes cyanotic, and recover immediately. In these cases there was no history of laryngismus or of heart diseases. None of the few cases of mine that gave rise to serious anxiety died. It struck me that they might be local symptoms belonging to the medulla oblongata, which Betz[3] tried to explain by rhachitical flabbiness of the ligaments of the cervix.

These symptoms are quite rare; others are more frequent; many quite common in rhachitis when it affects the nervous system. The absence of some does not militate against their rhachitical origin. No disease should be expected to exhibit in every individual case every possible symptom. No typhoid fever, no pneumonia, no fracture does so. Least of all, affections of the brain, which are as various as the thousand localities which may be subject to changes of substance, of circulation, and of physiological influences. The various manifestations of rhachitis need not, do not, all show themselves at the same time.

Muscles.—The muscles of the newly-born and of the infant are feeble. Their total weight compared with that of the adult is 1 to 40, while the relation of the skeleton is 1 to 26. It takes some time before the dynamometric effect of the child's muscles will increase. This takes place after the sixth year, but even then it is only temporary and not persistent, nor is the sustaining and persistent strength of the muscle satisfactory, perhaps in consequence of the relative absence in the rhachitical bone of phosphoric acid and calcium in its tissue. Experience certainly does not prove that weakness and atrophy of the muscles result from uneasiness or pain in any number of cases. The muscles in

[1] *Journal of Obstetrics*, 1873. [2] *Prager med. Woch.*, 1896, Nos. 43, 44.
[3] *Memorabilien*, 1887.

rhachitis are pale, and now and then fat is found deposited between the fibrillæ, the microscopical structure of which appears to be normal. Nor is there a chemical change, though olein was missed by Jenner. The frequency of traumatic joint inflammation in infancy and early childhood results in part from the incompetency of muscular resistance. Falls are very frequent, for the joints are flabby, the muscles being elongated as in poliomyelitis. It is not necessary to adopt as proven the assertion of Kassowitz that the ligamentous insertions are always inflamed. Squinting is common in small children simply because their muscles of accommodation and motion are insufficient, and more in rhachitical children than in normal ones. Scoliosis is frequent in infants and children not suffering from or affected by any ailment except rhachitis. "Growing pains" are often muscular only and the result of over-exertion. This is much more evident in those suffering from rhachitis. What has been called rhachitical pseudo-paralysis by Berg and others is but a confirmation of the fact that the muscular structure is insufficient. This condition may be universal, or only a certain number or combination of muscles are the principal sufferers. With strabismus, or sometimes without it, nystagmus, being in such cases the result of insufficient accommodation, is mostly noticed to be bilateral. In many of these exclusively muscular cases there is a motion of the head in the same or opposite direction. An instructive case of the kind was published by Caillé some years ago.

It is true that rhachitical children are apt to be very strong after recovery, but the pressure they are subjected to in our schools, the expensiveness of fresh air in large cities, and the exposure to in-door life one half or more of the year in our climate, allow, when the spring does come, but little Easter resurrection to our rhachitis-stricken little ones. Habitual scoliosis of the very young up to the tenth year is almost exclusively muscular, mostly dorsal, sometimes lumbar, with the convexity usually to the left. Rhachitical babies, particularly when carried on one arm only, are very liable to grow scoliotic. Even when sitting in their chairs, not supported by their own strength, they topple over in one direction or the other. Even before scoliosis occurs there is a general flabbiness of the muscles which prevents free sitting altogether, and causes an apparent or actual kyphosis. The latter, however, is easily distinguished from the spondylitic kyphosis, inasmuch as it does not exhibit the same angular shape. As long as no bones participate in the deformity, the diagnosis can be easily made between that caused by weakness and that resulting from actual bone disease. By raising the baby's heels and hips while the chest and chin are supported the apparent curvature depending on the weakened muscles will instantly disappear, and lordosis rather than kyphosis will be observed.

The feebleness of the infant's muscles when intensified by rhachitical malnutrition is evidenced by nothing better than the symptoms connected with the *insufficiency of intestinal muscular tissue* in early life, which is exemplified in different ways.

One of the reasons why, for instance, renal disorders are not at all uncommon in the intestinal diseases of early life (the others being, as I have shown but lately in a paper on "Nephritis of the Newly-born,"[1]

[1] *New York Med. Journ.*, January, 1896.

the disproportion of the large renal arteries and the small capillaries and the large size of the intestinal vessels and villi), is the feebleness of the intestinal muscle in the young, which is less capable of expelling decomposing fæces and toxins. This feebleness of the muscles shows itself, however, still more frequently in another symptom, which is quite common in rhachitical children, and even pathognomonic for early rhachitis. Take an infant born at full term, with good weight, breast-fed, and apparently well and increasing in weight. The bowels are reported to have been regular for four, six or eight weeks, then they become costive. There is no apparent change; the baby does not appear to be sick, is perhaps a little quieter and paler. Even the early cranial changes of rhachitis are sometimes not so perceptible as to be positive evidence of that disease. But this *constipation* is. Go on with the same feeding, air and other surroundings being the same, and more symptoms of rhachitis will soon develop.

In order to be certain of the diagnosis of rhachitis from this symptom every other cause of constipation ought to be capable of exclusion; for instance, chronic colitis and peritonitis, deficient or viscid mucus, local atrophy of the intestinal muscle, or stricture of the intestine, perhaps even—though they be excessively rare—cystic tumors. The apparent constipation which results from insufficient feeding, either intentional or not, and resulting in starvation; the superabundance of casein in milk, of starch in artificial food; the relative absence of sugar; hardened fæces in the colon; hydrocephalus and other causes of defective innervation; the drying up of the intestinal contents by excessive perspiration during the hot summer months, in hot rooms, under heavy clothing, or by diabetes insipidus,—after excluding all these possible causes of constipation we shall not fail to make the diagnosis of rhachitis, which will possibly be confirmed by other symptoms if we wait a short while, or, rather—and that is to be preferred—by the effect of the antirhachitical treatment.

It is, however, not the feebleness of the intestinal muscles alone that gives rise to constipation. The debility of the muscles of the abdominal walls contributes much to that effect, as the two together give rise to the flabbiness and inflation of the abdomen of rhachitical babies. Some deformity of the abdomen, however, may be due to the spleen, liver, or kidneys. When "Harrison's groove" is marked the liver and spleen are liable to be displaced, and for that reason these organs may appear larger than they really should. The kidneys may also be dislodged. Most cases of floating kidneys met with at an early age are found in children who were or had been rhachitical.

The differential diagnosis is also to be made from that variety of constipation which I have termed "congenital constipation" in the same article[1] in which I discussed rhachitical constipation. The former begins at birth, the latter in the second or third month of life.[2]

[1] *Journ. of Obstetrics*, 1860.

[2] In annual lectures and occasional papers, even in a discourse before the Eleventh International Congress, I have referred to the same subject because of its scientific and practical importance. It was not heeded much until some years ago, when foreign publications took up the subject; for instance, Marfan, in an essay published in *Arch. gén. de Méd.* a few years ago. The facts are briefly as follows:

The embryological intestine is formed in separate divisions. There is no ascending

In several instances many years ago I subjected to the faradic and the galvanic currents rhachitical children of the second year who could walk. It appeared to me that when they were tired the electrical current, to have any effect, had to be increased. But, indeed, such patients at that early age are not favorable subjects for experimentation.

Muscular incompetency is very apt to influence the general circulation. It would show itself in all those muscles of larger size which have muscular layers, but principally in the insufficiency of muscular activity all over the body. The sluggishness of rhachitical children in general, and some of the brain symptoms to which allusion has been made, are thereby explained.

Blood and Circulation.—Anæmia, or rather hydræmia, is common in rhachitis. Not to speak of the emaciated babies whose rhachitis developed in or after severe intestinal or pulmonary disorders, the fat, rotund, and flabby variety of rachitis exhibits also a pale skin and mucous membrane. Luzet found a diminution of red blood cells within three weeks from 2,110,000 to 1,596,000; Von Jaksch, within three months, from 1,600,000 to 750,000 in the c.mm. Hock and Schlesinger met with an average number of 2,500,000 red cells in a c.mm. of blood. In all of these cases the percentage of hæmoglobin seems diminished to 30 or 60 per cent., and the number of leucocytes increased to 25,000 or 36,000. This increase is caused by polynuclear and neutrophile cells, and does not depend on complications, for instance, with intestinal disorders. Hayem found only 685,000 red cells in an infant of two months; still, recovery took place. Cabot[1] met in one case of rickets with myelocytes which are mostly found in the blood of malignant disease, but do not justify through their mere presence an absolutely bad prognosis. Lymphocytosis is prevalent, but, as Cabot[2] justly remarks, it is to a certain extent the normal condition of an infant's blood, provided that mononuclear (young) leucocytes are not in such excess as in a case of Rieder's, where 75 out of 100 leucocytes were of that nature. In his "Study of the Blood in Rickets"[3] John Lovett Morse arrived at the following conclusions: "Most cases of rhachitis are accompanied by anæmia. This anæmia may be, or may not be, accompanied by leucocytosis. Leucocytosis occurs more fre-

colon before the fourth or fifth month of fœtal life. In the newly born it is very short. In spite of this, the large intestine of the mature fœtus is longer in proportion than that of the adult. In the new-born it is three times, in the adult only twice, as long as the body. This is the same proportion that is found for the small intestine, this being twelve times as long as the body in the newly born, eight times as long in the adult.

The ascending colon being very short, the surplus of length belongs to the descending colon, especially to the sigmoid flexure. As the pelvis is very narrow, the great length of the lower part of the large intestine causes multiple flexures instead of the single sigmoid flexure of the adult; consequently, now and then two or three flexures may be found overlapping and compressing each other. One of them is quite often found in the right side of the pelvis, and not at all as an anomaly, as Cruveilhier and Sappey thought. Huguier found it in that location so often that he proposed to operate for artificial anus on the right side in small infants.

This great length of the colon and the multiplicity of its flexures retard the movement of the intestinal contents, facilitate the absorption of fluids, and render the fæces solid. Time and again have I been compelled to manually remove hardened fæces from the recta of babies otherwise normal and fed exclusively on normal breast milk.

[1] *A Guide to the Clinical Examination of the Blood*, New York, 1897, p. 105.

[2] *Op. cit.*, p. 280.

[3] *Medical and Surgical Reports of the Boston City Hospital*, 1897.

quently in the cases with splenic tumor than in those without. It may be due to an increase of any or all of the varieties of white corpuscles. The specific gravity varies with the amount of hæmoglobin. Finally, there is no form of anæmia found in rhachitis which may not be found in other conditions, and no form of anæmia found in other conditions which may not be found in rhachitis."

There is no general constitutional disease but interferes with normal sanguinification. The condition of the blood is naturally impaired by all those diseases which alter the lymph bodies or large glands. Great tumefaction of the spleen—not in every instance, but often observed in rhachitis, sometimes congestive only, frequently hyperplastic—will always affect the blood and may result in leucocytosis. This may be quite dangerous, though it be not made manifest by an early leucocythæmic habitus. Rhachitis, in my experience, was several times complicated with pseudo-leucæmia. Indeed, those frequent cases of rhachitis which exhibit many lymph bodies in a condition of swelling and hyperplasia ought to have the blood cells counted and be examined for macro-, micro-, and poikilocytosis. These cases will improve but little, if at all, with iron. Arsenic does better, but, best of all, a thorough anti-rhachitical treatment with proper diet and phosphorus.

Almost every anæmia is liable to increase the function of the *bone marrow*. Thus, according to E. Neumann,[1] pathological hyperplasias and an occasional leucæmia are explained. Litten and Otto came to the same conclusion by experimental research. Indeed, bone marrow undergoes a lymphoid change in all serious diseases. How much more readily with such an alteration take place in rhachitis, where the most perceptible changes affect the osseous tissue!

In rhachitis the heart is mostly of normal size, while the arteries are large. The result of this latter condition is a low blood pressure, particularly in distant parts and where normally the circulation becomes slow. Is that the reason or one of the reasons why hyperæmia sets in in the epiphyses? If that be so, a very moderate irritant flowing in the circulation ought to suffice to get up the whole series of changes constituting what we call rhachitis, at least as far as the bones are concerned. In it we have to deal with an abnormally increased vascularization of the ossifying tissues, diminished or abolished deposition of lime, or even softening of parts already calcified, and that exorbitant proliferation, through augmentation of epiphyseal and subperiosteal cartilage cells, of the ossifying tissues. Thus, indeed, the whole rhachitical process should be called physiological, but for its intensity and duration sufficient to render the formation of bone irregular.

The size of the arteries resulting in slackened circulation contributes also to the swelling of the viscera, predisposed as they are by the succulence and expansibility of their tissues (liver, spleen, tongue). It is not, however, the size of the arteries alone which has that result. In all probability the feebleness of the voluntary muscles of the whole body results in sluggishness of the general circulation and the swelling of viscera.

The heart, being of fairly normal size, is very often diagnosticated as hypertrophied. This apparent hypertrophy results from an abnormal

[1] *Berl. klin. Woch.*, 1878, p. 135.

shape of the chest wall which will be mentioned below, and results in compressing the respiratory organs and impeding the circulation. These are sufficient reasons to cause a hypertrophy of the right (and left) ventricle.

Respiration; Tuberculosis.—Tuberculosis is a frequent complication of rhachitis: many of the cases of tuberculosis of the bone in infancy and childhood are met with in rhachitical children. The at first active, later on passive, hyperæmia of rhachitical bones, mainly of the epiphyses, predisposes to the local invasion of tubercle bacilli floating in the circulation. In the lungs the disposition is as marked. In the infant the sixth and seventh ribs have long cartilages; the eighth, ninth, and tenth do not touch the sternum at all; the ribs are soft, influenced by dorsal decubitus, by the pressure of the arms and of the atmosphere, and by the constant traction of the diaphragm, which is without a solid support. Thus the chest is narrow, the chest wall not elastic, and, moreover, encroached upon by the large abdomen and the frequently deformed clavicle. Besides, the muscles are feeble, and respiration impeded for all these mechanical and physiological reasons, to which is added the sensitiveness of the periostea.

The insufficiency of respiration, the non-expansion of the lungs, and the obstruction of circulation lead to bronchial hyperæmia and bronchitis in all its forms, also to mediastinal adenitis, and enhance the opportunities for microbic invasion. The universal effect of the above described conditions renders the tuberculosis of the rhachitical infant more general and not confined to a special (upper) lobe. Not infrequently the lower lobes, corresponding with the most compressible part of the chest wall, are pre-eminently affected and filled with extensive caseous deposits. Nor is the hilus a very frequent point of selection. All this makes the diagnosis of such a tuberculosis from chronic capillary bronchitis a difficult task. Sometimes the persistency of râles in a certain locality, while those of others change, is the only reliable symptom.

Otherwise, the usual forms of catarrh and inflammation of the *respiratory organs* should not be classed as complications of rhachitis. From what has been said, they are easily understood to be the direct results of the physiological and mechanical effects of the changes worked by the rhachitical process. The same may be said of anomalies of the lymph bodies. The swelling of the mediastinal "glands" found in rhachitis depends on the morbid changes of the intrathoracic organs; that of the lymph bodies of the neck on the irritation brought about by nasal and pharyngeal catarrh and by eczema of the head and face; that of the mesenteric lymph bodies on the hyperæmia and hyperplasia following diarrhœal diseases. Uncomplicated rhachitis—that is, a rhachitis not attended by local irritation of the integuments, either mucous or epidermal—has no tumefaction of lymph bodies.

The Skin.—The external integument, the skin, suffers from rhachitis to a certain extent. It is mostly pale, in accordance with general anæmia and that previous ill-nutrition which is sometimes brought on by, or attended with, respiratory or digestive disorders. Some of the changes of the skin have been mentioned in connection with craniotabes; here it is thin, with large veins and copious perspiration. With the

latter miliaria and other eruptions depending on perspiration—eczema, for instance—are observed. Those who become rhachitical after prodromal respiratory or digestive diseases are liable to have a dry, desquamating skin. Acute lichen is not uncommon, and pruritus is very annoying in occasional cases.

I mentioned *prodromi*. Is there anything deserving that name? In the infant, anæmia with good weight, muscular inactivity with rotundity, copious perspiration mainly of the occiput, later on insomnia and restlessness, are not prodromi; they are themselves symptoms of rhachitis. Nor is it permissible to speak of digestive or respiratory disorders as prodromi, because there are more such ailments without than with consecutive rhachitis. Whenever the latter follows them, they ought to be considered as co-operative causes.

Acute Rhachitis; Scurvy.—Is there anything like "*acute rhachitis*"? Cases developing within a few weeks, sometimes with elevation of temperature, with much pain over the ribs, long bones, and mainly over epiphyses, with rapidly increasing paleness and tumefaction of the spleen, deserve the name.

Such cases have been described, and I have seen them, though their number is rather scarce compared with the legion of chronic cases observed in any extensive practice. Many of them may have been erroneously diagnosticated, however, where the condition was one of "scurvy"—Barlow's disease. In connection with such doubts three questions ought to be answered, the first of which is: Is there such a thing as acute rhachitis? the second: Is there a *complication* of rhachitis, chronic or acute, *with scurvy?* and the third: Is infantile *scurvy* an independent disease or in every case *founded on rhachitis?* The first question I did not hesitate to answer affirmatively. The second may be answered by a very few historical facts, if not by personal experience. In Vol. 46 of the *Dictionnaire des Sc. méd.*, 1820, art. "Rhachitis," Jean Baptiste Montfalcon discusses the "relations existing between rhachitis and scurvy," and, after having quoted a case of Portal's of "scorbutic rhachitis," describes painful tumefaction of femur and tibia, with ecchymoses and swelled gums. He adds: "In other cases, which are rare, however, such a complication of rhachitis and scurvy has been noticed."

After describing some cases of undoubted rhachitis, I. O. L. Moeller published [1] that of a hitherto healthy child in a well-to-do family that was nursed until teeth came. During a cold and rainy season there appeared pain about the right ankle- and knee- and the left ankle-joints. Afterward a number of epiphyses exhibited marked swelling, and the baby became pale and flabby. Thus far, the description of rhachitis is clear. Afterward there appeared ecchymoses on the tibia, and the gums, mainly the upper, became scorbutic, bled, and swelled so as to cover the teeth. The gums covering the alveolar processes where there were no teeth participated in the process. Lemon-juice and vegetable diet caused the patient to recover rapidly. No doubt this was a case of scurvy after it had been one of rhachitis. That is, what Moeller calls it, both "acute rhachitis" and "scurvy." The same observation was made and the same name given by R. Foerster [2] and by Cheadle, who

[1] *Königsb. med. Jahrb.*, vol. i., 1859, p. 377. [2] *Jahrb. f. Kinderh.*, 1868.

published "three cases of scurvy supervening in rickets in young children." [1]

The problem whether infantile scurvy is always complicated with or depending on rhachitis would be solved at once if there were a single case of scurvy in which symptoms of rhachitis were absolutely absent. The, according to him, conclusive case of Baginsky,[2] is, however, not of that order. The baby was a year old when it was taken sick and died. Round the subperiosteal hemorrhages the recent ossification was found to be normal, and not of the low rhachitical character. But the remark added at another place of the report excluding only "marked" traces of rhachitis renders the conclusiveness of the observation doubtful.

From my own observations of Barlow's disease, altogether about 40 cases, I have arrived at the following conclusions:

Scurvy is often observed in mildly or thoroughly rhachitical babies; scurvy and rhachitis are met with at an early time; my youngest case of scurvy was six weeks old. The presence of teeth is not a *conditio sine quâ non* at all in scurvy. Both scurvy and rhachitis are found after improper foods have been given a long time. Cow's milk, sterilized, when used as exclusive food, is emphatically a frequent cause of either or both. Both are complicated with pain. Both occur in all walks of life—rhachitis undoubtedly more amongst the poor; scurvy, according to my observation and that of many others, most frequently amongst the well-to-do, who can afford to spend their money and their infants' health on proprietary foods. In both diseases the spleen may be found enlarged.

Scurvy appears not quite rarely, certainly more frequently than rhachitis, after the termination of the second year. Still, most of the cases belong to an earlier period. Some undoubtedly were observed in such as never had any rhachitis; they were principally those who had been raised, when two and more years old, on an exclusive diet of sterilized milk. It is not the epiphyses (in very rare instances a joint), but the diaphyses, which are affected. There are subperiosteal hemorrhages (and pain) in the lower and (or) upper extremities, also on the sternal ends of the ribs, possibly on scapula and cranium, frequently on the maxillæ, in the orbit, eyelids, skin, and subcutaneous tissue, mucous membranes, into the intestinal and the urinary tracts, and into the muscles. Rarely suppuration takes place into the joint, or a separation between diaphysis and epiphysis in scurvy, never in rhachitis.

Thus it appears that Barlow was right as early as 1883 when he claimed that there are too many essential symptoms belonging to scurvy exclusively, and that the relations of rhachitis to it are frequent, but variable, and that sometimes there are none.

Fœtal Rhachitis.—A certain number of babies are born while rhachitis is running its course or after it has completed it. In that respect the opinions of authors are very much divided. Fœtal rhachitis was declared rare by Virchow and by Ritter; on the other hand, frequent by Kassowitz and by Unruh; Schwartz (1887) claims that 400 out of 500 babies born in Breisky's clinic were rhachitical. The cause of these discrepancies must be sought in the fact that writers are not unanimous in their opinions as to their cases. They are not alike. Such as I have

[1] *Lancet*, Nov. 16, 1882. [2] *Berl. klin. Woch.*, April 12, 1897.

seen were decidedly cases of rhachitis in which formerly normal bone had become diseased. A small number of others have been described in which the osseous system was thoroughly decalcified, where torsions of limb and fractures of bones were found in a large number of places, which undoubtedly were the result of imperfect osteogenesis, such as Stilling described in 1889. E. Kaufmann, in his investigations on the "so-called fœtal rhachitis" (Berlin, 1892), removes fœtal rhachitis altogether from what is called rhachitis in after life. The cases thus far reported have a common basis—that is, defective ossification. He distinguishes three varieties—the malacic, hypoplastic, and the hyperplastic. In the first cartilage remains have become soft; in the second growth is interrupted, arrested; in the third growth is lively but inordinate. The final outcome of the process depends on the period of its starting. When this takes places late the fœtus may live with more or less deformity of head, trunk, or limbs, and more or less damage to intelligence. O. Lubarsch[1] collected 21 cases. Some of them were idiotic, some not at all. Extremities were straight or curved or shortened; sexual organs, infantile or fully developed. Some cases were hereditary. The skeleton was often infantile, the bone-forming cartilage persisted, and was sometimes present in small quantities only. Normal ossification that would take place in this small amount of cartilage at any time—for instance, at the base of the skull—would reduce the length of the latter. Ossification taking place prematurely in fully developed bone-forming cartilage in the same place would have a similar effect. If during fœtal life, it would cripple the base of the brain by shortening the base of the skull. In this way the different forms of "cretinistic," "cretinoid," and "cretin" conditions (Virchow) would depend either on the amount of the bone-forming cartilage or on the period of its changing into bone, or on both. The same changes when taking place in the fœtal vertebral column and in the extremities or in the pelvis would have the same effect. They remain short and become clumsy or deformed.

The malacic form is probably the one which exists in a few cases of unusual rarity of fœtal rhachitis which are described as something absolutely different from all other forms. The hypoplastic malnutrition of the cartilage ("chondro-dystrophia") is represented in dwarfism with its persistence of all cartilage, and when the thyroid gland is inactive or absent at the same time, in "congenital myxœdema." The hyperplastic chondro-dystrophia resulting in premature ossification of the basis cranii and shortening of the base of the brain, with retraction of the vomer and root of the nose and with flat palate, with or without the co-operative influence of an endemic miasm, and with or without the absence of thyroid function, terminates in cretinism complicated with more or less dwarfism. From this point of view the cases of Siegfried Müller[2] and John Thomson lose their mysterious look and gain in interest. Müller describes a case in which the proliferation of cartilage rendered the periosteum atrophic, thereby crippling the after-growth of the bone, and another case in which, *vice versâ*, the cartilage was rendered atrophic by the proliferating periosteum (fœtal chondro-malacia).

[1] *Results of General Etiology in the Diseases of Man and Animals*, Wiesbaden, 1896.

[2] Periosteal Aplasia with Osteopathyrosis under the guise of so-called Fœtal Rhachitis, Munich, 1893.

His case was a twin whose brother was healthy. Klein's case, quoted by Müller, was of the same nature. Porak had the case of a mother and child; Kaufmann, one that resulted from incest between brother and sister.

John Thomson[1] published a "Note on Three Living Cases of Achondroplasia (chondro-dystrophia fœtalis, or so-called fœtal rhachitis)." His cases were those of a boy of five and of two men of thirty-nine and thirty-six years, all the other members of whose families were well. What I tried to classify above he briefly defines as "absence, arrest, or perversion of the normal." In his cases "those bones which are formed entirely in membrane, and those which, though formed in cartilages, remain altogether or mainly cartilaginous till a late period of intra-uterine life, and found quite normal in size" (upper part of cranium, neck, trunk, clavicle, scapula, sternum, spine, carpal and tarsal bones); "those depending on endochondrial ossification for their growth in utero are dwarfed" (base and skull, ribs, pelvis, the long bones of the limbs).

Rhachitis Tarda.—There are exceptional cases in which rhachitis persists very long and appears late. These have been called retarded—rhachitis tarda. Recovery in the usual run of cases may be late and ossification very much delayed. The large fontanelle may persist open to the tenth year and longer; the temporary teeth may remain beyond the usual period, and when they fall out they are not replaced in due time. Some children could not walk because of the softness of their bones until they were eight or ten years old, or in otherwise healthy children rhachitis will break out in advanced age, bones soften, knock-knee make its appearance. Some such cases have been claimed as osteomalacia, if, indeed, it is possible to differentiate this from rhachitis except through the fact that it appears in the fully developed; rhachitis, however, in the growing bone. At no age should we expect all of the possible symptoms of rhachitis to occur together and uniformly. Rehn noticed at an advanced age softening of diaphyses, with but little epiphyseal swelling. Biedert the same, together with softening of ribs and the epiphyses. Local softening has been principally observed in the neck of the femur. A girl of fifteen, who complained since her third year, had a prominence and elevation of the trochanter (J. Schulz). Deformities of the same neighborhood were noticed by E. Müller in adolescents of sixteen, seventeen, eighteen, nineteen years. Bilateral rhachitical curvature of the neck of the femur was noticed in a boy of fifteen, with reeling gait, by Rotter. Royal Whitman has 4 cases, in boys of fifteen, sixteen, seventeen, eleven years, of bending of the neck of the femur. He accuses rapid growth, overwork, standing up, and carrying loads as predisposing causes. B. Farquhar Curtis published a similar case in a boy of seventeen, who, without other marks of rhachitis, had complained for fifteen months of pain in the right knee and two months of walking lame. The right lower extremity was shorter (rhachitical limbs remain short after recovery), the hips stiff, and flexion painful. When an operation was made the bone was simply found bent, not tubercular.

In such cases we ought not to forget that in many instances of early rhachitis the femoral neck is often distorted by rhachitis, and that the

[1] *Edinburgh Med. Journ.*, June, 1893.

ossification of the head and neck down to the trochanter is very slow in being completed.

DIAGNOSIS.—The diagnosis of rhachitis is easy where a number of its symptoms are met with simultaneously; when but a few, it may require some time to become positive. Among the earliest symptoms are—pallor of the integuments; insomnia and restlessness during nights; perspiration, mainly of the head; muscular debility; constipation, beginning between the fifth and eighth weeks of life, often with a placid kind of indolence; prominent frontal tubera; craniotabic spots on the parietal bones mostly (posteriorly), also on the occipital, together with widening of the sutures and patency of the fontanelles; falling out of the hair on the occiput, which feels unduly hot; sensitiveness of the ribs when they are touched, or an outcry when the baby is raised from its couch; swelling (sometimes painful on touch) of the epiphyses of ulna, radius, femur (the two latter, however, are large normally), tibia (and fibula), also of the insertion of the ribs; the curvatures of tibia, femur, or arm which were described above; early bronchial catarrh, particularly when persistent without an apparent cause; late or irregular teething; the swelling of the spleen, which may be distinctly felt below the edge of the ribs—they are all, some positive, some probable, symptoms of rhachitis.

Scoliosis in the rhachitical infant is apparent only, and depends on muscular debility, like *kyphosis*, which when rhachitical only is in the young recognized by its disappearance when the baby is supported by chest and heels. *Congenital dislocation* of the hip would show the head of the femur luxated. It should be remembered, however, that scoliosis and a moderate (not angular) kyphosis may result from rhachitis after recovery of the bones; also that rhachitis may complicate other causes of the above anomalies. The *curvatures* of the long bones are quite characteristic. If they be combined with swellings, the latter belong to the epiphyses, and are not to be compared with the gummatous periosteal irregularities sometimes noticed in *syphilis*. The latter are steep, circular, or spindle-shaped, and sometimes the adjacent soft parts are œdematous.

Still, rhachitis and syphilis may be found together. In both the localities of active growth are affected; in both the transportation of calcifying cartilage into bone is arrested; and in both there is ample proliferation of cartilage cells. The periepiphyseal lines of the extremities suffer in both. Disruption between epiphysis and diaphysis (more in the upper end of the humerus than in other localities) and suppuration of the joints may take place in syphilis, but not in rhachitis. *Craniotabes* is characteristic for rhachitis; in hereditary syphilis the cranium is rarely affected, and mostly at a later period, and the changes are granulomata, mostly on the parietal bones. Altogether, most bone symptoms of syphilis are earlier than those of rhachitis, unless the latter be congenital, and exhibit short and thick bones and some deformities of epiphyses and of thorax. Syphilitic osteochondritis is noticed four or five weeks after birth, with characteristic changes in the skin which are not found in rhachitis.

The *teeth* are defective both in rhachitis and in syphilis—the temporary in the former, the permanent more in the latter. The particulars of this difference have been mentioned above.

Genuine *hydrocephalus* and rhachitical hydrocephalus may be diagnosticated from one another in most instances. In some cases this is difficult, for the patent fontanelles and sutures will be met with in both forms, as also sometimes in the presence of tumors. It is easy when there is a history of previous rhachitis. The deposits on the forehead, the presence sometimes of characteristic cranial defects with steep edges in the middle part of the bones, and of laryngismus stridulus, which is rare in genuine hydrocephalus, the condition of the thickened epiphyses and the curvatures of the diaphyses, are our guides. During the first stages of rhachitical hydrocephalus the intellect is not disturbed. The hydrocephalic head of rhachitis is rather spherical; that of genuine hydrocephalus, when external, is often so bulging laterally that it appears flat on top; when external, bulging upward and even conical or pointed. In the latter two forms the face is comparatively small at an early period, and the intellect impaired. Hebetude, idiocy, paralysis are more easily found in the genuine form, though they be not absent in some rhachitical cases. In the former the orbit is soon depressed and ocular symptoms more prominent. Choked disk and other pressure symptoms are met with early. The fontanelle pulse and the systolic murmur disappear soon, while in rhachitical hydrocephalus they are apt to remain a long time. Still, there are cases in which the diagnosis may become very difficult indeed. It is desirable, however, to come to a conclusion very soon, for a rhachitical hydrocephalus is by no means unpromising. Many cases get altogether well, and proportionately very few carry their calamity into the second decade. In many the combination of phosphorus with judicious punctures proves efficient. Lumbar puncture, however, as a means of diagnosis does not answer readily, for the strength of the current through the exploring needle does not correspond with the amount of fluid (which is liable to be less in rhachitis than in genuine hydrocephalus) contained in the cerebral cavity. As a therapeutic agent, repeated lumbar punctures, each of which should not remove more than a few drachms, may be found serviceable, while the rhachitical condition, both the general and the local, is being corrected by antirhachitical treatment.

The *epiphysitis* of rhachitis is a frequent cause of what is called "growing pain." For diagnostic purposes it is advisable to remember that this term is used for a number of different conditions. Myalgia from fatigue, located near the knees and ankles, or connected with incipient fevers, or auto-infection; diseases of joints and bones other than rhachitical; acute or subacute articular rheumatism, which is much more frequent in infancy than many will still admit; dull pain depending on coxitis or pericoxitis; neuroses round the joints, which, though not common, are met with in children—they all suggest and give rise to the diagnosis of "growing pain." If it be localized in the epiphyses and connected with other symptoms of rhachitis, the exact etiological diagnosis suggests itself very readily, together with its indications.

The pain of rhachitical epiphysitis may be very severe. When it is combined with periosteal sensitiveness and some periosteal swelling a careful examination only, in some cases, will secure the diagnosis from (gonorrhœal or other) arthritis and from osteomyelitis.

PROGNOSIS.—The prognosis depends on the organs or tissues affected

and their number, and the slow or rapid course of the development of the symptoms. Spontaneous recovery is not unusual, mainly under the influence of fair hygienic conditions. When the bones are mostly affected the epiphyses may remain thickened, and curvatures may be visible through life. But, unless they be very bad, they will partly disappear during the progress of growth. A rhachitical pelvis may become dangerous during parturition. When the diagnosis is made early the prognosis is good, even when anæmia and dystrophy are marked. Symptoms belonging to the nervous system may prove dangerous, a single convulsion may cause permanent cerebral lesions; laryngismus may be the cause of sudden death; hyperæmia of the skull and meninges may lead to meningeal effusion and hydrocephalus; a rhachitical chest to atelectasis, bronchial catarrh, broncho-pneumonia, and mediastinal adenitis. Complication with tuberculosis is not uncommon for that very reason. The general health is always suffering through the first months of rhachitis; that is why every disease, and mainly infectious diseases, run a graver course in rhachitical patients. Still, with all these possibilities, the general prognosis in the vast majority of cases of rhachitis is favorable.

PREVENTION AND TREATMENT.—As no one cause can as yet be charged with producing rhachitis, prevention has to cover many points. Parental syphilis, anæmia, or tuberculosis yields more indications than it is always possible to fulfil. When discovered in the newly born it should be met at once. Anæmia or rhachitis in the infant, based upon parental syphilis, requires mercury, not iron—the latter as adjuvant only if at all. Attention should be paid to general hygiene. More can be accomplished by furnishing good air than by any other means. The air of the winter is no contraindication to keeping windows open and taking the young baby out as long as there is fit covering to be had. Sea-air is preferable as long as there is no contraindication—for instance, in the condition of the respiratory organs.

Sojourning in the country is beneficial only when the rhachitical infant is not locked up in the house. Sea-air, together with sea-bathing, warm, cool, or cold according to age, condition, and training, is an excellent preventive or curative measure. England commenced that practice on a large scale in 1750; Italy, France, Germany, America—these last twenty years only. The sea-hospitals have done an immense amount of good. To eradicate rhachitis, however, the children must be kept on the shore for years. In America we are always too much in a hurry, and expect the benefits of heaven and earth in a particle of a season. When no seashore is accessible, the bathing in salt water, with friction, massage, occasionally with electricity, may be done at home. When eczema is caused by it the salt-water treatment should be discontinued.

Plenty of air by day and by night, coupled with poor food, is still safer than the best possible food with bad air. Comby's saying that rhachitis gets access through the stomach, not through the skin or lungs, requires a good deal of modification. Still, the nature of the food is highly important. Lactation must not be continued beyond the appearance of two or four teeth. A wet-nurse should not be too young nor too old. An apparently proper age of even mother or wet-nurse does

not always exclude the possibility of a breast-fed baby becoming rhachitic. In such case a well-selected artificial food is preferable to breast milk. Pure cow's milk, no matter whether raw or boiled, is harmful. Artificial foods must be well selected and watched. The absence of pathogenic germs from them is not the only safeguard. Even Rotch's "modified milk" and Gärtner's "fat milk" do not protect against rhachitis, though they may be fairly proof against microbes. The addition at an early date of cereal decoctions (barley, oatmeal) and of animal broths renders all the known milk preparations safer and more wholesome. Too large percentages of starch in the foods of young infants should be avoided. A small quantity, however, is assimilated by the newly born. The addition of fat is not always a protection against rhachitis, and may easily be overdone. Diarrhœa, dyspepsia from whatever cause, and constipation should be corrected, and the warning, often expressed by me and urged by Comby, should not be forgotten—that over-alimentation is at least as dangerous as under-alimentation. That the skin requires intelligent attention was suggested above. Whether a bath should be given immediately after birth and continued regularly during the first weeks ought to depend on the nature of the individual case. As a general rule, which is valid for every child, bathing—first tepid, later on cooler, in salt water when there appears to be an indication for more surface stimulation, with appropriate friction—improves both the cutaneous and the general circulation.

Cod-liver oil, when tolerated, influences rhachitis favorably. As a rule, however, it is not so universally well borne in rhachitis as it is in "scrofula." I do not advise the use of its compounds, emulsions and so on, except when it is disliked or the latter have some other indications—for instance, diarrhœa, which is sometimes produced by the oil, mainly in the hot season. In that case it may have to be discontinued or may require the temporary addition of bismuth or phosphate of lime. The action of cod-liver oil is considered by some hygienic only, by others remedial. The former attribute its effect to the fat, and believe the substitution of any assimilable fat an equivalent. I do not believe its agency to be thus restricted, for three teaspoonfuls of cod-liver oil will never be replaced by three teaspoonfuls of cream or other fat. Universal experience teaches its wholesome influence in many morbid tissue changes. Possibly the mystery of its action is best explained by attributing to it a percentage, though ever so slight, of some organic-tissue juice of a nature and efficacy comparable with that of the thyroid gland. Tempted by that point of view, Heubner tried the action of thyroid gland in rhachitis, but without any tangible effect. He believes, however, that the general condition of the infants appeared to be improved by it.

"Lipanine" is not an equivalent or substitute for cod-liver oil. Malt and "maltine" preparations have found much favor, both with the profession and the public. Unfortunately, the market has been swamped with all sorts of combinations and mixtures to such an extent as to shake confidence in their honest composition in the same degree as the mere object of making money by it became pre-eminently clear.

Many of the serious results of softness of the bones can be either avoided or counteracted by precautionary measures. I repeat: babies

in general, and those with incipient rhachitis in particular, must not be made to sit up before their vertebral columns and their dorsal muscles are able to support them. They must not be carried about in the erect posture, nor on the same arm always—an invariable cause of scoliosis and of genu valgum of one and of genu varum of the other knee. They should be kept and carried about in a reclining posture, better on a hair pillow than on the unprotected arm, until they feel strong enough to do without it. Thus scoliosis may be prevented. A baby must be discouraged from walking before its limbs are sufficiently strong; no walking baskets must be employed, no proud grandparents allowed about. Thus the curvature of the diaphyses of the lower extremities, which in part results from the weight of the body on the feeble limbs, and the secondary deformities of the pelvis, are reduced to a minimum.

Craniotabes, with its local hyperæmia and excessive occipital heat, forbids the use of warm bonnets and of feather pillows. A soft hair pillow, or an air-cushion filled to one fourth part of its capacity, or a pillow with a central depression or perforation, such as recommended by Elsässer in his famous monograph (1843), should support the head. Excessive perspiration requires the washing with cool or cold water, or vinegar with water, or alcohol with water, or the application of a powder consisting of one part of salicylic acid with ten parts of oxide of zinc and twenty-five of starch. Craniotabes is relieved by the general hygienic and medicinal treatment of rhachitis. In former years I demanded six or eight weeks of treatment before I felt justified in giving a good prognosis. The administration of phosphorus, as taught by Kassowitz, has reduced that period of hesitation to one month. Concentric brain symptoms require appropriate treatment; great convulsibility, bromides, chloral hydrate, and mild opiates, which are well tolerated in this condition. It is particularly laryngismus stridulus in which they are indicated, alongside the antirhachitical, hygienic, and other remedial treatment. The single attack of crowing inspiration may be cut short by shaking the infant, by slapping the face or chest with a wet cloth, or by employing the spark of a Leyden flask; for the arranging and application of the interrupted current there is hardly any time. In cases of "difficult dentition," such as described above and due to the rhachitical deformities of the jaws, lancing of the gums becomes pardonable. Of the propriety of relieving hydrocephalus by lumbar punctures I have spoken above. They are certainly more promising and safer than repeated punctures through the fontanelles.

The therapeutics of rhachitis in all its forms has been greatly modified and improved by the introduction of *phosphorus*. Twenty-six years ago Wegner,[1] when feeding young animals on minute doses of phosphorus, found the medullary spaces which penetrate into the calcifying cartilage to be fewer and smaller than in animals not so phosphorized. When he fractured the bones of rabbits and fed the animals on phosphorus the bones would heal in a much shorter time than the fractured bones of animals not so fed. This experimental observation led me to employ phosphorus in all cases of subacute and chronic osteitis (mostly tubercular). My conclusions were that recovery or improvement was more readily accomplished under this treatment, but

[1] *Virchow's Arch.*, vol. 50.

it was not until 1880 that it struck me, preceded only by Trousseau, to suggest the use of phosphorus in rhachitis.[1] It was left, however, to Kassowitz—whose priority, therefore, is indisputable—to employ it on a large scale in that disease with what I know positively to be favorable results in all cases of rhachitis from the chronic to the acute form, in craniotabes and laryngismus, and in its scorbutic complication.

The dose of phosphorus in these cases is from one third to one half of a milligramme three times or twice a day. The oleum phosphoratum of the U. S. Pharmacopœia contains 1 part of phosphorus in 10 parts of ether and 90 of oil. Its daily dose is from two to three minims. Concentrated oil solutions are liable to decompose. "Thomson's solution" keeps fairly well, but the best preparation is the elixir of phosphorus of the U. S. Pharmacopœia, composed of 210 parts of the spirit of phosphorus, 2 parts of oil of anise, 550 parts of glycerin, and a sufficient quantity of aromatic elixir to make 1000 parts. Of this elixir a teaspoonful contains 1 milligram of phosphorus, and a dose, to be repeated three times a day, is from 6 to 15 minims. No temptation ought to be strong enough to employ phosphates, which will invariably reappear both in the urine and in the fæces. It appears probable, moreover, that the phosphates contained in nutriment are more digestible and more assimilable. Phosphates are much inferior in effect to the hypophosphites of the Pharmacopœia with or without iron. Phosphorus given simultaneously with cod-liver oil is a good combination, but it is a doubtful practice, as long as the latter is not a uniform compound, to dissolve phosphorus in that oil. Where anæmia is intense additional preparations of iron are required. The syrup of the iodide of iron may be given, as many drops three times a day as the baby is months old, or from 10 to 25 drops three times a day to children of from one to two or three years. When the spleen is large and the lymph bodies are tumefied, three daily doses of one half to one drop of Fowler's solution are beneficial. In scorbutic cases or complications fruit juices are required. Heubner's experience with phosphorus in bad hospital cases is negative. The famous teacher knows, however, as well as anybody, that bad rhachitis never does well in hospitals. If it requires anything, it is air, air, and again air! To his discomfiture, however, we owe some observations which, though they be negative, are valuable. Guided by what he takes to be a fact, that all irritations and inflammations from known causes are local,[2] but that the rhachitical anomaly of a skeleton is universal, and localized only when and where growth is going on (I have repeatedly in these pages stated that simply atrophic infants are not rhachitical), and supported by Lanz,[3] who found certain relations between the thyroid and bone development and suggested a trial of thyroid in rhachitis, Heubner gave from one half to one decigramme of Merck's thyroidin every other day or every day, with negative result as far as improvement of rhachitis was concerned, but with a favorable effect, however, he believed, on the general condition of the child.

[1] *Trans. Med. Soc. State of New York*, 1880, and *Therapeutics of Infancy and Childhood*, 1896, p. 102.

[2] Is that really so? The irritation by alcohol has very much more than local effects, which are by no means limited to the same class of tissue.

[3] *Correspbl. f. Schweizer A.*, 1895, p. 45.

Mere forcible reductions in the curvature in the soft bones are useless, for relapses must invariably follow. In cases of urgent necessity splints may be used until medical treatment has had its effect. Total fractures of rhachitical bones are rare. Infraction (subperiosteal, "greenstick" fracture) requires splints until the bone will have time to get normally hard. When the tendency to it is very marked, immobilization of the entire body may become necessary. The pigeon-breast, which has a tendency to remain for life, requires the earliest possible medicinal and hygienic interference and protracted gymnastic expansion of the lungs. Even crying is welcome, and in children of two or three years trumpet-blowing and soap-bubbling should be encouraged. The curvatures of the diaphyses are less marked than in the adult, because of the extension which takes place during growth. This clinical experience has been amply verified by the close observations continued through years in the clinical institutions of Tübingen and of Innsbruck. If other splints are to do any good, they should be applied before the bones become hard and resist every degree of permissible pressure.

The tendency to *flat-foot*, acquired through the flabbiness of the ligamentous apparatus during the attempt at walking, requires raising of the arch of the foot by a moderate spring and a support for the ankle; *scoliosis* of growing children of more than six or eight years, Sayre's plaster-of-Paris or a felt jacket; ugly and determined curvatures of the long bones, either *osteoclasy* (fracture of the curved bone, while the periosteum is mostly left intact, and resetting) or *osteotomy* (straightening of the bone after a cutting operation). Of these two, osteoclasy was the only operation resorted to formerly. The fracture of the bones was either manual or instrumental, mostly successful in the middle of the femur or tibia, mostly unsuccessful for genu valgum or varum, inasmuch as it often tore off the epiphysis or fractured the bone in an undesirable place, and was often followed by septicæmia.

Osteoclasy has been mostly replaced by osteotomy. It is a simple and open operation. It is seldom required on the upper extremity, mostly on the lower, not so often on the thigh, as for genu valgum, varum, or the curvatures of the diaphyses. The genu valgum of children results from the curvature both of the femur—usually the only one at fault in adolescents—and of the tibia. It requires the supracondyloid operation of Macewen, and often a supplementary operation on the tibia. The curvature of the tibia has mostly its concavity interiorly and posteriorly, and is usually found at its lower half. The operation may be either simply linear (transverse or oblique) or cuneiform. In bad cases the latter wedge-shaped operation is preferred, and not seldom a single operation is insufficient. An interval of a few weeks is ample between the several operations that may become necessary. The results of osteotomy are almost always absolutely good. Suppurations there are few only, and controllable.

RHEUMATISM.

By W. GILMAN THOMPSON, M. D.

Definition.—The name rheumatism is derived from the Greek ῥέω, I flow, and was in ancient times applied to describe a supposed acrid humor generated in the brain or elsewhere, and flowing from the body in catarrhal flux, which, when restrained, produced internal or joint inflammations.

The word rheumatism, as now used, is a somewhat vague and unscientific name descriptive of a variety of conditions, which, however, have three things in common: (*a*) general or constitutional symptoms, largely febrile or toxic; (*b*) localization of inflammatory lesions in the joints and sometimes in the muscles or skin; (*c*) a tendency to certain visceral inflammatory complications, notably in the heart and serous membranes, and sometimes the tonsils. The popular use of the word rheumatism or its derivatives in conjunction with other diseases, such as rheumatic gout or gouty rheumatism, rheumatoid arthritis, and gonorrhœal rheumatism, is misleading by implication of an etiological relationship which does not exist. Whether they have a common antecedent diathesis or not, there is little or nothing in common in the pathological anatomy or symptoms of gout and rheumatism or of arthritis deformans and rheumatism, and the so-called "gonorrhœal rheumatism" is not a rheumatism at all, but a form of specific septicæmia with localized joint manifestations. In the further discussion of rheumatism the following clinical subdivisions will be recognized: I. Acute articular rheumatism; II. Chronic articular rheumatism. III. Muscular rheumatism.

In this connection gonorrhœal rheumatism will be separately described under the heading of Gonorrhœal Arthritis (page 972), not because it is etiologically related to rheumatism, but because it has certain features of clinical resemblance.

ACUTE ARTICULAR RHEUMATISM.

Synonyms.—Acute inflammatory rheumatism; Rheumatic fever; Acute arthritic fever.

Definition.—An acute disease, characterized by pyrexia, inflammation of the joints, acid excretions, and a decided tendency to inflammatory involvement of the serous membranes and heart. Rheumatism is usually endemic, but may prevail in epidemic form.

Etiology.—*Heredity.*—A tendency to rheumatism is undoubtedly often transmitted by inheritance. The disease has occurred in the newly-born, and the children of rheumatic progenitors are more liable to the disease than are others in the proportion of five to one. The disease, like both gout and tuberculosis, may therefore be either directly transmitted through this agency, or more often a constitutional predisposition to its development seems to be inherited, in some cases as strikingly as in gout (Cheadle).

The statistics of inheritance variously show that it is a factor in rheumatism in from 20 to 50 per cent. of cases. Most of these patients have had but one rheumatic parent, but when there is a double inheritance, or the disease has been transmitted through several successive generations, it usually develops in a severe and recurrent or persistent type.

Age.—Acute rheumatism is usually a disease of youth and young adults, although it may arise at any age. According to Whipham's statistics, 80 per cent. of cases occur between the twentieth and fortieth years. It is not often encountered earlier than the tenth year, and, although numerous cases were formerly reported as developing in early infancy, many of them are now believed to have been due to scurvy with localized joint symptoms, produced by improper feeding with proprietary infant foods.[1] Rotch[2] has, however, seen general rheumatic polyarthritis in an infant two weeks old, and another case at the seventh month. Both patients recovered after several months' treatment. Of 8631 cases analyzed by Besnier, but 301 were found in childhood.

Among 176 cases reported by Davaine[3] and Lancereaux, the maximum frequency was found to be between the fifteenth and twenty-fifth years. Out of 1303 analyzed by Fagge,[4] only 30 occurred as a first attack after the fiftieth year. Later than this the subacute and chronic forms may develop, but first attacks of the acute type do not occur.

Sex.—In early life females are more prone to the disease than males, especially at the period of puberty, but after the twentieth year, when the influences of occupation, exposure, and hardship predominate, males are much more often affected. Taking all ages into account, about one third more cases occur in males.

Climate and Season.—Rheumatism is more prevalent in cold, damp climates than elsewhere, although it is not uncommon in such climates as that of California. It is seldom seen in the tropics unless recently imported there. Sudden changes of weather are particularly bad, but have more influence upon the chronic than the acute form. Probably for these reasons rheumatism is more often developed in this country in the late autumn and early spring—seasons which are productive of unsettled temperatures and melting snow or rain. Sudden variations in the weather produce more effect upon chronic than acute rheumatism.

Soil.—Formerly attempts were made to attribute the frequent development of rheumatism to the nature of the soil, damp, mouldy soils being thought most injurious; but this belief has been modified by the supposed infection theory. Longstaff has found the disease more common

[1] See *Scurvy in Childhood*, by the writer, p. 000.
[2] *Pediatrics*, p. 1085.
[3] *Journ. de Méd. interne*, vol. ii. No. 3, p. 75, Feb. 1, 1898.
[4] *Principles and Practice of Med.*, vol. ii. p. 816.

where the soil is dry, and Newsholme points out the fact that a low subsoil water with high temperatures favors the breeding of many germs in the soil. As the disease originates in so many different surroundings, it is doubtful whether the question of soil has much, if anything, to do with it.

Checking of the excretions, if sudden, of the kidneys or skin, or of the excreta from the bowels from any cause, by determining accumulation of waste products in the blood, constitutes a strong influence; in other words, anything which renders the blood less solvent and restricts elimination and free oxidation is pernicious.

Exposure to cold alone cannot be regarded as ever causing rheumatism, but it may precipitate an attack when additional influences prevail. Getting chilled and wet in the rain, perspiring heavily and sitting in a draft, or falling overboard may result in an attack under such conditions.

Injuries to joints, such as sprains, blows, falls, etc., while not directly causative, are believed to often localize the inflammatory symptoms in the particular joints affected. Unusual fatigue or overwork of certain groups of muscles and joints may have like influence. Repeated injuries of rheumatic joints while inflamed may cause the disease to assume a chronic type.

Social Condition.—Rheumatism may attack persons of any social class, but it is less often encountered among the well-to-do than among those who are poorly clad, ill-fed, and who live among damp, dark, unhygienic surroundings.

Occupation.—Certain occupations predispose to the acquirement of rheumatism, notably those requiring prolonged exposure to cold and wet. Thus it prevails among coachmen, drivers, stokers, sailors, fishermen, 'longshoremen, blacksmiths, laundresses, and scrubbing-women.

Associated Diseases.—Malaria is often associated with rheumatism, and it is quite possible for gout and rheumatism to occur in the same individual, although this combination is much less common than is popularly supposed.

Severe nervous shock, exhaustion, general debility, starvation, chronic alcoholism, and anæmia may all be regarded as predisposing influences. Severe scarlatina is often accompanied by definite rheumatic symptoms, although these are probably of septic or of special toxæmic origin. Diphtheria and mumps may be accompanied by rheumatic symptoms, and chronic endocarditis is regarded as a predisposing factor. In a series of 56 cases of goitre reported by S. West he found that 11 per cent. were associated with a history of rheumatism.

PATHOLOGY.—The pathology of articular rheumatism is still involved in doubt, and is even more obscure than that of gout. Naturally, a disease at once so prevalent and so severe has stimulated research as to its cause and preventive treatment, and many able arguments have been adduced in support of conflicting theories. The fundamental question is whether or no the disease should be regarded as an active manifestation of a general arthritic diathesis—*i. e.* a constitutional tendency, either hereditary or acquired, to the localization of disease in joint structures. Widal says that the arthritic diathesis is to rheumatism what the scrofulous diathesis is to tuberculosis. Haig, who has contributed many years

of study and a large clinical experience, believes in such a diathesis or common dyscrasia as the starting-point from which in one individual gout may develop, in another rheumatism, in a third arthritis deformans. Other clinicians maintain with equal emphasis that rheumatism is a disease of independent origin, possessing no pathological affiliation with either gout or arthritis deformans, and having nothing in common with this disease beyond the clinical fact that in all three the joints are the chief sufferers. Of late years there has been a decided tendency to forsake the doctrine of diatheses in general, which has been largely fostered by a wider knowledge of the pathology of the blood and of the toxæmias of infections. A good illustration of this fact is found in the modern view of the infectiousness of tuberculosis as of far more importance than a supposed tubercular or scrofulous hereditary "diathesis." It will facilitate the study of this phase of the subject of rheumatism, which can be but briefly entered into here, to subdivide the hypotheses concerning its origin under the headings (1) Chemical theories; (2) Neural and neuro-chemical theories; (3) The infection theory.

(1) *Chemical Theories.*—In the light of present knowledge no one ingredient of the blood, accidental or otherwise, and not excepting lactic and uric acids, can be defined as alone causative of rheumatism. In gout uric acid is held responsible for the arthritic changes, but the theory of Prout and Richardson, that lactic acid fulfilled a like rôle for rheumatism, is no longer tenable. The failure of the exclusive alkaline treatment did much to discredit this theory. The most that can be admitted is the rather vague statement that the chemical cause is begotten of disturbances of absorption of digestive products, glandular function, and ultimate tissue metabolism, combined with imperfect oxidation and elimination of waste; but these broad general conditions alike favor the development of gout, rheumatism, and many other disorders. Wm. H. Porter believes in the influence of wrong diet upon rheumatism, especially the eating of more starches or sugars than can be oxidized. The percentage of urea excreted falls, and both uric and lactic acids are produced in excess. Sir Dyce Duckworth accepts Prout's theory, that an excess of lactic acid is present in the blood in rheumatism, but believes that it probably exists there as the product of bacillary action. Latham supports the uric-acid theory, but believes that the action of uric acid in rheumatism is aided by the coexistence in the blood of lactic acid; and Richardson long ago claimed to reproduce the disease by administering lactic acid by injection and per os. Nevertheless, lactic acid has been given in large doses to diabetics without ever exciting rheumatic symptoms, and many recent writers deny the presence in excess of either lactic or uric acid in the blood or system in acute rheumatism.

Haig is one of the strongest advocates of a chemical (uric-acid) origin of rheumatism, which he attributes in many cases to the influence of improper diet, drugs, etc., and he maintains that heredity has but little influence beyond the transmission of food habits. He finds that sodium salicylate can increase the uric-acid output by thirteen times. This is possibly due to its overproduction, for treatment by giving large quantities of water and alkalies alone does not wash out much more uric acid than normal. He also finds that iron and other metals capable of forming insoluble urates are harmful in rheumatism, whereas those things

which, like alkalies, colchicum, and salicylic acid, increase the solvent power of the blood for uric acid do good, and *vice versâ*. In response to the denial of the presence of uric acid in the blood, which was made by some of the older writers, like Sir A. Garrod, and also by Cheadle, Haig adduces the fact that modern methods of testing for the acid (such as Haycraft's) are far superior and more delicate; but his theory fails to explain satisfactorily why uric acid should cause tophi and certain typical visceral lesions in gout, which are wholly absent from rheumatism. Certainly, the clinical phenomena of the two diseases, rheumatism and gout, are too widely distinct to make it easy to believe in their common origin in the same chemical poison.

A theory which has been advanced, but which has met with little or no support, is that rheumatism and allied disorders all take origin from perverted glycogenic function of the liver. Some writers have argued that the essential pathology is to be found in the lymph (Latham), and in a stagnant circulation in the lymph spaces, so that the nutrient fluids around the joint structures, muscles, and nerves are imperfectly renewed (Frölich).

Whatever be the nature of the chemical cause of rheumatism, it is believed that it possesses a selective irritative action upon the fibrous tissues of the body, or that these tissues, being feeble, are irritated by it. This is most strikingly shown in the inflammatory reaction of the fibrous structures of the various joints and the tendons and fascia and muscle sheaths, as well as in the serous membranes (pleura and pericardium). The irritant also disturbs the nerve centres, the fibrous, chordal, and valve structures of the heart (endocarditis), and sometimes the skin (nodules, erythema, etc.).

(2) *Neural and Neuro-chemical Theories.*—The problem which the nervous theory attempts to solve is that the mere chemical peripheral irritation of the nerves by a simple ingredient, such as uric or lactic acid, is insufficient to directly cause all the phenomena of the disease, and that a pre-existing neurosis—*i. e.* some functional nervous disorder—exists (excited by exposure to cold or toxic substances), which, through trophic impulses, modifies the joint tissues, rendering them more susceptible to irritation and inflammation. This theory, also applied, perhaps with more force, to gout by J. K. Mitchell and others, is fully discussed by the writer under the heading Pathology of Gout (p. 992). It is the combination of the theory of special nerve action with that of toxins or organic chemical irritants of the body which constitutes the neuro-chemical or tropho-neurotic theory.

The pure nerve origin of rheumatism has not been so warmly upheld as have other theories, mainly on the ground that it fails to convincingly account for all the varied irritations present in typical cases. Its advocates point to analogous arthritides occurring in nervous diseases, such as myelitis, locomotor ataxia (Charcot's disease), syringomyelia, and sometimes with chorea.

(3) *The infection theory*, first advocated by Hueter, has been gaining adherents during the past decade, during which time it has come into vogue, and among its supporters are Bertholon, Duckworth, Maclagen, Newsholme, Guttman, and Sahli. In acute cases germs have been found from time to time in the blood serum, synovia, pericardial fluid,

and again in vegetations upon the heart valves, but no constant variety is uniformly present, nor have inoculation experiments yielded uniform results in either man or animals. Maragliano[1] has recently described a specific bacillus obtained from the blood of rheumatic patients with which he claims to have reproduced the disease in rabbits.

The chief grounds for this theory of infection consist in the following facts: (1) The disease is sometimes epidemic; (2) it is self-limited; (3) it mainly affects the young; (4) the severer symptoms and complications are suggestive of those of some other infections; such are: hyperpyrexia, endo- and pericarditis, pleurisy, pneumonia; (5) there is a tendency to leucocytosis, albuminuria, and anæmia, as well as to (6) development of a rash, erythema, to profuse sweating and high fever, as in pyæmia; (7) many infections, like pyæmia, gonorrhœal or other, scarlatina, cerebro-spinal meningitis, etc., present joint symptoms; (8) toxæmia best explains the number and variety of the different symptoms. The relapses are explained by Maclagen as due to development of new generations of bacilli, and he believes that salicylic acid is inimical to the bacilli. Sir Dyce Duckworth, while willing to accept this theory provisionally, does not think that it fully explains all the phenomena of the disease.

The fact that rheumatism sometimes prevails in epidemic form is one of the strongest arguments in support of the infection theory. Mantle refers to often finding two or three cases simultaneously in the same household, and Newsholme has observed distinct epidemics in Norway. In London of late years epidemics have been reported in 1868, 1874, and 1884. Strümpell alludes to its periodical increase in Leipzig, and Lange of Copenhagen refers to its variations in prevalence and intensity there. Various recent writers have reported cases which appeared to be derived by direct contagion, but this is certainly not a usual experience. Sacaze claims that in many cases a prior infective wound or inflamed throat can be discovered which might afford entrance for a bacillus of rheumatism; but the majority of cases certainly present no such solution of continuity, and cases of septicæmia or pyæmia with arthritis should be differentiated from true rheumatism.

The miasmatic theory of rheumatism has had many warm supporters, and Maclagen[2] states that he "believes the rheumatic poison to be malarial in nature," and "that the poisons of rheumatism and of ague, though specifically distinct, are similar in nature and mode of action."[3]

A summary of the hypotheses of the infection theory is the following: The specific bacillus of rheumatism, the growth of which is favored by a prolonged hot, dry season, in some unknown manner enters the system of one who is predisposed by inheritance and by chilling, exposure, fatigue, debility, or the like. The entrance of the germ is attained by unknown means, but once within the system it produces toxic materials in the blood, one of which is lactic acid (Duckworth), uric acid, or, more likely, some specific toxin, which in turn irritates the nervous system, causing various nervous symptoms and trophic disturbances, notably in the joints. As stated above, this is as yet mere theory, but it consti-

[1] *Gaz. degli Ospedale e delle clin.*, June 20, 1896.
[2] *Twentieth Century Practice of Medicine*, vol. ii. p. 228.
[3] *Loc. cit.*, p. 262.

tutes a plausible working hypothesis until a better explanation can be evolved.

The opponents of the infection theory claim that no germ has been found; that the disease has not been inoculated; that there is no positive proof of any case of direct transference of infection; that there is no recognized period of incubation; yet these things were said with equal force scarcely three decades ago in regard to both tuberculosis and malaria.

Pathological Anatomy.—The morbid anatomy of acute rheumatism is in nowise distinctive. Most patients recover with permanent lesions in the joints, and, unless valvular cardiac disease follows as a sequel to endocarditis, they are wholly free from pathological remains. During the acute stage hyperæmia is pronounced in the synovial membranes, and there is much uniform swelling of the joint structures and ligamentous tissues from serous infiltration. The synovial fluid becomes somewhat turbid with flakes of fibrine and leucocytes, and thickened with albumin; but there is no pus or blood, except in very rare cases. It may be acid in reaction. The synovial membranes may be covered with fibrine. The inflammation may extend along tendinous sheaths near the joints, especially in the hands and feet, and sometimes involves the bursæ.

The *blood*, next to the joints, is most affected. Few diseases, other than diphtheria, are capable of causing anæmia so quickly. The red blood cells are diminished by one half or more, the hæmoglobin falls below 50 per cent., and there is decided tendency to leucocytosis. The salicylates, so often given in treatment of the disease, greatly increase this anæmic condition if their use be long continued. An excess of fibrine is frequent in the blood, which may reach double the normal percentage. Davaine[1] found the percentage to rise from 3 to 7 or 8 parts per 1000. In bad cases capillary dilatation, ecchymoses, and extravasations may occur on mucous or serous surfaces or in the skin (purpura rheumatica).

The *heart* is inflamed in about one third of all cases, for, although bruits are heard in a larger percentage, some of them are due to previous attacks of rheumatism or some other condition, such as anæmia. The left side is oftenest affected, probably on account of its greater functional vigor and activity, for the poison of acute rheumatism seems to irritate the tissues while they are most vigorous, selecting the joints of younger persons by preference. The edges of the mitral cusps where they come in contact present swollen ridges, and may become covered with vegetations of fibrine, of which the blood has been shown to contain an excess. The process is rarely productive of ulceration, but contraction and distortion of the cusps may follow. For a full description of these lesions the reader is referred to the article upon Endocarditis (Vol. II. p. 392). Pericarditis and myocarditis present no peculiarities in rheumatism, and descriptions of these lesions will also be found in Vol. II. pp. 359, 439.

Symptoms.—Acute rheumatism begins with sudden invasion, characterized by fever and severe pain, tenderness, swelling, and redness in one or more joints. There is also local heat in the joints. Usually one

[1] *Journ. de Méd. interne*, Feb., 1898, vol. ii. No. 3, p. 75.

joint after another is quickly involved, so that within a day a half dozen may become inflamed. In a few cases only are there prodromal symptoms lasting for a day or two, consisting of headache, lassitude, anorexia, a coated tongue, indigestion, constipation, muscular pains, chilliness, and mild tonsillitis, pharyngitis, or laryngitis. The tongue is pale, flabby, coated, and indented by the teeth. Epistaxis may be present.

The Joints.—In first attacks, as a general rule, the larger joints only are affected, such as the knee, ankle, shoulder, or wrist, whereas in subsequent attacks the smaller phalangeal joints are commonly involved as well, but there are many exceptions. The fingers are seldom involved alone in the acute as they often are in the chronic type.

In bad cases almost all the joints in the body seem to suffer from the inflammation; usually, but not invariably, the symphyses and vertebral and temporo-maxillary joints are exempt. Sometimes the disease is confined to one or two joints throughout its course, but more often several joints, from three or four to a dozen or more, are involved, either simultaneously or in quick succession, the typical signs of inflammation, local heat, pain, redness, tenderness, and swelling, appearing in some fresh joint before they have wholly left another. With the general exceptions mentioned above, there is no typical order or combination in which the joints are involved.

Upon analysis of 706 cases of rheumatism treated at the Presbyterian Hospital by the medical staff during the past nine years, I find the proportion in which the principal joints were involved approximately as follows: knees, 65 per cent.; hands (wrists or fingers), 45 per cent.; ankles, 40 per cent.; shoulders, 10 per cent. It will thus be noticed that the lower extremities are more apt to be involved than the upper.

Rarely but one joint is involved, with severe constitutional disturbance. I have seen this happen two or three times in the case of the sterno-clavicular articulation, a joint which is usually exempt. In many cases there is some degree of symmetrical involvement of joints upon opposite sides of the body, but this is by no means uniform.

The swelling varies much and is most marked where the skin is thinnest. Effusion is not extensive, and most of the swelling is due to periarticular infiltration. Extensive bursitis is uncommon. It is mainly in cases of gonorrhœal septicæmia that the swelling is considerable around and between the joints, but in rheumatism the hands and feet may become greatly swollen over the dorsal surfaces from implication of tendon sheaths in the inflammation.

Pain about the inflamed joints is constant, most severe, and wearing. The slightest pressure or change of position intensifies it, and it wears upon the patient's nerves, producing restlessness and insomnia. It may be quite severe with mild arthritic lesions. Unlike the localized pain of gout, that of acute rheumatism, often equally severe, is felt in one joint after another as the intensity of the inflammation shifts from joint to joint. The pain renders the patient completely helpless, for it prevents any movement in the affected joints, and he is in constant dread of fresh invasion. Quite often it is worse at night, but, unlike syphilitic periosteal pains, it does not tend to disappear in the daytime.

The *pulse* is somewhat accelerated, usually not above 100–105. It is soft and compressible.

The *respiration* is unaffected unless there be cardiac complications.

The *temperature* varies much in different cases, and is not always proportionate to the intensity of the joint symptoms, although the involvement of new joints or the beginning of an endocarditis is apt to beget a sudden rise. It commonly ranges between 102° and 104° F., reaching a maximum within two days, remaining elevated for several days, and falling with the gradual subsidence of the acute inflammation, and showing at all times wide fluctuations and great irregularity.

Hyperpyrexia is a well-known occasional symptom in rheumatism. The temperature exceptionally rises to a high degree suddenly and without known cause. Such cases usually exhibit delirium, a feeble pulse, sweating, and become alarmingly ill, but DaCosta reported a temperature of 110° F. without delirium. Ringer lost a case with the same temperature, and Wilson Fox reported a recovery after a temperature above 110° F., which was treated by cold baths; another case met with fatal issue at 112° F. Patients with rheumatic hyperpyrexia are apt to die in coma. The temperature may continue to rise after death.

Sweating is a common accompaniment of fever in rheumatism, and as the patient perspires the temperature may drop a degree or two, to rise again later; but the perspiration does not uniformly influence temperature fluctuations. Defervescence, which is gradual, is particularly apt to be accompanied by much sweating. The perspiration has a rather typical sour odor from decomposition of fatty acids, and it gives a strongly acid reaction at first, although later it may become neutral or alkaline. It was at one time supposed that the acid perspiration represented Nature's method of ridding the system of too much acidity, but Sir William Gull effectually upset that theory by remarking that the sweat may be both acid and alkaline in different parts of the body simultaneously, the acid reaction depending solely upon sebaceous decomposition. The profuse perspiration gives rise to widespread pseudamina and miliary vesicles.

The *urine* presents the characteristics of fever. It is lessened in amount, is of red-brown color, high specific gravity, and strong acidity. It is loaded with urates, and often with uric-acid crystals, but the chlorides, as in some other fevers, are notably diminished or wholly absent while the fever persists. There is often a temporary trace of albumin with a few hyaline or granular casts, but serious nephritis is not usual. Of 300 cases at the Presbyterian Hospital, New York, 54 had albuminuria.

The *saliva* reacts strongly acid with litmus-paper, and it contains sulpho-cyanide of potassium in excess. The mouth is dry and parched, and the patient complains greatly of thirst.

Peri- and endocarditis occur so often in rheumatic patients that they deserve to rank as symptoms rather than as complications. Either may develop alone, both conditions may be present simultaneously, or one may follow the other. These inflammations are decidedly more common in youth than later, and they are not always proportionate in severity to the joint symptoms; in fact, they may antedate the latter, or in a rheumatic patient they may appear as independent attacks between those of severe arthritis. Cheadle[1] believes that acute endocarditis

[1] *Loc. cit.*

when not of septic origin is rarely attributable to any other cause than rheumatism. It is said to be present in about 25 per cent. of all cases. It is of the simple, non-ulcerative type, affects chiefly the mitral valve, and is usually recovered from, but it leads to subsequent sclerosis and deformity of the cusps, and thus constitutes one of the commonest causes of chronic valvular disease in children. The diagnosis of this condition should not be made upon a blowing systolic murmur alone, for some other cause, such as anæmia, may produce a murmur. The mitral valve is most often affected; next the aortic is the seat of vegetations or fibrinous deposit, and rarely the tricuspid or pulmonary.

Pericarditis may be simple fibrinous, sero-fibrinous, or more rarely purulent. Delirium may accompany this condition.

Myocarditis, with granular and fatty degeneration of the myocardium, is exceptionally observed, and it may be followed by dilatation of the left ventricle. It usually follows upon endopericarditis. The onset of cardiac inflammations should be suspected, even without bruit or friction sound, when such symptoms suddenly occur as præcordial pain, palpitation, dyspnœa, or an increase in temperature without exacerbation of the arthritis.

The importance of daily examination of the heart in all cases of rheumatism, even those of the mildest type, cannot be too strongly urged. From records of the 706 cases of rheumatism above referred to, treated at the Presbyterian Hospital in the past nine years, I find that there have been heart murmurs present in 329—approximately 45 per cent. These were mainly mitral or aortic systolic bruits or both, but diastolic bruits were quite common; 403 of the patients had had previous attacks of rheumatism. These figures correspond quite closely with similar data from St. Thomas's and Guy's Hospitals in London.

The *mind* usually remains clear throughout, excepting in cases of hyperpyrexia, when the patient may become delirious or dull and stupid. In a few cases with ordinary temperature delirium is present. The mental symptoms sometimes produced by too energetic dosage with salicylates or gaultherium must not be mistaken for delirium due to the disease.

Coma and *convulsions* have been recorded in some instances, but are unusual, and are apt to occur with either hyperpyrexia or some nephritic complication. *Meningitis* and *melancholia* are still more rare.

Cerebral emboli, derived from vegetations on the heart valves, may give rise to the typical symptoms of brain embolism, but this is rare. Such a case recently occurred at the Presbyterian Hospital.

Some writers, notably Lyman and Maclagen, have laid much stress upon what they term "cerebral rheumatism," a localization of the rheumatic irritation in the brain, accompanied by such symptoms as violent headache, delirium, convulsions, stupor, etc. No very definite lesions are described, however, and it is doubtful whether the condition is more than an ordinary cerebral hyperæmia or congestion, such as may develop in the course of any intense toxæmia.

The Skin.—Erythema nodosum or erythema multiform (Hebra) is sometimes present with rheumatism. Garrod, Mackenzie, and Cheadle found that 70 per cent. of cases of erythema nodosum were of rheumatic origin. Urticaria and petechiæ are occasionally seen.

Purpura is not common if Schönlein's peliosis rheumatica (or urticaria rheumatica) be excluded, as it should be, from rheumatic etiology. Its occurrence has been fully discussed in Vol. III. pp. 792, 793. When present it is often associated with erythema papulatum or marginatum.

General subcutaneous and mucous hemorrhages have been seen exceptionally.

Nodules, varying in size from a large pinhead to a third of an inch in diameter, are found sometimes in the rheumatism of children and youth, but they seem to be less often observed in the cases occurring in this country than in England. Edge[1] observed them in a boy of eight years. They are subcutaneous formations, composed of round and spindle cells, and are attached to the tendons and fascia. They feel firm, but are slightly movable. They are most abundant on the fingers, dorsal or lateral surface of the hands and wrists, but they may occur about any of the larger inflamed joints or over the vertebral spines and scapulæ. They are very irregular in their development, sometimes appearing during convalescence or between attacks, and they may develop in rheumatic subjects who have either endocarditis or chorea or tonsillitis without arthritis. They are not usually painful or tender, but sometimes become acutely inflamed. They are often very numerous, being counted by the score, and they last for several weeks or months and then disappear.

Although the subcutaneous nodules are chiefly met with in connection with rheumatism, they are not diagnostic, for they are occasionally observed independently of arthritic disease. Such nodules have been found in the periosteum also, and even, as a rarity, in the pericardium.

Follicular tonsillitis has for a long time been regarded as intimately associated with rheumatism, and there are arguments in favor of a pathological relationship between them, although it must not be assumed that all tonsillitis is necessarily rheumatic. This view has led to the extensive employment of salicylates in tonsillitis, but the results are often disappointing. Mackenzie has reported 5 cases of suppurative tonsillitis in a series of 40 cases of rheumatism.

Complications.—A complete list of the complications which from time to time have been associated with rheumatism would include a very large number of diseases. The most common of them all are endo- and pericarditis, tonsillitis, and chorea. The first two of these have been already mentioned as of such frequent occurrence as to fairly rank as symptoms.

Chorea may be a complication of rheumatism, but it is more often observed as a sequel, occurring in a subject of rheumatism between the rheumatic attacks. There has been much discussion as to the real relation existing between the two diseases, some holding that all chorea is evolved from a rheumatic diathesis, and that endocarditis occurring in a case of chorea is a manifestation of rheumatism, although no arthritis develops at any time.

Sir Dyce Duckworth has estimated that fully 78 per cent. of cases of chorea are of true rheumatic origin. German writers, however, attach much less importance to this association, and Steiner found but 4 cases of rheumatism among 252 of chorea. Osler[2] found the per-

[1] *Brit. Med. Journ.*, Jan., 1894.

[2] *Practice of Medicine*, p. 930.

centage of association not above 21. The two diseases are apt to occur at about the same season of the year, and chorea often follows within a month after a rheumatic seizure. The subcutaneous fibrous nodules described on page 959 may be present in chorea, but pericarditis belongs essentially to rheumatism, while endocarditis, leaving permanent valvular lesions, is quite common in both diseases. Cheadle,[1] in a recent discussion before the British Medical Association, stated that out of 94 cases of endocarditis he found that 62 to 66 per cent. had had previous rheumatic fever, and that 77 per cent. of his cases of chorea had evidence of endocarditis.

Next in order of frequency as complications are pleurisy, acute catarrhal pneumonia, and bronchitis. When present they usually accompany endo- or pericarditis. Pulmonary œdema may occur.

Rarer complications affect the genito-urinary apparatus, but are much less common than in gout. A mild degree of parenchymatous or interstitial nephritis is sometimes inaugurated. Peritonitis, cystitis, orchitis (Eugene Beach), urethritis, and prostatitis have been observed. Cheadle mentions thyroiditis also. Muscular atrophy has been known to follow the rheumatic polyarthritis.

One of my cases presented the signs of an acute neuritis of the right shoulder as the rheumatic symptoms were subsiding, which resulted in extreme deltoid atrophy and loss of power in the arm.

Among other infrequent complications and coincident disorders not mentioned above, which have been noticed among the 706 cases above referred to which have occurred at the Presbyterian Hospital, are the following: grippe, German measles, eczema capitis, eczema of the legs, acne, acute dermatitis, drug eruptions from salophen and phenacetine, gaultherium-poisoning, gastritis, gastric ulcer, diarrhœa (4 cases), facial erysipelas, scarlatina, ulcer of the heel, sciatica (3 cases), tic douloureux, delirium tremens, conjunctivitis, chromophytosis, mammary abscess, asthma, hydrothorax (2 cases), tuberculosis, periostitis, glycosuria, submaxillary-gland inflammation, pachymeningitis, obesity, aneurysm of carotid artery.

Thrombo-phlebitis may complicate acute rheumatism. Two such cases have been seen at the Presbyterian Hospital, and Marcel Léné[2] reports an interesting series of 7 cases.

Pregnancy in connection with rheumatism was found in 16 cases reported by Clivio to result 3 times in premature delivery, and the pregnant state was found to protract the recovery from the arthritis. Two cases of abortion in subjects of acute rheumatism have occurred at the Presbyterian Hospital.

Subacute cases of rheumatism are very often encountered, which are characterized by mildness of symptoms and tardy recovery. The temperature in such cases does not exceed 101° or 102° F., but few joints are involved, and these not severely. Endo- or pericarditis may be present, especially in the young, and anæmia is often pronounced. This type of rheumatism is apt to yield slowly to treatment, and often after a couple of months or more it merges into the permanently chronic form. The subacute type commonly occurs as a sequel to the acute, but

[1] *Brit. Med. Journ.*, Jan. 11, 1896, p. 65.
[2] *Journ. de Méd. interne*, Jan. 1, 1898, No. 1, p. 53.

the symptoms may be subacute from the beginning. This type is very common among children.

Acute Articular Rheumatism in Children.—Cheadle emphasizes the important fact that the young, whose joints are well developed and in good condition, are less assailed by the arthritic manifestations of rheumatism, whereas the heart suffers more and the skin lesions are common. In old age, on the contrary, the reverse obtains, the joints, perhaps already showing some degree of senile atrophy, are severely affected by rheumatism, which often becomes chronic, but cardiac complications are less common, and cutaneous nodules are seldom found after puberty. Children are more apt than adults to have accompanying erythema, tonsillitis, or chorea, and they sweat less. The common occurrence of rheumatic nodules among children and youth has been commented upon (page 959).

PROGNOSIS.—The course of acute rheumatism is very uncertain. Some quite severe cases recover in a week, others, less severe or subacute, last for four, six, or eight weeks, or severe cases may show some improvement under treatment, and then a recrudescence prolongs the disease for several months. The excessive anæmia is apt to prolong convalescence. Immediate relapses are not common, following about once in 40 or 50 cases, but patients who have had an attack are very liable to others, though they may be separated by intervals of years. One patient seen at the Presbyterian Hospital had had twelve attacks. I have occasionally seen relapses when patients were allowed to get up too soon after an apparent cure under treatment. Such patients may recover wholly from the arthritis, get up, and in a day or two develop endo- or pericarditis. These latter conditions will, however, very often develop in like manner, although the patient may remain in bed and under treatment for a week or more after the joint symptoms are entirely gone. The joints recover without permanent lesions of any kind, without even fibrous adhesions. Exceptionally, cases of the acute type drag along and become subacute or even chronic. Rheumatism in the subjects of chronic alcoholism is apt to run a very tedious course.

Death from acute rheumatism is rare (the mortality not being above 1 or 2 per cent.), but it may result from hyperpyrexia, myocarditis, endo- or pericarditis, or one of the complications, such as pneumonia or pleurisy.

DIAGNOSIS.—Most cases of acute rheumatism are easily diagnosed upon the history of the case, the course and distribution of the arthritic symptoms, characterized by mobility and instability, and the simple inflammatory nature of the lesions, combined with the pyrexia and tendency to cardiac inflammations. Occasionally, however, doubt may arise in regard to other affections. Secondary arthritis may develop in connection with acute injections, such as scarlatina, gonorrhœa, dysentery, puerperal or other sepsis, and cerebro-spinal meningitis. In each of these cases there are present the symptoms of the complicating disease, and in all severe septic cases there is marked tendency to joint suppuration and structural change affecting the cartilages, ligaments, etc., which have no part in ordinary rheumatism. In pyæmic states the constitutional symptoms are usually more severe than in rheumatism, and the temperature is often intermittent rather than remittent. The

typical chills of pyæmia are absent from rheumatism; the joint symptoms do not migrate, and the typhoid state is common.

Gonorrhœal arthritis will be independently described (page 973), and monoarticular arthritis is more apt to be confounded with this disease than with rheumatism.

Acute tuberculosis of the joints and osteomyelitis have given rise occasionally to fatal mistakes in diagnosis by confusion with rheumatism and neglect of prompt surgical relief.

Osteomyelitis is confined to one joint, and this fact and the involvement of the bone, epiphyses and shaft, together with the serious general condition of the patient, establish the diagnosis. Rigors are prominent, and sweating is not so.

Glanders has been mistaken for rheumatism, and so has ulcerative endocarditis with septic joint involvement.

The acute arthritis of infants is now usually regarded as an arthritic form of scurvy, and the diagnosis is discussed by the writer under that heading (page 1060). Some purulent cases are due to pyæmia.

Arthritis deformans of multiple type, acute gout, and acute rheumatisms present few difficulties in differentiation, and the salient features of the diagnosis I have given in the table upon page 988.

In scurvy, purpura, hæmophilia, etc. sanguineous effusion may cause swelling with pain in the joints which may resemble rheumatism, but these hemorrhagic diseases all have definite features apart from rheumatism. There is usually hemorrhage elsewhere than in the joints, and fever, if present, is not intense.

Treatment.—The treatment of acute articular rheumatism is conveniently subdivided into (1) prophylaxis and hygiene; (2) general management of the acute attack; (3) medicinal treatment; (4) local treatment.

(1) *Prophylaxis and Hygiene.*—Patients who have strong rheumatic inheritance or who have had rheumatic symptoms should especially protect themselves from cold, wet, excessive muscular fatigue, and exposure. They should wear the Jaeger camel-wool or other woollen undergarments to absorb perspiration in winter, or the warm linen-mesh garments which admit of evaporation of the perspiration as it forms. They should abstain from over-indulgence in sweets, and especially from malt liquors of all kinds, and should avoid constipation and functional inactivity of the liver. The occurrence of uric acid or calcium oxalate in excess in the urine should be the signal for a temporary reduction in animal food and the drinking of more fluid. In a word, such treatment applies as that recommended for lithæmia, on page 1030. The skin should be kept in good condition by daily cold bathing, followed by vigorous friction, and outdoor exercise should be encouraged.

(2) *General Management of the Acute Attack.*—When the acute attack begins, the patient should be put to bed on a smooth and comfortable mattress, which is covered with blankets, and a careful nurse should be secured, having sufficient strength to lift the patient when necessary and save him all voluntary motion. If the patient is willing to lie between blankets they are much better than sheets. Throughout the illness a bed-pan and duct should be used to save the patient unnecessary effort. The bowels should be freely opened at first by a dose of calomel, and

kept open thereafter by daily morning doses of Rochelle salts or bitter water.

The diet must be confined to milk or milk and Vichy, plain broths and farinaceous gruels, with crackers or milk toast, while the acute symptoms last. As they subside the list of foods may be slowly increased, but still be confined principally to milk and eggs and articles made from them, and farinaceous foods. It is not well to give meat for a week after the temperature and joint symptoms are normal, and then it should be allowed sparingly but once a day. Stuffing rheumatic patients with strong meat soups, meat extracts, and red meat is always harmful. At the same time, sweets must be forbidden, as well as all fermented liquors and sweet wines.

(3) *Medicinal Treatment.*—Since the introduction of salicylic acid by Stricker and Riess in 1876, and of the salicylates shortly after by Germain Sée, the latter remedies have constantly gained in favor, and, in spite of many disadvantages attending their use, have held almost the position of specifics for rheumatism against many other drugs which are constantly being experimented with. I have given extensive trial to salophen (ʒj – ʒiss per diem), phenocoll (ʒj in twenty-four hours), salol, salicine (gr. x every two hours), salicylic acid, and similar preparations, but am inclined to believe, with almost all recent writers, that sodium salicylate is the best drug for the majority of cases, although much depends upon its mode of administration. Its chief value is in relieving the severe articular pains, and as the joint symptoms subside its use should be gradually discontinued, for otherwise it tends to increase the anæmia and perspiration. It fails to materially prevent or influence cardiac symptoms, possibly because the heart cannot be placed at rest like a joint. It fails to prevent relapse, and it may cause toxic symptoms, such as vomiting, tinnitus aurium, and temporary deafness or vertigo, although it is less apt to do so than salicylic acid. The drug is best given in wafers or gelatin capsules, followed by the ingestion of a tumblerful of fluid of some sort, or the powder may be dissolved in lemonade or peppermint-water. In a bad case it is well to begin with large doses, 15 or 20 grains every two or three hours for a few doses, to be reduced to daily doses of 10 or 15 grains given three or four times in twenty-four hours as pain subsides. Lesser initial doses are often disappointing and fail to relieve. Effort should be made to secure a fresh and reliable preparation, as it deteriorates on keeping. Some patients cannot bear salicylic remedies in any form, and, as often is the case with drug idiosyncrasies, small doses are as apt as large ones to be toxic. I have seen four doses of salicylic acid of 5 grains each produce severe vertigo, which was felt whenever the patient turned in bed, and caused staggering when walking for two or three days after the drug was discontinued. Epistaxis may be produced. In exceptional cases either sodium salicylate or salicylic acid may produce maniacal delirium. The same is true of gaultherium, which, however, ranks next to sodium salicylate in usefulness, and is often better borne by children. It should be given to adults in 5-minim capsules (dose three or four capsules) or in emulsion, in milk or mucilage of tragacanth. The toxic symptoms of salicylates may be averted by dilution and by giving sodium bromide (gr. xx) in combination. If the salicylates cause gastric irritation,

Erlanger suggests their use per rectum with 10 minims of tincture of opium.

Salicylic acid is promptly converted, by combination with sodium in the blood, to a salicylate of that metal.

The salicylates produce a decided increase in the uric acid and urea-elimination by the kidneys. Sodium salicylate is not believed to act as an antiseptic unless it is split to salicylic acid again; and one theory of its action is that this latter process occurs in the tissues through influence of CO_2, which promptly decomposes it, forming a nascent acid of strong antiseptic power.

When these remedies prove beneficial they usually mitigate the articular pains within four or five hours, and after two or three days the swelling abates, the patient can move with more freedom, and the temperature gradually falls. If the dosage is reduced too much, or discontinued before four or five days have elapsed after marked improvement has occurred, the symptoms are apt to return as a genuine relapse.

The salicylates often afford a valuable means of diagnosis of rheumatism from gout, the acute variety of arthritis deformans and pyæmic polyarthritis, in none of which latter diseases they have so striking an effect as in rheumatism. The salicylates are not only valuable in polyarthritis, but also in cases of rheumatic iritis they are of more use than local remedies. I have lately seen two cases of recurrent iritis originating between rheumatic arthritic attacks which yielded to no other remedies, but improved in a few hours upon salicylate treatment.

Patients who cannot tolerate either of the foregoing remedies may take salophen (gr. xv every three hours) with some benefit, or phenacetine or antipyrine may be employed, but, as a rule, the two latter remedies are too depressing to the heart.

The *alkaline treatment* of rheumatism, while deemed of less importance than formerly, cannot be disregarded, and the belief is still very general that it tends to reduce the liability to cardiac inflammations. It is best combined with the salicylate treatment, and, as with that method, it is best to give large doses for a day or two, or until the urine becomes distinctly alkaline, and then taper off. The favorite remedies are potassium or sodium bicarbonate in doses of gr. xx or xxv, or potassium citrate in like amount, given every three hours in effervescing mixture with lemonade or as powder in wafers. Ammonium chloride with potassium acetate is still used by some clinicians, although less in vogue than formerly, and it is apt to disorder the stomach. It must be remembered that the potassium salts, as well as the salicylates, are both cardiac depressants, and their combined use must not be prolonged. By gradual reduction of dosage after the first six or eight doses are taken the danger of heart failure is reduced to a minimum; but directions should be very explicit on this point. The alkalies are believed to increase the excretion of uric acid in rheumatism.

Moderate *fever* up to 104° F. may be controlled by sponging with cold alcohol and water in equal parts, but any such temperature as 106° F. or above requires more energetic measures, and the full cold bath at 65° or 70° F. must be given, if possible, for fifteen minutes and repeated every two hours until the temperature falls to the safety limit of, say, 103° F. No halfway measures suffice, and, despite much pain, the

patient must be got into the tub. The only cases of extreme hyperpyrexia (110° F.) which have been saved have been treated in this way. A full hypodermic injection of morphine may be given to ease the pains while the patient is being moved, and cardiac stimulants may become necessary. The use of the coal-tar "antipyretics" in these cases cannot be too strongly condemned.

The development of *peri-* or *endocarditis* should be met by the use of an ice-bag over the præcordium, and if the pulse is full and bounding small doses of tincture of aconite are serviceable. If there is much pain over the heart, restlessness, or dyspnœa, codeine, or moderate doses of Dover's powder or of morphine should be given. Beyond the use of these remedies it is usually best to let the heart alone.

(4) *Local Treatment.*—The proper local treatment of acutely inflamed rheumatic joints affords marked relief. The first consideration is absolute rest of the joint, and the second is the application of remedies to soothe the pain, and, if possible, reduce the inflammation.

Rest.—The joint should be at once enveloped in a thick layer of cotton batting covered by oilsilk, and bound to a splint with a few turns of a light gauze bandage. This simple measure alone often gives the greatest relief from the pain. It serves the threefold purpose—(1) of fixing the joint and preventing the friction of the inflamed surfaces; (2) it protects from jarring or accidental pressure; and (3) it maintains a uniform temperature about the joint. If the elbow or knee is involved, the parts above should be immobilized; if it be the shoulder which is affected, the arm should be bound to the side; if the wrist, it must be laid upon a padded arm-and-hand splint. Smaller joints must be similarly protected. When the joints of the lower extremity are affected, it is often desirable to lay sandbags on either side, for in this manner perfect rest is secured and the patient is less likely to injure himself by turning in his sleep. A good supply of pillows of different sizes are of great service in easing the constrained positions of the body. As a rule, in the acute stage of rheumatism moderate flexion of the larger joints is the position in which they are most comfortable if duly supported. As the inflammation is apt to pass rapidly from one joint to another, much skill is required in the nursing of the patients in order to mitigate their pain.

There is but one objection to the above suggestions, and that is the impossibility of making local applications to a joint while it is enveloped in cotton; but many of these applications do not require frequent repetition, and may be made once for all day, or, if desirable, a hole may may be left in the dressing or the cotton may be omitted and the joint is simply bound above and below to a splint.

Measures adopted to Relieve Pain and Reduce Inflammation.—Heat and Cold.—As a rule, warm applications are preferred to cold, and in most cases the warmth retained by the cotton, as above described, is all that is necessary. Occasionally flaxseed poultices give relief, more particularly to the less acutely inflamed joints.

Blisters.—It seems perhaps paradoxical, but when the larger joints are most acutely inflamed few applications give as much relief as one or two active cantharidal blisters applied directly over the focus of inflammation. They should be large—in case of the knee two to three

inches square—and sufficiently active to withdraw an ounce or two of serum, after which the blister is to be punctured and dressed and the joint enveloped in cotton.

Lotions.—The number of evaporating lotions, liniments, and embrocations of all sorts recommended for rheumatism is very large. They constitute a chief part of the sales of proprietary and patent medicines, many being worthless, some relieving one case, some another.

As might be expected, most of these applications are of more benefit in subacute and chronic cases, where their favorable use may be accompanied by friction, than they are in very acute inflammation. For the latter, after extensive trial, I have become convinced that two remedies excel all others—viz. oil of wintergreen and guaiacol. Their application is often followed by immediate and lasting relief, although like all remedies, they may sometimes fail. The gaultherium oil is applied by soaking a piece of lint freely with the undiluted oil, and the joint is then encased in batting. The application may be renewed once or twice in twenty-four hours.

Guaiacol I apply in a similar manner, first diluting it with an equal part of glycerin. It is even more efficacious than gaultherium, but it possesses an odor not unlike creasote, which is more disagreeable to many, and which has a most peculiar penetrating and persistent property. Efforts to overcome this objection are thus far futile. If the application should cause itching or slight burning, the guaiacol may be further diluted. The drug is well known to produce a remarkable fall in temperature when rubbed into the skin in doses of half a drachm or more, which may be accompanied by collapse. Dana has reported a drop of 9° F. in the temperature in a case of enteric fever which was produced in this manner, and I have caused an even greater fall experimentally in animals; but for some reason, in rheumatism I have never found any marked thermic reaction, and do not hesitate therefore to apply the remedy quite freely. On only two or three occasions has it seemed to reduce the general body temperature, and then to the extent of only 2° or 3° F. I have used it often, and have found it most efficient. Methyl salicylate has been lately very highly recommended. 50–100 drops are applied to the joint.

I have sometimes employed salicylic-acid ointment with success. Bourget found this acid in the urine twenty-four hours after its local application, proving that much of it was absorbed. He prescribes salicylic acid, lanolin, oil of turpentine, *āā.* 10 parts, with lard 70 parts.

Singer of Vienna, believing in the microbic origin of rheumatism, claims that intravenous injection of corrosive-sublimate solution is abortive and preventive of cardiac complications, but the method is too heroic for general use.

Convalescence demands watchful care. After a severe attack the patient should not leave his bed for fully a week or ten days after subsidence of all symptoms. The diet should be as nourishing as milk and eggs, cereal foods and fresh vegetables, with fresh fish, chicken, etc., can make it, and anæmia should be combated with iron and cod-liver oil.

Enthusiasts in therapeusis should remember that acute articular rheumatism is a self-limited disease, and that most cases, left strictly to themselves, recover spontaneously in from one to six weeks.

Mild, subacute, and *afebrile cases* are best relieved by alternate douching with cold and very hot water, by rubbing with liniments—such as linimentum saponis, camphoræ, terebinthinæ, or belladonnæ—or by painting with iodine or methyl salicylate. If the inflammation shows any tendency to increase, the joint should be protected as above described. Obstinate joints with persistent pain may improve faster by being encased in plaster of Paris for a week or ten days, to secure absolute rest. This is especially useful for the knee- or ankle-joint. Bursitis is relieved by strapping with strips of rubber plaster.

As tonics, quinine, iron, cod-liver oil, and the iodides are recommended.

CHRONIC RHEUMATISM.

Definition.—Chronic rheumatism is a disease having the same etiology with acute rheumatism, but characterized by gradual and permanent changes in the joint structures, mainly of the nature of fibrous thickening and contraction, producing more or less deformity.

The disease may result exceptionally from an acute or a subacute attack, but it often begins quite insidiously as a chronic disorder.

Etiology.—The disease has the same predisposing causes with the acute form of rheumatism (see page 950), but is more common in females than in males, and rarely develops before the fortieth year, the common period being between the fortieth and sixtieth years. Bad hygiene, exposure to cold and dampness, malnutrition, and debility, all are important factors: likewise heredity and such occupations as lead to the disproportionate use of special joints, as certain fingers used constantly by sewing-women and tailors.

Pathology.—The pathology of chronic rheumatism is not known to differ in any manner from that of the subacute and acute types (see page 951). It possibly concerns an attenuated toxin or some obscure change in the fibrous tissues, reducing their functional vigor, rendering them less acutely susceptible to the rheumatic excitant.

Pathological Anatomy.—The gross lesions of this disease are not prominent. They are confined to moderate thickening and distortion of certain joints, less pronounced usually than in either chronic gout or arthritis deformans. The phalangeal joints of the fingers are commonly affected; so also are the knees. Other joints become involved in long-standing cases. There is often some degree of bilateral symmetry in the joints diseased, but this is usually less striking than in arthritis deformans.

In mild cases the joints exhibit only a moderate degree of synovial injection and effusion, for the disease shows more tendency to attack fibrous structures than serous membranes, therein differing from the acute type. In more marked cases there is fibrous thickening of ligaments, synovial membranes, capsules, and sheaths of tendons. More or less contraction of the fibrous tissue combines with the thickening to produce deformity or distortion of the joints. The thickening is usually most prominent at the sides, over the lateral ligaments, and here, too, the tenderness is apt to be localized. Superficial erosion of opposing

cartilaginous surfaces is present in advanced cases, but is not common. Some degree of fibrous ankylosis finally immobilizes certain joints.

Like arthritis deformans, but unlike chronic gout, there is a marked absence of lesions aside from the arthritides, and the tendency to cardiac inflammations, so strong in acute rheumatism, is very slight in the chronic form.

Symptoms.—The symptoms are mainly confined to the joints, and there is little tendency to involve the heart. The fingers are often distorted by the contracture of the tendons or ligaments, so as to produce moderate deflection, especially of the distal phalanges, or a flexion and extension of the alternate phalangeal joints is observed, which Jaccoud included in his description of *rheumatisme chronique fibreux*. The deflection is often said to be ulnar, but it is quite as apt to be radial, and at the present time I have four or five patients under treatment in whom both deformities are decided, the finger tips being approximated to each other. The general ulnar deflection ("seal-fin" type) of all the fingers of a hand is not often seen, as it so commonly is in arthritis deformans. The knees become stiff and thickened, especially at the sides, and the legs can be only partially extended.

A fibrous crackling may be both felt and heard upon attempting to forcibly extend the joints. Redness and decided œdema are usually absent or slight in degree. Often the joints, although much deformed, are not painful unless overworked or injured or subjected to fresh exposure. In other cases pain is severe, especially at night, and is accentuated by movement. The pain and stiffness are more influenced by changes in the weather than is the case in any other joint ailment, and many sufferers from chronic rheumatism can predict approaching storms twenty-four hours beforehand with astonishing accuracy by the temporary increase in intensity of the symptoms which they experience. Atrophy of the muscles about the affected joints may occur, both from disease and from trophic insufficiency.

Constitutional symptoms are practically absent. Slight fever, 101° F., may accompany an exacerbation of the joint symptoms, but even this is uncommon. The urine remains normal. The general health continues remarkably good so long as the patient can move about, but sometimes anæmia and debility occur from protracted suffering and insomnia caused by pain or dyspepsia. I am at present caring for an old lady of eighty-eight years, whose fingers are so deformed by distortion and pseudo- or fibrous-ankylosis as to be quite helpless, yet who walks about and displays astonishing activity and vigor of mind and body.

Prognosis.—The sufferer from chronic rheumatism once well established never recovers, yet the disease is not fatal, and patients die of some intercurrent malady. Cases seen very early may be much improved and held in check for many years by treatment.

Diagnosis.—The diagnosis is not difficult. The disease has mainly to be differentiated from arthritis deformans, and in some cases from chronic gout. The diagnostic features of these diseases are contrasted in the table which I give upon page 988. The finger deformities of Dupuytren's contraction and of old injuries are asymmetrical, and careful examination of the affected joints will differentiate them, in connection with the history and progress of the case.

TREATMENT.—*Medicinal remedies* are of very little use. I have sometimes seen a measure of improvement in cases in which gout could be positively excluded by the use of wine of colchicum ♏x, and potassium iodide gr. x, *t. i. d.* I have employed piperazin internally in 15-grain doses in a considerable number of cases, but the results have not been sufficiently decisive to recommend it, although it is of service externally. In some cases the simple alkalies or a course of alkaline waters may prove beneficial, and debilitated patients need tonics, of which cod-liver oil and arsenic are the best.

Local treatment is much more satisfactory, and will often produce decided improvement, or check the further progress of the disease, especially if resorted to early. The patient must be convinced that any such treatment must be persevered in, as fully a month may elapse before decided results are obtained. Hydrotherapy yields the best results, and recently I have seen great benefit from the local hot-air or hygothermic treatment. The usual course consists in applying a stream of air heated by a lamp to 240°-280° F. for forty-five to sixty minutes, the hand or knee being suspended in a box which has been ingeniously adapted by the inventor to fit about any joint by means of adjustable sides. Strong local perspiration is produced, with dilatation of the peripheral vessels and improvement in the activity of the local circulation, which promotes absorption. Great relief from pain and stiffness is often experienced, especially if the application be followed by Swedish movements. Alternate douching with cold and very hot water, or wrapping the joints in flannels wrung out in steaming water, acts in a similar manner. If the knees are involved, occasional cantharides blisters or the Paquelin thermo-cautery should be applied, but in elderly or debilitated subjects care should be taken not to irritate the skin too much. The wearing of woollen knee-caps and gloves gives comfort, as does the packing of the swollen joints in cotton wool and oilsilk.

Galvanism is of doubtful value, but general massage is useful.

I have sometimes derived benefit from encasing the joints for several hours in a 2 per cent. solution of piperazin in water. Other topical applications to relieve pain are tincture of iodine, iodine ointments, ichthyol ointment (gr. xl, with lanolin ʒj), guaiacol and glycerine in equal parts, and methyl salicylate.

Hygienic measures must not be neglected. It matters little what diet the patient has, so that it is ample, nutritious, and easily digestible. It should comprise chiefly meats, fresh vegetables, and cereals. Sweets, pastry, sweet wines, and malt liquors should be avoided. Claret or well-diluted whiskey is allowable. Tea and coffee may be drunk.

Great care should be taken to avoid exposure to cold and wet or fatigue and injury of the joints.

Patients whose means permit do well to spend their winters in a warm and equable climate, like that of Egypt or Tangiers, or at least to spend the early spring months in Florida or Southern California. Innumerable spas, both in this country and in Europe, lay claim to the improvement afforded chronic rheumatics, and a course of from three to six weeks at one of them will often accomplish much by staying the progress of this incurable malady. Among the best in this country for this class of cases are the sulphur springs of Sharon and Richfield in

New York, and the Hot Sulphur Springs of Virginia, of Arkansas, and of Glenwood in Colorado. In Europe, Aix-la-Chapelle is one of the most successful resorts, as the system of douching and massage practised there is very thoroughly understood.

MUSCULAR RHEUMATISM, OR MYALGIA.

Definition.—Muscular rheumatism, or myalgia, is that variety of rheumatism in which the symptoms are localized in various muscles, with usually little, if any, constitutional disturbance. Various names, more or less descriptive of the part of the body affected, are applied to this disease. Such are chiefly lumbago, torticollis, and pleurodynia.

Pathology.—The pathology of this disease is believed to be identical with that of acute articular rheumatism. W. Leube believes that the disease may be epidemic at times, and it is possibly due to an attenuated form of the toxin of the articular type of the disease.

Pathological Anatomy.—The morbid anatomy of myalgia is imperfectly understood, as the disease is never fatal, and there is some doubt as to whether the lesions are to be found in the muscles themselves or their nerves. It is generally believed, however, that there is an acute inflammation affecting certain voluntary muscles, their tendon-sheaths, and fascia, and that portion of the neighboring periosteum to which they may be attached. In fatal cases of acute articular rheumatism in which the muscles have been involved their fibres have exhibited swelling and granular degeneration with vacuoles. In severe cases decided muscular atrophy has been observed, indicating a tropho-neurosis, but this condition is rare as a result of simple myalgia. It may be prominently seen in the deltoid muscle when the shoulder joint is involved. In chronic myalgia there may be round-celled infiltration, increased nuclei in the muscle-fibres, and increased connective tissue around the fasciculi.

Etiology.—The etiology is much the same as that of the articular type of rheumatism (see page 950), but those muscles are especially susceptible which have been weakened by strain, injury, such as bruising, or by exposure to cold and wet, as from becoming chilled while actively sweating. Exposure to a draft is apt to produce an attack, as when one sleeps, with the muscles of the neck or lumbar region uncovered, near an open window.

Gouty and lithæmic persons are very apt to have myalgia. Heredity is a potent factor. Occupations involving exposure of various kinds are predisposing, lumbago being very often developed in common laborers who have strained their backs in lifting or digging in damp soil.

From the influence of occupation the disease is most often found among adults and males, but it may occur in children, especially as torticollis. One attack is frequently followed by others. The muscles affected are usually the larger ones concerned in effort, such as the lumbar, deltoid, sterno-cleido-mastoid, rectus, scapular, pectoral, intercostal, etc., but almost any of the larger voluntary muscles may be

attacked. Among 30 cases treated at the Presbyterian Hospital, New York, were several in which the symptoms were observed in muscles of the thighs, calves of the legs, and soles of the feet.

SYMPTOMS.—The chief symptoms are local pain and tenderness. There is little if any visible swelling or redness or heat.

The pain varies from mild soreness increased by motion, to violent, lancinating, stabbing sensations, or a dull, constant aching, a feeling as if the muscles had been bruised with a mallet. The pain is stationary and does not migrate as in the arthritic type. It is often worse at night, and may be influenced by barometric or thermometric changes in the atmosphere. Uniform pressure may relieve it, but irregular pressure may elicit much tenderness and be almost unbearable. The patient assumes whatever position will give most ease by relaxation of the affected muscles, as every voluntary contraction of them may cause excruciating pain, causing him to cry out. Severe cramps may occur.

The local symptoms often begin with great suddenness. I have known laboring men to be seized so quickly with lumbago while at their work as to be unable to stand, sit, or move. There is no true paralysis, but simply incapacity to use the muscles on account of the soreness and pain.

In *torticollis* or wry-neck the antero-lateral region of the neck is affected, and the patient rotates the body to avoid turning the head. This condition is usually unilateral, whereas lumbago is bilateral.

Pleurodynia is also unilateral, oftenest upon the left side, and is very painful, for the respiratory muscles affected can be less completely set at rest, and coughing or sneezing causes agony. The intercostal muscles are those oftenest affected in this type, but the pectorals and serratus magnus are often involved, so that the natural respiratory movements are much restricted.

Cephalodynia is a less common variety of myalgia in which the muscles about the head and scalp are involved.

Constitutional symptoms there are none, excepting slight fever which occasionally occurs in transient form. According to W. Leube, about one third of the cases have slight elevation of temperature (101° to 102° F.) for a day or two. The highest temperature that I have seen was 103.8° F. Cardiac complications are rare. Leube found transient heart-murmurs in about one-sixth of his cases. I have observed cardiac palpitation and oxaluria in some instances.

PROGNOSIS.—Most patients recover in a few days, or at the most a week, but cases of exceptional severity or occurring in debilitated or highly rheumatic subjects may prove very intractable and linger for several months.

DIAGNOSIS.—The diagnosis is commonly easy, and is based upon the absence of constitutional symptoms and the pain which is increased by muscular contraction more than by local pressure.

Protracted lumbago must be differentiated from the pain of abdominal aneurysm, and which sometimes accompanies acute nephritis, but this presents little difficulty.

Intercostal neuralgia is diagnosed from pleurodynia by the tenderness along the nerves and the greater paroxysmal character of the pain in the former affection. With pleurodynia the chest should be care-

fully auscultated to find pleuritic râles, and periostitis of the ribs must be excluded.

Myalgia is differentiated from severe neuritis in general by the presence of stiffness and immobility, rather than paralysis and contracture, and the absence of tenderness along the nerves, and dysæsthesiæ.

Treatment.—*Prophylaxis.*—Since one attack predisposes to another, preventive treatment should consist in avoidance of exposure to cold and wet, the sudden checking of perspiration, muscular fatigue or strain, etc. Rheumatic and gouty patients should observe the hygienic rules elsewhere detailed (pages 962, 1019). They should wear woollen under-garments in winter, and keep the skin in good condition by cool bathing and friction. An anticipated attack may sometimes be averted by a Turkish bath followed by massage.

Local treatment consists in the application of counter-irritants and soothing liniments. Of the latter, turpentine, chloroform, and belladonna are the most useful. A hypodermic injection of morphine placed in the affected muscle gives instant relief, but it is best to avoid the use of this remedy in all recurring maladies unless the pain be unendurable. Anders has used a 20 per cent. ointment of salicylic acid with benefit. Among local applications I have found none so effectual as the Paquelin thermo-cautery, applied gently and rapidly over a large surface. If the application is not too vigorous, it can be repeated several days in succession if need be, but its effect is often magical. Hot-water bottles, hot flaxseed or mustard poultices (one part of mustard to six or eight of flour) may be applied with much benefit. Turpentine stupes also give relief. In obstinate cases cantharides blisters may be used, but hot-water douching is better. For lumbago acupunture has been tried, and deep hypodermic injections of distilled water probably act in a similar manner as counter-irritants. Galvanism sometimes affords relief. Common domestic remedies are porous plasters and ironing the muscles with a hot flat-iron through a thick piece of paper or flannel. As improvement proceeds massage should be practised.

Medicinal remedies are not often indicated. Some acute cases are benefited by the salicylate of sodium (page 963), and in very protracted cases the general tonics, such as arsenic, nux vomica, and cod-liver oil are serviceable. Potassium iodide, sulphur, and guaiacum have all been extolled, but their value is questionable. The coal-tar products, antipyrine, phenacetine, etc. are usually disappointing. Good, nourishing mixed diet of plainly cooked food, with meat once or twice a day and plenty of fresh vegetables, is desirable.

GONORRHŒAL ARTHRITIS.

By W. GILMAN THOMPSON, M. D.

Synonyms.—Gonorrhœal synovitis; Gonorrhœal rheumatism.

Definition.—An acute infectious disease characterized by a specific urethritis (due to the gonococcus of Neissen), and by localized inflammation in one or more of the larger joints of the extremities.

Etiology.—Although often erroneously called gonorrhœal rheumatism, this disease has no etiological relation whatever with acute articular rheumatism, being caused exclusively by the migration of the gonococcus or the transference of its irritating toxins from the urethra to the joint. In a number of instances the gonococcus itself has been demonstrated in the joint; in other cases where it has not been found it may have been previously present and destroyed, or the germ products have been conveyed by the vessels from their site of development in the urethra to set up a fresh irritation in the fibrous structures of the joint.

The etiology of gonorrhœal arthritis is therefore that of gonorrhœal urethritis. It is accordingly much more prevalent in young unmarried males than in females. It may, however, be acquired by any one who has been infected with gonorrhœa. The villainous superstition held by certain of the lower social classes, especially ignorant Italians, that a man can rid himself of a "dose of the clap" by imparting it to a young child, results sometimes in infection in extreme youth. Thus Chiaiso[1] and Isnardi of Turin have reported a case in a child of ten years, and Richardiere[2] of Paris reported cases in girls between five and ten years, and says that he has even known of its occurrence in a child before twenty months of age. Illustrating the other extreme of life, I have now under treatment a woman of fifty-eight years, who contracted a very obstinate attack from an erring husband. Conditions which influence the development of acute articular rheumatism, such as exposure to cold and wet, have little or nothing to do with gonorrhœal arthritis, but injury to a particular joint may have some influence in localizing the inflammation there.

It is the belief of a few clinicians of experience that this type of arthritis may complicate a simple non-specific urethritis in the male, or even leucorrhœa or menstrual disorders in the female, but such cases must be very rare.

Pathological Anatomy.—The affected joint presents the appearance of common synovitis, and the capsule, synovial membrane, and ligaments become thickened and inflamed. There is often effusion into the joints, but although the fluid may appear turbid with leucocytes and fibrin, it is rarely purulent. I have often made exploratory aspirations in protracted cases, thinking pus might be present, but have seldom

[1] *Gaz. méd. di Torino*, Feb. 15, 1894. [2] *L'Union médicale*, Oct. 26, 1893.

found it. It is more apt to appear in the wrist-joint than any other, for some unknown reason. The gonococcus, first found in the fluid by Petrone and Kammer, is present in only a limited number of instances, and no special toxins have yet been isolated, nevertheless there seems good reason to believe that it is the sole cause of the arthritis (Finger, Councilman), either directly or through absorption of its toxins from the local inflammation in the urethra. When pus is found in the fluid, it is due to pyogenic organisms or toxins which have made their way in with the gonococci. Staphylococci, streptococci, and pneumococci have all been discovered within the joint cavities. In most cases the inflammation is not confined to the fibrous structures within the joint, but becomes periarticular, and extends for some distance along the tendon sheaths, or sometimes the periosteum, with considerable infiltration and œdema. In this manner the gonorrhœal joint acquires a somewhat different appearance from the acute rheumatic joint, the swelling of the former being more fusiform, less circumscribed, extending for some distance beyond the joint, and gradually tapering off. In this way the entire dorsum of the hand and the fingers to their tips become uniformly swollen, so that the natural curve of the wrist is obliterated, and the ankle is affected in the same way. The tissues eventually acquire a much more "boggy" feel than in rheumatism, and one is often deceived by the impression of apparent fluctuation, which, however, does not usually exist except in a bursitis about the knee. The elbow-joint may be so swollen as to make the arm appear like a cylinder, the natural bony outlines being wholly obscured. In one form of gonorrhœal arthritis, usually of very chronic type, there is much effusion, constituting a true hydrarthrosis. Such cases, as I have seen them, are commonly localized in the knee, whereas in the wrist and ankle the inflammation tends more toward œdema.

As the inflammation subsides, the joint recovers less completely than from rheumatism, and fibrous adhesions and thickenings remain for long, and may cause much impairment of motion or even pseudo-ankylosis.

Symptoms.—The disease occurs in an acute form, usually rather mild, with a tendency to involve several joints simultaneously, and in a severe and more chronic form, commonly confined to one joint and very rebellious to treatment.

Local Symptoms.—The joint or joints may become inflamed within four or five days of the development of the gonorrhœal urethritis, or may not appear inflamed for several months or until the disease has run on into a chronic gleet, but the usual time for the arthritic complication to appear is at about the fourth or fifth week after the urethritis begins. The patient often denies that he has any discharge, and in women especially the primary gonorrhœa is often overlooked. Usually but one joint is affected, but there is an acute variety of the disease in which a number of joints are simultaneously involved. The knee-joint is most often affected, and next to it the wrist or ankle.

König[1] of Berlin states that in males the knee is most often attacked, in females the wrist, but I have had a number of cases recently in my wards in Bellevue Hospital in which these relations are reversed, so that sex has seemed to me to have little to do with the matter.

[1] *Ann. of Surg.*, Mar., 1897.

The finger-joints are not often involved except by extension from the wrist, but such extension usually follows along the tendon sheaths of the finger muscles, omitting the smaller joints themselves. In a similar manner the dorsum of the foot becomes swollen, red, and tense, but the toes usually escape. Other joints may, however, be affected, and I have seen bad cases localized in the elbow or shoulder, and one in the sterno-clavicular articulation. In one or two cases seen at the Presbyterian Hospital the plantar fascia or the heels have suffered. The inflammation is not fleeting, as in acute articular rheumatism, but once established in a joint remains for weeks or often for three or four months or more. There is but moderate redness, and the tenderness is usually less exquisite than in rheumatism. The pain, however, is usually constant and intense, and is apt to be worse at night, and is such as to prevent all motion of the affected limb. If the wrist is involved, the fingers are usually immobile. Bursitis is common, especially about the knee. A number of my cases have required aspiration. In a protracted case with much fluid it is well to perform at least an exploratory aspiration to make certain that the fluid is not purulent. The pain usually subsides long before the swelling, and a very œdematous joint after two or three weeks can often be gently massaged without causing much suffering.

There is no uniform relationship between the amount of urethral discharge and the local joint symptoms, but in some cases it has been observed that as the discharge lessened the joint became worse, and *vice versâ*, a statement which some, but not all, of my cases would seem to corroborate.

Constitutional symptoms are usually mild. Fever is moderate (101° to 102.5° F.) and subsides some time before the joint has recovered. Hyperpyrexia does not occur, and sweating is not common. Anæmia is marked, and the patients show considerable pallor. There may be anorexia and constipation or other slight digestive disturbances, mainly produced by the confinement, discomfort, and pain—conditions which may also produce restlessness and insomnia.

Complications are not common. The heart is rarely involved and the other viscera escape, but exceptional cases of a malignant type of endocarditis have been reported. Hering found the gonococci in endocarditis, and Councilman found them in the cardiac muscle. Such cases are very uncommon.

Iritis or sclerotitis may occur in one or both eyes, or there may be catarrhal ophthalmia, which, however, is not to be confounded with the direct suppurative infection produced by carelessly getting gonorrhœal pus into the eye.

Prognosis.—Recovery is very tedious, but it is often complete, and I have seen many very hopeless looking joints eventually become normal. Once cured, the disease may return even without a fresh gonorrhœal infection, and Osler reports a case which with relapses lasted for ten years, but usually recovery is not followed by relapse without re-infection. Broadhurst reported a case of ankylosis occurring after three attacks of gonorrhœa, and fibrous thickening may remain for a long time, producing stiffness and immobility, or adhesions may require to be broken up under an anæsthetic.

Diagnosis.—The diagnosis of gonorrhœal arthritis is not difficult in the presence of a gonorrhœal urethritis. Doubt may arise as to whether the case is an example of the uncommon type of monoarticular acute rheumatism, but certain symptoms of rheumatism are lacking from gonorrhœal arthritis, such as the greater constitutional disturbance, sweating, fever, cardiac complications, etc. Moreover, there are certain characteristics of the gonorrhœal joint inflammation, such as its greater diffuseness, œdema, prolonged course, tendency to involve sheaths of tendons, etc., which are very distinctive.

Pyæmic synovitis can be differentiated by the history, the absence of urethritis, and the constitutional symptoms of chills, fever, sweating, etc.

Gout of the knee-joint—a rare type without other involvement—might be confounded with gonorrhœal synovitis in its early stages, but the history and course of the two diseases is wholly different. In gout tophi are found sooner or later, and the constitutional and visceral symptoms are distinctive.

Treatment.—The treatment is chiefly local, and the usual antirheumatic remedies, salicylates, gaultherium, alkalies, potassium iodide, etc., have very little influence, and some of them, like the iodide, may make the urethritis worse. Morphine may be required for the first few days to relieve the pain. I have had the most gratifying results from the continuous application of cold in the form of icebags or "ice-poultices," made by mixing pounded ice with flaxseed meal, by which means the ice does not melt too rapidly and the cold can be uniformly and conveniently applied. For the first few hours the pain may be increased, but relief is almost sure to follow, and the treatment should be persevered in for weeks together, if need be. Guaiacol and glycerine in equal parts, or a 40 per cent. ointment of ichthyol with lanolin may be smeared over the inflamed area. A. Dezanneau[1] strongly recommends the topical application of turpentine with an equal part of a watery emulsion of green soap. Absolute immobility of the joint must be secured by splints, or in the case of the knee, sand-bags should be used, while the weight of the bed-clothing is kept off with a "cradle." As the very acute symptoms subside, it may be well to encase the joint in plaster of Paris for a week or ten days, but not for long, lest adhesions form which are difficult to eradicate. This formation can usually be prevented when pain is gone and the swelling has in great part subsided by abandoning cold and massaging the joint after soaking it for ten minutes in water as hot as can be borne. This should be done two or three times a day. Injections of bichloride of mercury have been used, but I have had no experience with them.

Tonics are usually needed, such as cod-liver oil, iron, and the simple bitters. The diet may be such as the patient desires, as it matters little what is eaten, so long as digestion is good and the bowels are kept free by salines. Inflammation of the conjunctivæ should be treated by cold applications, protection from light, the instillation of a saturated aqueous solution of boric acid, and if iritis is present atropine must be used.

[1] *Le Scalpel*, Oct. 4, 1896.

ARTHRITIS DEFORMANS.

By W. GILMAN THOMPSON, M. D.

Synonyms.—Rheumatoid arthritis; Rheumatic gout; Osteo-arthritis. French, rhumatisme noneux. The better name, arthritis deformans, was given by Virchow.

Definition.—An exceedingly chronic deforming joint disease, exhibiting slowly progressive changes in the synovial membranes and articular cartilages, with extensive osseous periarticular growths. Quite exceptionally the disease runs a somewhat acute course.

Etiology.—This disease bears no definite relation to either gout or rheumatism, as originally supposed by Charcot [1] and others, and the synonyms suggestive of such relationship should be abandoned. It is an independent disease of unknown origin, although its coexistence with either chronic rheumatism or gout is possible. Of the latter, William Ewart [2] writes: "The coexistence with gouty deposits of changes resembling osteo-arthritis led Fuller and Hutchinson to believe in a blending of the rheumatoid with the gouty element, at least in some cases. This is the view also taken by the writer [Ewart]. Mixed forms may arise in which the osteo-arthritic changes are conspicuous, and uratic incrustations may also have occurred. This in no way invalidates the statement as to the independent character of the two diseases; indeed, their dualism always asserts itself either clinically or in their anatomical appearances."

This is the representative modern view, but Haygarth [3] in 1805 believed that the disease was "clearly distinguishable from all others by symptoms manifestly different from the Gout and from both acute and chronick Rheumatism." E. Wynne [4] has demonstrated that even in those severer cases of gout, in which there is cartilage erosion and lipping of the periphery of the bones, there are always pathological features which are absolutely distinct from those of rheumatoid arthritis. It may be added, in conclusion, that it is quite possible to believe that in some cases repeated attacks of acute rheumatism or gout may, like other injuries, by the insult offered to the joints, pave the way for the localization in them of subsequent trophic changes peculiar to arthritis deformans, and this in nowise calls for the belief of a common origin of any of these diseases.

J. K. Mitchell originally suggested the possible nervous origin of the disease, and Senator,[5] Ord,[6] Sir Dyce Duckworth,[7] Weber,[8] Garrod,[9]

[1] *Thèse de Paris*, 1853.
[2] *Gout and Goutiness*, p. 161.
[3] *Nodosity of the Joints*, 1805.
[4] *Lancet*, 1889, vol. i. p. 933.
[5] *Von Ziemssen's Cyclopedia*, vol. xvi., 1877.
[6] *Trans. Clin. Soc. of London*, 1879, vol. xiii., and *Brit. Med. Journ.*, 1884, vol. ii. p. 263.
[7] *Liverpool Med.-Chir. Journ.*, July, 1891, p. 245.
[8] *Journ. Nerv. and Ment. Dis.*, 1884, N. S., vol. ix. p. 72.
[9] *Loc. cit.*, p. 566.

and others have adduced many facts in support of a neurotrophic theory, the most striking of which are as follows: (1) Bilateral symmetry in distribution of the lesions. (2) Associated trophic changes, notably in the skin and nails. (3) Disproportionate muscular atrophy. Ord dwells upon the resemblance of the latter to progressive muscular atrophy, and suggests that the primary cause may reside in the trophic centres of the spinal cord, or that it may be due to peripheral irritation from trauma, uterine or urethral lesions, etc. (4) Frequent pre-existing mental disturbances, such as shock, worry, grief. (5) The fact that certain cord lesions are productive of arthropathies (dystrophies), such as are observed in locomotor ataxia with the symptom known as Charcot's disease, syringomyelia. In two autopsies made by Falli[1] upon typical cases of arthritis deformans there was atrophy in the anterior horns of the cord, through which trophic impulses pass outward. In one of these cases degenerative changes were also present. Pitres and Vaillard[2] have described concurrent lesions of neuritis in several instances of arthritis deformans, and the centripetal progression of the disease from the periphery in its multiple type accords with the hypothesis of its nervous origin. Garrod[3] gives an excellent summary of this hypothesis as follows: "If in arthritis deformans we have merely a dystrophy of joints associated with dystrophic changes, primary and secondary in other structures, it may easily be supposed that such dystrophies will sometimes be local, limited to a single joint, which may or may not have been the seat of injury, may occur in very chronic forms in the course of senile decay, or under depressant conditions, or may assume a more generalized form, attacking many joints and adopting a more or less symmetrical distribution." Dor and others claim to have proved that the disease is of microbic origin, but their researches as yet lack confirmation.

The disease is unquestionably of great antiquity, for skeletons of prehistoric Egyptian and other remains have been discovered showing the permanent changes in the bones.

Sex and Age.—Arthritis deformans is most often seen in females, in the proportion of five to one, as shown in 500 cases collected by A. E. Garrod,[4] and the frequency with which it first develops increases from the twenty-fifth to the fifty-fifth year, when, after reaching a maximum, it rapidly subsides. It may, however, first appear at eighty years, and cases are sometimes observed in childhood. One of the worst cases I have seen was in a boy of nine years. Osler[5] reports 4 cases under twelve years. In over 300 cases treated at the Devonshire Hospital, a half dozen only began before the tenth year of age. It is noticed that in women the majority of cases begin during or after the menopause, and in other cases which began earlier exacerbations occur at this period (Garrod).

Heredity seems to be responsible for some cases, although its influence does not compare with that seen in gout. In a series of 500 priuate cases analyzed by A. E. Garrod,[6] 64 gave a positive family history of gout, 48 of rheumatism, and 84 of rheumatoid arthritis. Although the

[1] *Il Policlinico*, Dec., 1894.
[2] *Rev. de Méd.*, 1887, vol. vii. p. 456.
[3] *Loc. cit.*, p. 567.
[4] *Loc. cit.*, p. 528.
[5] *Practice of Medicine*, p. 283.
[6] *Twentieth Century Practice of Medicine*, vol. ii. p. 526.

proportion of gouty inheritance is great, he points out that this may be in part coincidence, as gout is so common in England. It is often noticed that several cases of rheumatoid arthritis occur in members of the same family. Another problem of interest, somewhat allied to the question of direct inheritance, is whether or not it be possible to inherit or develop an "arthritic diathesis" or a predisposition to affections of the joints, which may lead in one case to rheumatism, in another to gout, in a third to arthritis deformans. The possibility of such a condition has been under discussion for many years, and that it may exist as far as gout and rheumatism are concerned is the belief of some very competent observers (see Gout, p. 1000), but there is no definite ground for including arthritis deformans as a manifestation of a peculiar general arthritism.

Mental strain, especially anxiety, worry, care, prolonged grief, or undue excitement often antedates the symptoms. Garrod[1] found 34 such cases among 500 patients collected from private practice. It is quite certain that such influences, if not at first causative, tend to make the disease much more rapidly worse.

Other factors, such as social condition, exposure, bad hygiene, injuries, are of very doubtful influence. The disease is quite common among the affluent and among those who have suffered no exposure, although it is perhaps more so among the poor. Its onset is so insidious that it is the more difficult to determine the cause.

It is often associated with tuberculosis, or follows influenza, acute rheumatism, or gonorrhœal rheumatism, and is said to be more common among sterile women, or those having local disease of the generative organs (Ord); but here, again, as in the case of gout, there may be much in mere coincidence. No definite dietetic errors are known to be productive of arthritis deformans, but the disease is made worse by poor food and starvation. It is the opinion of Pye-Smith[2] and Garrod[3] that the very chronic arthritis deformans developing late in life is practically merely a senile atrophic change in the joints—a part of the general atrophy of old age. In conclusion, it must be frankly admitted that a very large number of cases furnish no assignable cause.

PATHOLOGICAL ANATOMY.—*The Cartilages.*—The lesions begin in the centres of the articulating cartilaginous surfaces of the joints, where friction is naturally greatest, and where the circulation is poor. There is proliferation of cells in the synovial membranes and peripheral parts of the cartilages, and the latter become fibrillated by disappearance of their cells, leaving only the matrix. The cartilages become velvety, soft, and are eroded and absorbed. Many cases are "dry," but effusion of synovia is also common, and if present it is an early phenomenon and may later disappear. Hoppe-Seyler[4] found an excess of mucin in the synovial fluid.

The Bones.—As the cartilaginous surfaces disappear, the opposing ends of the long bones become rounded, smooth, extremely hard, and shiny or "eburnated," resembling polished ivory. Similar absorption may take place in the extremities of the bones, but to a slighter degree. Sodium urate is not found as it is in gout. The proliferation of carti-

[1] *Loc. cit.*, p. 532.
[2] *Guy's Hosp. Reports*, xix. p. 311.
[3] *Loc. cit.*, p. 518.
[4] *Virchow's Archiv*, 1872, liv. p. 225.

lage cells proceeds undisturbed at the periphery, forming nodular masses, "chondrophytes," which in time ossify (osteophytes), producing greater or less immobility and deformity. Some joints, like the phalanges of the fingers, may retain slight motion, but others, especially

FIG. 92.

Arthritis deformans, showing great deformity and absolute rigidity of joints, which were immovable from the positions shown (a patient in Bellevue Hospital).

the knees and other large joints, may become firmly interlocked and immobile on account of the lipping of the rims of the articular bony surfaces.

As the disease progresses the periosteum near the joints may become

thickened and ossified, forming nodules here and there along the shafts. The latter quite exceptionally become hardened and thickened (Adams[1]). All the joint ligaments and the capsule become greatly thickened, adding to the deformity already produced by the increase in bone. Fig. 92 illustrates extremely well several pathological features, namely, the pseudo-ankylosis, thickening and deformity of the joints, and the wasting of the muscles. This patient, for some time under treatment in Bellevue Hospital, had suffered for many years from rheumatoid arthritis. So great was the rigidity of all the leg and pelvic articulations that he was like an image of wood. If the right leg were pressed down, the left would go up, and *vice versâ*.

On the eburnated surfaces of the bones are seen minute perforations, the orifices of Haversian canals, and the surfaces are often deeply grooved by attrition. This may even occur before all cartilaginous covering has been lost (Volkmann[2]). The hardening of the bones has received various explanations. It may be of inflammatory origin, but Cornil and Ranvier[3] believed it to be due to the discharge of cartilage capsules into medullary spaces, which alike compress the bone and favor its absorption and yield to abrasion. Morrant Baker held that the osteophytes represent a conservative effort toward repair. In advanced cases the entire relations of the joint may be altered. A surface previously convex may become concave by erosion, and one previously concave may become convex by heaping up of the rim. Fibrous adhesions and genuine bony ankylosis do not occur in any of the joints of the extremities, although the latter has been observed by Bowlby[4] in the vertebral column. Sometimes subluxation or dislocation is produced, especially in old persons, owing to extensive cartilage or bone absorption, as illustrated in the head of the femur (morbus coxæ senilis).

The synovial membranes become inflamed coincidentally with the changes in the cartilages. Their cells proliferate, their fringes become greatly thickened, and may either form cartilaginous nodules or undergo fatty degeneration, or very rarely ossify. The nodules are often pediculated, and may break loose into the joint cavity. Such nodules are common in the bursæ, and may often be felt over the olecranon.

The Muscles.—Aside from the above described lesions of the cartilages, bones, and synovial membranes, the only other fairly constant pathological change is found in the general muscular wasting. The skeletal muscles become brown, dry, and show varying degrees of fibre degeneration. The atrophy is no doubt in great part due to inability to exercise the muscles, but it is also due to the same trophic disorders which are believed to initiate the joint lesions, for it often occurs early and may be present in muscles which are still in use.

Clinical Varieties.—One cannot do better than follow Charcot's original division of the clinical types of the disease into the forms: (1) generalized or multiple progressive type; (2) localized or monoarticular type; (3) Heberden's nodosities.

The first variety invades especially the smaller joints, but may soon include all. The second variety affects only one or two of the larger

[1] *On Rheumatic Gout*, 1873. [2] *Billroth's Handl. der Chirurgie*, Bd. ii. p. 555.
[3] *Manuel d'Histologie Path.*, Ed. 1881, i. p. 465.
[4] *St. Bartholomew's Hosp. Reports*, 1890, xxvi. p. 77.

joints—*i. e.* is central, not peripheral; while the third occurs in the terminal articulations of the fingers. It will be found convenient also to separately describe the peculiarities of the disease in children.

Symptoms.—(1) *Multiple Type.*—This form of rheumatoid arthritis presents itself under two distinct clinical pictures—(*a*) the acute, and (*b*) the chronic.

(*a*) *Acute Form.*—This form of the disease is much less common than the chronic. It may affect young children, or women at the menopause, but is usually seen in young women during the childbearing period, and it is often associated with the functions of that period, such as parturition lactation, too rapid child-bearing, etc. The patient complains at the outset of pains in many joints, with swelling and tenderness over them. Redness is not marked, but in other respects the local appearance somewhat resembles acute rheumatism, but there is no migration of

Fig. 93.

Arthritis deformans, showing permanent deformity of the fingers, with deflection toward the ulnar side. The thumbs are normal (from a patient in the New York Hospital).

symptoms from joint to joint, and no implication of the heart. The temperature may rise to 101° or 102° F., but the constitutional symptoms are not striking. Malaise, headache, anorexia, dulness, and depression are complained of. Large and small joints are simultaneously involved, and there is serous effusion within the joints and bursæ. Some improvement may follow, but the symptoms recur and the disease progresses.

(*b*) *Chronic Form.*—The chronic form begins very slowly, at first involving one joint of the hand or foot or the knee, spreading gradually to the corresponding joint of the opposite side, and gradually involving a large number of joints, the tendency being to extend from the periphery centripetally. In over one half of all cases the fingers are first involved (Garrod). The pain is not great as a rule, but excep-

tionally it may become so intense as to require the use of morphine. It is often only felt on movement. The joints are uniformly swollen and give the signs of moderate effusion, but with little tenderness or redness. The disease tends to progress by stages, temporary delusive improvement may last for several weeks or months, or in some cases for years,

Fig. 94.

Arthritis deformans, showing permanent extension of the feet, deformity of the toes, and advanced muscular atrophy (from the same patient whose hands appear in Fig. 93).

but with each renewal of the symptoms the joints become a little worse and show less tendency to improve.

After some months or years, practically all the joints of the extremities are involved and show more or less deformity, thickening, rigidity, or distortion. The osteophytes and enlargements of the long bones present swellings which become the more prominent in connection with the wasting muscles.

The deformity of the hands and feet is pathognomonic. This is well illustrated in Figs. 92 and 93, and both photographs of these joints are of a patient who, several years ago, was in my service at the New York Hospital. The figures as a whole become flexed and bent toward the ulnar side, sometimes overlapping each other. The distal phalanges, however, are sometimes bent by osteophytes toward the radial side. The nails tend to dig into the palm, which may have to be protected with cotton. Extension of ten or fifteen degrees may be obtained, but the joints will then be felt to lock firmly, and further motion may be impossible. By manipulation the extension may be slightly increased beyond the patient's voluntary power, and this without causing much pain, but the fingers promptly return to their accustomed position. The thumbs curiously escape, and the patient is thus able to grasp various objects, and by slipping the handle of a knife or fork within the palm,

FIG. 95.

Arthritis deformans, showing permanent rigidity of the knee- and ankle-joints, flexion of the feet and fingers, and advanced muscular atrophy (a patient now in Bellevue Hospital. While in this condition he was successfully operated upon for a gangrenous appendicitis).

and holding it with the thumb, he may still be able clumsily to feed himself. While some finger-joints are firmly locked in pseudo-ankylosis, others are less so, and others, again, may be abnormally movable and capable of dislocation or subluxation. "Toggle" joints are thus formed, the backward and forward motion being nearly equal in extent. Sometimes a universal or ball-and-socket joint is formed. On moving the joints to and fro a coarse crepitus is often both felt and heard. While these changes in an advanced case are usually very pronounced in the hands, this is not always so, and they may almost escape while other joints fare much worse. A woman of twenty-eight years of age, who has been under my care, was bedridden for several years, all the larger limb-joints being bent, rigid, distorted, and drawn up toward the body,

while the fingers were free. In such cases, of which I have seen several, the legs are bent in close to the thigh, the thighs are semi-flexed, and one knee is usually more or less crossed over upon its fellow, which is everted. The feet are in ordinary cases in a position of complete flexion (Fig. 94), and there is usually much more absolute rigidity in the ankle- and toe-joints than in those of the wrist and fingers. The toes are everted from the tibia (in opposition to the finger deformity), and are apt to overlap each other. The great toe, in contradistinction to the thumb, is often rigidly fixed and much distorted. In other cases, instead of flexion of the legs and thighs, they are extended and rigid, as shown in Fig. 95. This is a photograph of a patient who is at the present in my wards in Bellevue Hospital, where he has been under treatment for between two and three years. The legs, feet, toes, fingers, hands, and wrists are rigidly fixed in the positions shown in the photograph, but the shoulder-joints, elbows, and spine are little if at all affected. The marked atrophy of the muscles of the legs, thighs, and forearms, which is such a typical symptom of the disease, is also well illustrated. The case is of special interest because the patient, in spite of so great deformity, is still able to do much. He can feed himself, and even hold a pencil and write an intelligible scrawl. When propped up in a wheeling-chair he manages to propel himself about the ward. Five months ago he had a sudden attack of appendicitis, and my colleague, George Woolsey, removed a gangrenous appendix with several ounces of pus. I feared the effect of this complication, as well as that of administering ether to a man in his condition, but no untoward result followed, and he made a brilliant recovery from the operation. While he was anæsthetized I had an excellent opportunity to examine the joints, but was unable to make any impression upon their rigidity by any justifiable degree of force. The knees, ankles, and toes were as inflexible as if joints had never existed in them. The man is very intelligent, and being wholly free from pain, is remarkably contented and cheerful, being still hopeful of improvement which will never come.

In some cases there is alternate flexion and extension of joints, especially of the wrist and phalangeal articulations (see Fig. 92, p. 980); but even with such deformities there is still a fair degree of symmetry in the involvement of the opposite sides of the body. In long-standing cases nearly every joint in the body is affected, including the vertebral and the temporo-maxillary articulations. In the latter case the patient must be fed with fluid food through spaces between the teeth. The distortion of the joints is explained by the asymmetrical growth of the osteophytes, the ligamentous thickening, and irregular wasting and contraction of the muscles. Dyspepsia and anæmia of mild grade are often present, but are seldom worse than might develop in any bedridden patient. The heart, lungs, and even kidneys are not subject to lesions. There is no fever except in very acute exacerbations, but the pulse is usually quickened, often 90–100, or 105.

As early symptoms of arthritis deformans dysæsthesiæ are sometimes encountered, such as formication and numbness of the extremities. Some patients, fortunately the fewer number, suffer intense exacerbations of pain and sometimes of muscular cramps, especially at night

and whenever the disease is actively advancing. As a rule, the pain is less in the older cases. Trophic disturbances of the skin are common. It becomes dry, smooth, and glossy, especially over the swollen joints, and is sometimes irregularly pigmented or "freckled" (Spender[1]) there. Anders[2] reports three personal cases of onychia, a condition also observed by others. He also refers to free perspiration in advanced cases, which I have not seen. Bedsores are less common than would be supposed with so much emaciation and tendency to trophic disorders.

Subcutaneous nodules like those seen in the rheumatism of children have been occasionally reported.

Gowers[3] finds that the trophic muscular wasting, which as a symptom is second only to the joint lesions, is often accompanied by increased myotatic irritability. The atrophy affects the extensors in greater degree than the flexors, and hence flexion is more common than extension, but extension may easily be produced by osteophytes. Other clinicians find that the reflexes vary, and in so far as they can be tested in rigid limbs may be either much increased or diminished. Clonic spasm in extension of a limb has been observed by Garrod.

As a great rarity may be mentioned deafness from implication of the ossicles of the tympanic cavity in the arthritic process.

(2) *The Localized or Monoarticular Type.*—This partial form is mainly found in patients past fifty-five or sixty years of age, and is usually confined to one or two of the largest joints, such as that of one hip (morbus coxæ senilis), shoulder, knee, or those of several vertebræ. The pathological anatomy differs in no wise from that of the general or multiple type, which makes it the more difficult to account for the peculiar limitation to a single large joint. Clinically, there are other differences, for this type of the disease is much more often observed in men and in those who have sustained previous injury to the joint.

When the hip- or shoulder-joint is affected, dislocation with shortening of an inch or two is common from the relatively great absorption of the articular surfaces of the bones, and dislocation is aided by local atrophy of the muscles (glutei, deltoid, etc.). The vertebræ may be involved in any portion of the spinal column, and they become locked so as to prevent rotation and lateral flexion. This condition is called spondylitis deformans. More than one vertebral articulation is usually involved in this subvariety, and rotation of the head may be prevented. Exceptionally the whole vertebral column becomes immobile.

Heberden's nodes (see page 987) may be present in the monoarticular type, and sometimes the corresponding joint of the opposite side is involved. Morrant Baker[4] has described the occurrence of large cysts about the diseased and also other joints, which contain synovial exudate and sometimes herniæ of the membrane.

Whichever joint is affected is stiff and sore. Movement excites pain, or severe pain may occur independently. Audible crackling, followed by grating crepitus, is obtained. Osteophytes may be felt, or the joint appears either immobile or dislocation can be produced on handling.

[1] *Brit. Med. Journ.*, 1891, May 30.
[2] *Practice of Medicine*, p. 390.
[3] *Diseases of the Nervous System*, i. p. 381.
[4] *St. Bartholomew's Hosp. Reports*, 1835, xxi. p. 177.

Garrod[1] describes a rare variety of this type in which the carpo-metacarpal joints of both thumbs have been the only ones involved. He attributes his cases to strain from occupations involving the excessive use of the thumbs.

(3) *Heberden's nodes* were described by him in 1805[2] as occurring in the multiple type of the disease, especially in middle-aged women, and being limited to the sides of extensor surfaces of the distal interphalangeal joints. When first developing there may be tenderness with swelling and some redness, and slight injury accentuates these signs, but later they give rise to little inconvenience, except from deformity and interference with manual dexterity. Irregular acute attacks may recur, being excited by local injury or apparently sometimes by indiscretions in diet. After some time cartilage destruction and eburnation of the bones result, and the nodosities remain incurable, but the process seldom extends to the larger joints, although the monoarticular form of rheumatoid arthritis is occasionally associated. Typical cases present two symmetrical immovable osteophytes, one upon either side of the joint, but the enlargement may surround the joint and appear fusiform. Cystic enlargements or herniæ of the capsules may be present, filled with clear fluid. Radial deflexion of the joints may occur in opposition to the ulnar deflexion which is typical of involvement of the entire fingers and hand. As in gout the thumbs usually escape. The nodosities are not associated with urate deposit, and they bear no relation to gout or goutiness, although such has been maintained.

Arthritis deformans in children is not common, but is more often met with than tophaceous gout. In them it seems to be more dependent upon bad hygiene than in adults. Poor and insufficient food and foul air or exposure to cold and damp are apt to develop it. It runs a rather more acute course as compared with the disease in adults, the sexes are more equally represented, and in some cases it has been associated with goitre, presenting a rapid pulse, tremors, and skin discoloration (Spender,[3] Diamantberger[4]).

DIAGNOSIS.—*Monoarticular arthritis* of the shoulder, with neuritis and local muscular atrophy, must be differentiated from the localized type of arthritis deformans when it happens to affect this joint. Anders[5] reports 5 cases of this kind, and Osler refers to others. The morbid anatomy is wholly different in the two diseases, for in the first-named the bones and cartilages are not involved but only the capsule and ligaments, and, moreover, the patients recover after a subacute and painful attack.

Sciatica.—Morbus coxæ senilis is differentiated from sciatica by the absence of tenderness, though pain be present along the sciatic nerve, the limited rotation of the diseased hip, the increase in pain produced by crowding the femur against the joint, and the difficulty or impossibility of crossing the diseased leg over the sound one while in a sitting posture (Garrod). There is also flattening of the glutei, and the knee jerk is increased on the diseased side.

Progressive muscular atrophy can be promptly diagnosed from the

[1] *Loc. cit.*, p. 558.
[2] Heberden: *Commentaries*, 1805.
[3] *Loc cit.*
[4] "Du Rhumatisme noneux chez les Enfants," *Thèse de Paris*, 1891.
[5] *Practice of Medicine*, 1897, p. 391.

atrophy of muscles in arthritis deformans by the entire absence of joint symptoms in the former.

Charcot's disease, which is the name given to that form of locomotor ataxia which presents destructive joint lesions somewhat resembling those of rheumatoid arthritis, is distinguished from the latter by a tendency to greater and more rapid destruction of bone, with usually less prominent osteophytes, although in both diseases there is loss of cartilage and hypertrophy of synovial membrane.[1]

Hæmophilia occasionally presents joint lesions analogous to those of arthritis deformans, which, however, are often accompanied by effusions of blood into the joints and fibrous adhesions (Bowlby)[2], which are absent in the latter disease.

TABLE OF DIAGNOSIS BETWEEN

	Arthritis deformans.	Chronic gout.	Chronic rheumatism.	Acute rheumatism.
Heredity.	Slight.	Over 50 per cent.	Slight.	Slight.
Course.	Slowly progressive.	Periodic, frequent exacerbations.	Variable, often considerable improvement.	Often complete cure.
Origin.	Usually in many small joints—fingers or toes.	Usually in one great toe.	Usually in a few larger joints.	Do.
Progress.	Centripetal.	Do.	Irregular.	Often first involves small joints only in later attacks.
Migration from joint to joint.	None.	Do.	Irregular.	Decided tendency.
Typical visceral lesions.	None.	Arterio-sclerosis, granular contracted kidney.	Cardiac valvular disease.	Endo- and pericarditis.
Pain.	Usually moderate or none.	Agonizing, localized	Varies, not very intense.	Acute, severe, and often migrating.
Signs of acute inflammation.	Not marked.	Intense at first and in exacerbations.	Insidious.	Most intense.
. . . . .	Spinal and temporo-maxillary joints sometimes involved.	Never.	Very rarely.	Never.
Permanent deformity due to—	Osteophytes.	Tophi.	Fibrous ligamentous thickening.	None.
Fixation.	Greatest.	Intermediate.	Least.	None.
Dislocation and crepitation.	Common.	Rare.	None.	None.
Enlargement of ends of bones.	Moderate.	None.	None.	None.
Sodium biurate in joints.	None.	Excessive.	None.	None.
Symmetry of lesions.	Remarkable and characteristic.	Usually none.	Irregular.	Irregular.
Nodules.	Heberden's nodes.	Tophi.	Irregular nodes.	Irregular cutaneous nodules.
Tenderness.	Slight, if any.	Greatest at sides of joint.	Greatest in front and back of joint.	Excessive and diffuse.
Salicylates.	No effect.	Little if any effect.	Slight effect.	Marked effect.
Colchicum.	No effect.	Marked effect.	No effect.	No effect.
Etiology.	A tropho-neurosis of unknown origin.	Uric acid, often dietetic.	Unknown.	Microbic (?).
Predominant sex	Females 5 : 1.	Males, decidedly.	Alike.	Males slightly.
Commonest age.	Progresses 25th-55th year.	35th to 55th years.	40th to 80th years.	15th to 25th years.
Social position.	Chiefly poorer class.	Chiefly richer class.	Little influence.	Do.
. . . . .	. . . . .	Excess of uric acid in the blood; tophi in the ears.	. . . . .	High temperature and sweating.

Syringomyelia may sometimes present joint lesions similar to those of arthritis deformans, which may endure for ten years (Beevor and

[1] I have just presented a case of Charcot's disease at my clinic in which the femur and tibia were more enlarged than I have ever seen them in rheumatoid arthritis.

[2] *St. Bartholomew's Hosp. Reports*, 1890, vol. xxvi. p. 77.

Lunn),[1] but other symptoms of spinal cord disease are always present to establish diagnosis.

Gout and Rheumatism.—It is highly important to discriminate between rheumatoid arthritis and chronic gout and rheumatism, especially as the treatment for these several diseases, as well as the prognosis, is radically different. To facilitate this study I have arranged the preceding table. To its individual statements there are occasional exceptions, as such a table can naturally only include average or typical cases. It is, however, the irregular or obscure cases which are deceiving, but in these usually some half dozen symptoms can be selected from the table by which a positive diagnosis can be made.

As a sequel to acute rheumatism, fusiform enlargement of the joints, called by Jaccoud "*rheumatisme fibreux*," may remain for months and bear some clinical resemblance to arthritis deformans, especially as there may be accompanying muscular atrophy. This condition is especially apt to occur in the knees, and I have seen two such cases, in both of which a pseudo-ankylosis prevented walking for upward of two years, but complete recovery with full return of muscular power followed treatment. The condition is not always recovered from, but the subsequent course of the disease is wholly different from rheumatoid arthritis, and the pathological changes involve neither bones nor cartilages.

Prognosis.—The prognosis for ultimate cure is hopeless, but for relief or arrest in early cases it is very good. Even such advanced cases as those represented in the illustrations (Figs. 93, 94, 95, pp. 982–984) accompanying this article, derived some benefit from treatment, and patients may live in such condition in fair general health for many years if properly cared for. This is no doubt owing to the striking lack of any tendency in this disease to involve viscera or bloodvessels, such as characterizes rheumatism (endo- and pericarditis) and gout (arteriosclerosis, cirrhoses of kidney and other organs).

The natural course of the disease in its multiple type, if uninterrupted by treatment, tends to a slow progression from the smaller to the larger joints, and renders the patient more and more dependent, helpless, and finally bedridden. The young are more amenable to treatment than the aged, but in the latter the disease advances more slowly and with less permanent crippling. The disease may last twenty years or more, or the patient may die from some intercurrent affection. Anæmic patients do poorly. Patients who present the greatest deformities of the lower limbs may have the least in the fingers, and are able to do much for themselves within the limitations of lying in bed.

Treatment.—A patient presenting the symptoms of arthritis deformans must be treated as an invalid with lowered vitality, and no graver mistake can be made than by treating him by depletion, as if he had gout. Aside from occasional simple local measures, the treatment should be almost wholly hygienic and dietetic. The patient should remove from a damp locality or ill-ventilated apartment, and seek abundant fresh air, sunshine, good food, and cheerful surroundings, avoiding as far as possible all sources of anxiety and worry. He should dress warmly, wearing woollen next the skin and sleeping in flannels in winter.

A reasonable variety in diet may be allowed, so that it is ample and

[1] *Clin. Soc. Trans.*, London, 1894, vol. xxvii. p. 209.

nourishing. Cereals, with fresh vegetables and fruits, and especially abundant animal food, should be eaten. Plenty of good milk, cream, butter, beef, mutton, poultry, eggs, and fish must make the basis of the *menu*. The use of malt liquors—ale, stout, porter, malt extracts—with meals is also to be recommended. In many cases, if the appetite fails from lack of ability to exercise and be in the open air, it is desirable to supplement the three regular meals of the day by offering food between them, such as milk, egg-nogg, or broth with biscuits.

Internal medication is of little avail in any case of long standing. Many clinicians prescribe potassium iodide (gr. x., *t. i. d.* well diluted in milk or Vichy), and Garrod prescribes free iodine (♏j) in sherry. I have given piperazine faithful trial, but have seen no benefit from it. The syrup of the iodide of iron is a useful tonic for children (♏v, *t. i. d.* diluted in water).

Cod-liver oil is the best tonic for arthritis deformans, and if anæmia is decided iron and arsenic may be advantageously prescribed. The remedies for gout and rheumatism—colchicum, salicylates, alkalies—are worse than useless, and the less medicine given the better. Opium also should be avoided on account of the exceeding chronicity of the disease, and effort should be made to relieve pain by simple anodynes or local applications.

Muscular exercise must be limited by the onset of either pain or fatigue. Muscular cramps may be relieved by use of tincture of hyoscyamus (Garrod), and muscular atony may be somewhat counteracted by gentle friction and massage. Electricity is of value only in so far as it exercises the weakened muscles by causing their contraction.

Local Treatment.—If the swelling of the joints is acute and painful, relief may be obtained from belladonna liniment or from application of glycerine and guaiacol in equal parts, combined with rest and support upon splints, the affected joints being wrapped in cotton and oilsilk.

The stiffness of chronic cases is to be relieved more by hydrotherapy with judiciously applied massage or Swedish movements than in any other way, but all such treatment must be mild at first or it will make matters worse, and the patient should understand that it must be continued for fully a month or six weeks before very decided results are obtainable. Gentle friction with oil or lanolin tends to promote the absorption of effusions and increase motility.

I have found the daily soaking of the joints for ten minutes in water as hot as can be borne, accompanied by their massage, to often produce very considerable improvement in reduction of ligamentous swelling and fluid accumulation, though of course exostoses cannot be influenced by such treatment. Patients who are fairly strong derive improvement from the Scotch douche, alternate hot and cold water douching under considerable pressure. I have recently made successful trial of the new local hot-air apparatus in several cases. The joints are enclosed in an adjustable box, made to fit about any variety of joint, and air heated by a lamp to 180° or 200° F. is passed around them for three quarters of an hour. If a cloth be laid over the joints, it becomes moistened with the intense local perspiration excited and acts very much as a poultice, so that for an hour or two after removal of the apparatus the skin looks almost parboiled, but it soon regains its natural appearance.

Hot local vapor baths have been employed, and Haygarth, Strümpell, and others extol the use of hot sand. General hot-water or vapor baths are too debilitating for advanced cases, and their use should be condemned. As regards mineral baths, more usually depends upon care in the mode of giving them than upon the quality of the waters themselves, although the sulphur waters appear to be the best.

Such systems of bathing, douching, and massage as are practised at Aix-les-Bains in Savoy with hot sulphurous water, or at Aix-la-Chapelle, or in this country at Glenwood hot sulphur springs in Colorado, and the hot sulphur springs of Virginia, often give great relief to those whose means enable them to travel, and a "course" at such institutions should be taken at least once a year, but only in the early stages of the disease, or more harm than good follows.

The progress of the disease is often stayed by winter residence in a warm climate, such as that of Southern California near Los Angeles, or in Cairo or Tangiers. A mere change of scene and climate is often beneficial by improving the general health and occupying the mind with new interests, and keeping the patient cheerful.

GOUT.

By W. GILMAN THOMPSON, M. D.

Synonyms.—Podagra; Fr. Goutte; Ger. Gicht; Ital. Gotta.

Definitions.—Gout is a disease characterized by recurrent arthritis, gradual deposition of sodium biurate in the joints of the extremities and their vicinity as well as elsewhere in the body, and by more or less severe and irregular constitutional symptoms.

Clinically, it is convenient to recognize acute and chronic forms, while the irregular manifestations of gout, both hereditary and acquired, are classed under the subheading Goutiness (Ewart), or the gouty diathesis (page 1020).

Legitimate gout is a term used to describe the more active or typical form of the disease with prominent localization of uratic deposits in the joints, which provokes an intense inflammatory reaction in the surrounding tissues, with constitutional disturbance exhibited by the nervous, circulatory, digestive, and excretory systems.

Irregular gout is a term applied to various indefinite manifestations of gout which may be either hereditary or acquired; such, for example, may be attacks of asthma, peripheral neuralgias, headache, and eczema, or other cutaneous lesions. These and similar phenomena will be considered under Goutiness, page 1022.

Latent gout, *hidden gout*, *suppressed gout*, are terms used to express the existence of conditions similar to those of irregular gout, but frequently less pronounced. Such are often manifested by gastric or cardiac crises, modifications in the urinary secretion, etc.

As insisted upon by Bouchard, it should be emphasized that gout in any form is really a chronic malady—a *very* chronic malady—often insidiously operating from infancy if hereditary, and hence the form clinically known as acute is merely an epiphenomenon—a climax reached in the development of conditions which have perhaps been in operation for decades.

Ewart[1] defines gout as primarily a perversion of nutrition capable of producing eventually structural changes; "in a word, it is a functional derangement fraught with organic consequences."

History.—For many centuries gout has been well known, and even in general literature it has filled an important rôle in furnishing moralists and satirists with lessons and gibes aimed at the affluent or those whose habits of ease and luxury, or lack of self-control in resisting the good things of the table, have always, in the public mind at least, been regarded as essential predisposing factors.

Arétæus of Cappadocia in the first century of the Christian era was

[1] *Gout and Goutiness*, London, 1896, p. 299.

the first to differentiate gout of the foot (podagra) from general articular inflammation. Cœlius Aurelianus in the sixth century wrote a more accurate description of the malady, which was then much studied by the physicians of the Alexandrian School. In 1560, Baillou of Paris published the first treatise discriminating between gout and rheumatism, and this was followed a little more than a century later (1683) by the famous classical description of the immortal Sydenham,[1] in which he maintained that the local or articular manifestations of gout originated in an effort of Nature to eliminate the morbific products of a general or constitutional nutritional disorder. In 1795, Tennant and Pearson derived uric acid from gouty concretions, and two years later Fourcroy and Wollaston pronounced these deposits urate of sodium. In more recent years the pathogenesis of gout has been elaborately investigated from all standpoints, from the chemical, clinical, and theoretical sides as well as from that of pathological anatomy, and the names of many noted authors have become still more famous from their researches in this important field. In Germany Virchow, Senator, Ebstein, and Pfeiffer, and in France Charcot, Rendu, and Bouchard, among many others, have been prominent contributors to the literature of gout. It is, however, England which seems more directly than any other European country to have followed early Greece and imperial Rome in fostering the malady which many an emperor or king would have given half his sovereignty to be freed from, and it is in England that by far the most important additions to the knowledge of the disease have been made during this century by such men as Scudamore, Prout, Graves, Todd, Watson, Sir Alfred B. Garrod (1848), Sir Dyce Duckworth, Sir William Gull, and, quite recently, by Alexander Haig, Sir Willoughby Wade, and William Ewart. It is a curious fact that many among the notable authorities upon gout have been sufferers from it themselves for years, and hence have given singularly graphic clinical descriptions.

Etiology of Gout in General.—Fundamentally, gout is the result of a disturbed metabolism, consisting, probably, in defective oxidation of proteids, and combined with faulty elimination of their waste products. The theories of the underlying conditions which produce this faulty proteid metabolism will be better appreciated after reference to the

Predisposing causes.—*Heredity* is chief among predisposing causes. It is the rule rather than the exception for parents who are strongly gouty to transmit the gouty diathesis to their children. Especially is this true in the male line of descent, and of all cases of gout more than one half have been shown to be of hereditary origin. It should be observed, however, that in addition to the inheritance of the gouty vice an individual often inherits the affluence which enabled his father to acquire those habits of indolence and luxurious living which originally contributed to the development of the disease, and, if he adopts those same habits of life, he is the more apt to precipitate an acute attack. An interesting phenomenon of the heredity of gout is its variation in type in successive generations, "the arthritic being succeeded for one or even two generations by the cutaneous, the calculous, the nervous, or some other visceral form, after which arthritic gout may again occur" (Ewart).[2]

Age.—The disease has been quite rarely recognized even among the

[1] *A Treatise on Gout.*

[2] *Op. cit.*, p. 26.

newborn, and in those cases in which the heredity can be traced for several generations or is otherwise intensified the symptoms are sometimes apparent by the period of puberty. In general, however, the disease does not fairly develop until after thirty years of age, although in hereditary cases, latent or irregular symptoms—such, for instance, as a tendency to catarrhal attacks or neuralgias—may be recognized many years earlier. Trousseau [1] reported a case of gout and asthma in a child of five years. The period from thirty to fifty years is that in which the disease becomes acutely manifest for the first time in by far the larger proportion of cases, and it will be observed that this corresponds with the age at which a man accustomed to energetic outdoor exercise in his youth naturally passes from the stage of development and youthful activities into that of greater cares and responsibilities. Feeling still vigorous and ambitious, he gives less time to exercise and recreation and more to the pleasures of the table, which his increasing prosperity enhances. He continues to eat and drink more than he needs in his more sedentary life, and the activity of his excretory organs is no longer stimulated by vigorous exercise. It is now that the long latent or the hereditary gout first manifests itself by an unmistakable acute attack, and gives a warning which, if not met by a radical change in mode of life, will inevitably be repeated at no distant date.

The disease is also rare at the opposite extreme of life, unless, indeed, it has begun in middle life and become chronic. Fagge saw a case at seventy years, and Garrod one at eighty, and one at nearly ninety years of age.

Haig [2] suggests that one reason for the greater prevalence of gout in those past middle life is that the joints of such persons are less vascular and less alkaline than in early youth, and are more sensitive to exposure to cold.

Sex.—Typical gout is fully twenty times more prevalent among men than women, although "goutiness" is nearly equally represented in the two sexes. This difference is apparently greater than is to be accounted for by habits of life, of eating and drinking, etc. In the allied affections, rheumatism, rheumatoid arthritis, etc., the numerical difference between the sexes is less striking. Women often show the gouty diathesis or "goutiness" even when they may not have acute attacks of gout, and it is more often manifest in them than are the severe acute and chronic forms. Fagge [3] suggested that the periodical outlet afforded by the catamenia may have some bearing upon the relative infrequency of acute gout in women. It is certainly more prevalent among women after the menopause, as originally observed by Hippocrates.

Social Condition.—From the frequent references made to high living and gout the impression often prevails that this causative relation is always present. It is quite true that the majority of cases of gout occur among those whose means enable them to live luxuriously, eat richly-cooked food, including abundant meats, and drink rich wines, while at the same time they take too little exercise and live an indolent indoor life. It is equally true that there exists the "poor man's gout," found

[1] *Clinical Medicine*, vol. ii. p. 317.
[2] *Proceed. Royal Med. and Chir. Soc.*, April, 1890, p. 109.
[3] *Principles and Practice of Medicine*, vol. ii.

among hard-worked, ill-fed laboring men, chiefly past middle life, who also drink much heavy malt liquor. In Bellevue Hospital, where patients of this class predominate, I see such cases every year.

Geographical Distribution.—It is doubtful whether geographical distribution, *per se*, has much to do with the development of gout aside from furnishing favorable conditions for luxurious living. The European, prone to gout, who goes to live among natives of any other clime, who never have it, if he adopts the habits of a gourmet will develop an attack as promptly as if he were at home. It is therefore owing mainly to different social customs and local habits of eating and drinking depending upon the natural products of a country, that variation in the distribution of the disease is due. Poverty, with temperance, and outdoor life do not beget the disease: poverty, as well as affluence, with intemperance in both eating and drinking, and indoor life do beget it. It is therefore most common in luxurious aristocratic capitals and social centres, while it is almost unknown on the plains, in mountainous regions, or farming countries.

Formerly, and again owing rather to habits of life, during the Empire, gout was very prevalent in Greece and Italy. To-day its chief centres are in England and Holland. In France it is commonest in Brittany and Normandy and in the champagne and rich wine-growing provinces of Lorraine and Burgundy. It is far less common in Germany and Russia, and in Scandinavia it is rare (Fagge[1]) except in a few large cities.

In the early history of the United States, in days which involved much hardship and exposure, when the simplest habits of life were a necessity, gout was seldom encountered, except as an hereditary disease imported from Europe. To-day two classes of American residents exhibit it: first, foreigners among the poorer population who crowd the larger cities of the Atlantic States, and, second, the well-to-do natives of larger cities throughout the country, who, as the country grows richer, are in greater danger of courting gout by their habits of life. It is doubtful, however, whether the disease will ever become nearly so prevalent in the United States as it has been for centuries in England, and the timely revival of athletic sports, especially golf and wheeling, will do much to prevent it. Certainly at the present time the gouty diathesis, or "goutiness," is much more prevalent in the United States than chronic gout, and far more so than typical severe acute gout, such as Sydenham[2] described, which is a comparatively rare affection here. H. C. Wood[3] of Philadelphia, in referring to this subject, says that in all his experience he recalls but two cases of "Sydenham's gout."

Climate and Season.—The same observations may be made in regard to climate which have already been applied to geographical distribution. The disease is undoubtedly most prevalent in the mid-temperate zone and in climates where a considerable portion of the year is spent indoors, and where the inhabitants seem to feel the need of frequent alcoholic stimulation. Gout is more prevalent, or rather acute exacerbations are more prevalent, in the autumn and early spring than in seasons which

[1] *Text-book of Medicine*, vol. ii. p. 804.

[2] *Works of Thomas Sydenham, M. D.*, London, printed for the Sydenham Society, 1850, vol. ii.

[3] *Journ. Amer. Med. Assoc.*, 1897.

are less changeable or more tempting to outdoor life and exercise. A first attack will often occur in the autumn or winter, and later ones in the spring. In general, climate and season have less effect upon gout than upon rheumatism, and chronic gout seems usually quite independent of season, but its acute exacerbations may be sometimes forestalled by a winter residence in a warm climate, like that of Egypt or Algeria.

Toxic Agents.—Many poisons have acquired the reputation of causing gout, some by direct irritation, others by the insoluble compounds which they are believed to form with uric acid, or by causing uric acid to accumulate in the tissues, perhaps by modifying the normal degree of alkalinity of the blood. Among these various substances lead ranks as of first importance, and Haig[1] mentions opium, cocaine, antipyrine, strychnine, iodides, nitrites, some sulphates, hypophosphites and chlorides, lithia, mercury, acids, etc. Chronic lead-poisoning is often associated with gout, more particularly, however, with the gouty diathesis. Garrod and Haig found that when pills of lead acetate were administered to the gouty, less uric acid was eliminated by the kidneys than before, and existing gouty conditions became worse. It is, however, doubtful whether lead-poisoning can ever excite gout in an individual who neither inherits the diathesis nor who is in process of acquiring it; for the majority of lead-poisoning cases are never gouty. I have seen many among the worst of them without gout. The metal can produce a granular contracted kidney undistinguishable from that of gout, and it also causes advanced degrees of arterio-sclerosis, both being factors strongly tending to enhance existing gouty tendencies; but beyond this, as a primary cause of gout, lead has no true claim.

Nobécourt[2] has made a critical study of saturnine gout, and finds it most common among housepainters, who are very slowly poisoned. More rapid lead intoxication from working in white lead is apt to develop symptoms of lead palsy or colic early, and the workmen give up before they have become gouty.

(Further reference to this matter will be found in the article by Finley upon Chronic Lead-poisoning, Vol. III. p. 722.)

Other Diseases.—It is possible that the frequent association of gout with particular diseases, notably diabetes mellitus, may be due to the development of toxic agents in the blood.

Gout is often associated with tuberculosis, and especially the condition known as scrofula, and it may occur in connection with rheumatism, although there is no definite etiological connection between the two diseases. It may develop in those having granular nephritis, but the latter is more often a complication or sequel of the former. The same is true of asthma. Ewart[3] writes: "In a man confined to his bed with hemiplegia, the leg recovering quickly and the arm remaining paralyzed, gout may develop in the paralyzed hand." Fully 30 per cent. of cases of cholelithiasis occur among gouty victims (Bouchard.)

Exposure to cold, fatigue, and other debilitating conditions predisposes somewhat to the development of gout. Roberts lays great stress upon the influence of cold. Cold acting locally upon the toe or other

[1] *Uric Acid in Causation of Disease*, 2d ed., pp. 23, 24. [2] *Edin. Med. Journ.*, 1897.
[3] *Op. cit.*, p. 156.

joints is regarded by some authors as a determining factor in localization of the inflammation in them, and Garrod says that persons having cold ears, from feeble circulation, are most apt to have tophi in them.

Injury to a joint may focus the inflammation in it. The common explanation of the greater prevalence of gout in the big toe is that it not only is at a remote part of the circulation, but in walking is subjected to constant shock and the strain of bearing the weight of the body. This view is controverted by Wade (see page 1002).

General Condition.—It cannot be positively said that gout occurs exclusively in any particular class of persons as regards their general condition, for it is recognized in the underfed as well as the overfed, in those of nervous as well as those of phlegmatic temperament, in the scrofulous or in the full-blooded or the anæmic.

Diet.—Whatever views are held in regard to the influence of dietetic errors in originally causing gout, it is everywhere admitted that, once established, the disease is maintained and fostered both by gluttony and by eating special articles of food, especially sugars. It is, moreover, a fact that if some forms of alcoholic beverages are taken with these articles of food, the effect of both is much enhanced. Such a combination as a sweet pasty and a glass of rich port or madeira may nearly cost the gouty victim his life. Sweet and effervescing wines, champagne, etc., in combination with saccharine food, cause fermentation and hyperacidity in the stomach. It is also not to be overlooked that under some conditions starvation is equally capable with gluttony of precipitating an attack of gout in one who is subject to the malady. In summary, no one article of food or drink may cause gout, and the worst dietetic habits for the gouty are overeating and overdrinking in general—*i. e.* gluttony—and in particular the eating of too much meat and the combination of sweets with certain other foods, notably sweet or acid fruits and wines. Sir Dyce Duckworth holds the combination of sugar with vegetable acids especially at fault. Sugar under some conditions of gastric and intestinal fermentation forms lactic acid, which in turn may split to yield carbon dioxide, which, according to Ralfe, forms acid salts of sodium and potassium from their neutral compounds, thereby reducing the alkalinity of the blood and synovia.

Sir Dyce Duckworth believes that there is "present in the gouty a peculiar incapacity for normal elaboration within the whole body, not merely in the liver or in one or two organs of food, whereby uric acid is found at times in excess or is incapable of being duly transformed into more soluble and less noxious products;" and he agrees with Ralfe that the failure to complete the uric acid metabolism is due primarily to deficient or disturbed innervation. According to such doctrines, excessive consumption of meat must be a strong factor in the production of gout, and this idea corresponds with the distribution of the disease mainly among civilized races whose diet consists very largely of animal food, whereas it is comparatively unknown among vegetarian savages like the South African plantain-eating negroes. On the other hand, an exclusive meat diet is not alone causative, for the most northern Eskimos, who possess no foods but meats and fats, and who have nothing to make alcohol out of, are not subject to gout. Moreover, gout may develop in persons who have been most abstemious all their lives in both eating and

drinking; but such cases are usually hereditary. It is, therefore, not meat alone, but meat taken in excess, with either malt liquors or sweet wines, champagne, etc., which may be responsible for exciting gout in those who are susceptible to it.

With regard to the habit of overeating, Sir H. Thompson says that in early life it may give rise to occasional attacks of biliousness, but after the first half of life has been spent the remaining half may be affected in a different way, and "recurring attacks of gout perform the same duty or nearly so at this period of life that bilious attacks accomplished in youth."

In regard to the primary influence of erroneous diet, Ewart[1] holds that gout is not due so much to faulty alimentation—not to meat-poisoning—but to faulty nutrition or behavior of the tissues toward the nutritive fluids of the body, aided by faulty innervation.

Alcohol exerts one of the strongest influences in the production of gout, but it seems to be certain that forms of alcoholic drinks, notably those containing sugars and fermentation products, are the most injurious. Among the laboring classes in England, who drink porter, stout, and heavy ales, gout is often seen, whereas it is rare among whiskey-drinking Scotchmen. It is not believed that alcohol alone in any form is apt to produce gout, although Garrod believed that pale bitter ale was capable of so doing. The heavy sweet wines, port, madeira, sherry, etc., have an old-established reputation for causing the disease. So also have sweet champagnes and rich burgundies. Sweet fermenting cider is similarly injurious.

PATHOGENESIS.—Of the many theories of the pathogenesis of gout, three are deserving of special notice. These are—

1. The uric-acid theory.
2. The nervous theory.
3. The theory of special nutritive-tissue disturbance.

Of these, the older uric-acid theory of Garrod has had the most adherents in the past, while the nervous theory has lately gained much ground, and the possibility must be admitted that in some cases all the factors under discussion may be combined. All are agreed upon the final presence of uric acid in excess, the resultant deposition of biurates, and the irritant effect upon the joints and tissues which these substances exert. The majority of authorities concur in the belief that this irritation is mechanical rather than toxic in character, but both Haig and Ebstein regard the circulation of urates in solution as distinctly toxic. Be this as it may, all connective tissues, especially the fibrous and cartilaginous tissues of the joints, are particularly prone to urate infiltration. The lymph and synovia most readily lose alkalescence and favor the urate deposit, and next in frequency this applies to the subcutaneous connective tissue, where tophi may accumulate. The difficulty lies in accounting for that original peculiar disturbance of normal proteid metabolism which underlies the clinical and anatomical phenomena.

1. *The Uric-acid Theory.*—Garrod found a decided increase of uric acid in the blood of the gouty, as well as a considerable lessening of alkalinity. This combined condition causes the precipitation of urates in those parts of the body where the circulation is least active. The

[1] *Op. cit.*, p. 37.

lessened alkalinity may be brought about by various conditions—in the case of overeating of animal foods by indigestion, and consequent formation of lactic and fatty acids in excess, which, on being absorbed, influence the blood in the manner described. The process is often further aided by defective elimination through the kidneys. The condition of the blood accounts for the constitutional symptoms, and the sudden local precipitation of biurates is so irritating to the tissues as to cause an acute inflammation. In brief, this theory comprises an accumulation of proteid waste in the blood, which may be due to (1) increased consumption, (2) imperfect assimilation, (3) imperfect elimination. Probably either one of these factors may originate the condition of gout, but in any protracted case they sooner or later all take part.

Latham[1] has followed Murchison in assigning chief importance to failure of hepatic function as a cause of increased uric acid in the body, followed by irritability of the nervous system. Chemically speaking, he ascribes the trouble to the non-conversion of glycosine to urea.

Pfeiffer believes the acute gouty attack to be due to a resolution of uratic deposits previously formed, and in support of his view points out that the pain is increased by alkalies and lessened after acids, such as salicylic, but this view has been strongly controverted by both Roberts and Ebstein.

Haig originally argued that diminished oxidation of the blood from any cause gives rise to an excess of uric acid in the system; this excess is accompanied by lessened activity of the capillary circulation, increased vascular tension, with consequent impaired metabolism, and increased formation of urea, acids, and the acid salts allied to urea. His theory is therefore that of the action of uric acid in solution as a toxin, and he argues in favor of the overproduction of uric acid in distinction from Garrod, who found retention more important than overproduction, except perhaps in the "poor man's gout" of inanition.

No excess of uric acid is necessary, as the diminished alkalinity of the blood is sufficient to cause precipitation of biurates.

In summary of his conclusions Haig[2] writes: "In place of rheumatism and gout, I see but one disease, an arthritis, due to the presence of urates which under some circumstances will be limited to one joint (gout) or affect several joints contemporaneously or in succession, and the heart also (rheumatism)." More recently his views have undergone some modification, and he states[3] that all the latest evidence points to the breaking down of some special nitrogenous substance, probably nuclein, rather than to simple deficient oxidation of ordinary products. Vaughan[4] also derives uric acid from the breaking down of nuclein.

Klemperer finds that uric acid exists in normal blood in quite inappreciable amount, but that in gout it reaches .067 to .091 grammes, similar quantities being present in leucæmia and nephritis. He further finds no definite relation between the uric acid in the blood and that in the urine, nor between the amount present in the urine at any time and the attacks of acute gout, the blood in gout being by no means saturated

[1] *Croonian Lectures on Rheumatism, Gout, and Diabetes*, 1886.
[2] *Uric Acid as a Factor in the Causation of Disease*, 1894, 2d ed. p. 83.
[3] *Brit. Med. Journ.*, vol. i., 1896, and *Edinb. Med. Journ.*, vol. xli., ii.
[4] *Assoc. Amer. Phys.*, May 4, 1898.

with uric acid. He therefore concludes that an excess of uric acid is not the sole cause of gout, for the solvent power of the blood serum is greater than the requirements of all deposits.

Roberts found that a proportion of uric acid in the blood greater than 1 part in 6000 amounts to over-saturation, with consequent precipitation, whereas a proportion of 1 part in less than 10,000 is a state of under-saturation which enables the blood to reabsorb urates. He believes in an increased absorption of urates by the blood, aided by diminished renal excretion, and regards even the visceral symptoms of gout as due to a temporary urated deposit.

Levison attributes the increase of uric acid in the blood to the action of leucocytes, and thinks that uric acid accumulates mainly through non-elimination by granular contracted kidneys. Harbaczewsky agrees with Levison that the formation of uric acid is due to the breaking down of nucleinic-acid compounds of albumin, derived especially from the leucocytes.

The poisonous effect of uric acid in the system, alone and aside from other injurious agencies, has been no doubt much overrated. Bouchard maintains that it is quite innocuous even in large quantity, and it is promptly eliminated as urea. "Neither does the gouty man, saturated with uric acid, present on the eve of his attack any of those symptoms which have been described as resulting from uric-acid intoxication" (Ewart).[1] I have many times given it to animals hypodermically in quantities far exceeding their average daily output, without producing any noticeable toxic symptoms, and considerable doses may be taken by man per os without injury (Haig).[2] In all fairness it must be admitted that uric acid cannot now be regarded as the "essence of gout"—*i. e.* it must be regarded either as a by-product or as one of several agents contributing the symptoms of the disease. In support of this statement I quote again from Ewart's[3] admirable work: "We still hesitate, then, to admit without further investigation that gout is primarily dependent upon uric acid as a cause, or that uric acid necessarily exerts any exclusive influence in the production of the phenomena of goutiness." Bunge[4] expresses a similar opinion.

It should be emphasized, moreover, that other uricacidæmias besides the gouty do not precipitate urates. Thus in leucæmia the blood continually contains more uric acid than the normal (Bunge),[5] with a correspondingly low coefficient of excretion in the urine. In this case the fault seems to lie chiefly in defective elimination. The same may be said of pneumonia; but in anæmias the accumulation of excess of uric acid is ascribed by von Jaksch[6] to diminished oxidation.

At the time of the acute attack of gout the blood is found to hold much less uric acid than when the pain is absent and the temperature normal (Garrod and Haig).

2. *The nervous theory*, which was advanced by Cullen in explanation of the origin of gout, has been modified somewhat by Sir Dyce Duckworth[7] to include a diathetic "habit," which may be either inherited or

[1] *Op. cit.*, p. 92.
[2] *Brit. Med. Journ.*, Oct. 3, 1896.
[3] *Op. cit.*, p. 143.
[4] *Pathological Chemistry*, p. 333.
[5] *Ibid*, p. 340.
[6] *Deut. med. Woch.*, 1890, No. 23.
[7] *A Treatise on Gout.*

acquired, and from which, on the one hand, rheumatism may be derived, on the other, gout. The nervous system is held responsible for the non-regulation of the nutritive processes of the body whereby proteid metabolism is incomplete, uric acid accumulates, and gout results. The influence of a disturbed nervous system in accentuating the manifestations of gout, and the additional nervous symptoms so commonly associated with gout, lend support to this theory.

Ord, Laycock, Mortimer Granville,[1] and others have favored the theory of altered innervation, and Buzzard assumed a hypothetical centre for controlling the nutrition of joints, which was supposed to exist in the fourth ventricle. Duckworth suggests that the seat of the nervous disorder may be in the medulla. Latham also upholds the nervous theory, believing that a central nerve lesion or functional disorder initiates hepatic derangement as an antecedent of gout. Sir Willoughby Wade believes that the origin of gout is a neuritis rather than a neurosis. He also advocates the neural theory of the pathogenesis of gout.[2] According to his doctrine, some aberration of proteid metabolism causes an excess of quadriurate of sodium in the blood, lowering its alkalinity. The stability of the nerve trunks is thereby upset and the nerves become an easy prey to irritation. Neuritis or neuralgia ensues, and the final outcome is a neuropathy affecting any physiological division of the nervous system—vasomotor, trophic, or motor. Local outbreaks are determined through the vasomotor nerves. The neuritis may be excited by a primary inflammation of the joint or originate independently. Wade emphasizes the frequent tenderness over the knuckles, backs of the hands, hypothenar region, the heels and plantar surfaces, while the trophic lesions are illustrated in the skin, thin, brittle nails, joints, etc. He finds that tenderness extends beyond the zone of redness and swelling of the great toe, following the dorsal branch of the peroneal nerve, over the inner side of the great toe, the bend of the ankle, etc. He argues that a primary mechanical irritation of the joint produced by biurates would not act with the suddenness with which an acute attack begins, which is better explained by an initial neuropathy. The common localization of the lesion in the great toe joint is not due, he believes, to its remoteness or to the weight of the body, for the other toes are as remote in regard to their circulation, and the ankle is subjected to even more weight and pressure than the toe.

Ewart is disposed to attach importance to many of Wade's views, and believes that in a limited sense, at least, a tropho-neurosis explains the periarticular nerve complication, which is congestive and accompanied by the œdema started by the irritation with biurates of the intra-articular nerve endings.

Among the striking features of gout which point to a possible nervous origin are the heredity, the paroxysmal character of the symptoms, the nervous irritability, the depression, and the various accompanying neuroses, such as asthma, neuralgia, sciatica, etc. It has been suggested also that the kidneys may fail in function through deficient innervation.

[1] *Lond. Med. Press and Circ.*, Feb. 15, 22, Mar. 1, 1894.
[2] *Gout as a Peripheral Neurosis*, London, 1893.

3. *The nutritive tissue-disturbance theory* of Ebstein is based upon the existence of a fundamental weakness of the tissues, especially the muscles, whereby they undergo prior necrotic change, leading to the subsequent deposition in them of urates. An over-production of urates may excite the local inflammation and necrosis, followed by urate precipitation. Ord holds an analogous view in regard to a predisposing specific tissue weakness, combined with altered innervation. Ebstein claims to have produced gout artificially in fowls by ligation of the ureters, and he believes that uric acid is formed in the bone marrow and muscles, whence it is conveyed to the cartilages. Cantani holds that it is formed in the cartilages and connective tissue. Ewart[1] writes: "Modern research indicates that, as suggested by Parkes and others, there are two main aspects to gout—the degenerative changes in the tissues and the uric-acid trouble—and that these may be simply the two phases of a single process connected with cell malnutrition."

PATHOLOGICAL ANATOMY OF GOUT.—Since acute gout is merely an exacerbation of a really chronic disease, no rigid line of morbid anatomical distinction is to be drawn between them. Each succeeding acute attack will leave more and more structural change in a joint, while more insidious changes may be slowly progressing between the exacerbations. The latter are to be regarded merely as the expression of an unusual degree of irritability temporarily excited in certain tissues and organs by processes which have fundamentally the same cause—namely, deposition of crystalline acid sodium biurate. It is therefore best to study both the acute and chronic lesions together, the one merging into the other in rotation.

The essential morbid changes in gout concern the blood, the joints, the viscera, notably the kidneys (and urine), the heart, and vessels. Of these changes, by far the most serious, as regards prolongation of life, are chronic nephritis and arterio-sclerosis. Other minor lesions will be described in turn.

The blood was shown by Garrod (in 1848) to contain an excess of uric acid, as it does oftentimes in lithæmia, hepatic cirrhosis, chlorosis, and leucæmia. Its presence is easily detected by the following method, originally devised by Garrod. The skin of the outer arm is cleansed and a small circular piece of cantharides-paper is applied, and over this an ordinary corn-plaster is bound. The resulting blister rises in the hollow of the plaster, which limits and protects it. The serum is then withdrawn in a watch-glass and two or three drops of 28 per cent. acetic acid are added. In a few hours uric-acid crystals will begin to precipitate, and if a linen thread be left immersed in the serum the crystals will adhere to it in sufficient number to be readily seen with the microscope. This thread test must be regarded merely as confirmatory, not as positive. Serum drawn from the immediate vicinity of the inflammation contains no uric acid, as the latter is in some manner altered by the inflammatory process, being probably converted thereby into the practically insoluble sodium biurate. Roberts[2] found this salt to be insoluble in blood serum, synovia, and other body fluids in proportion greater than 1 : 10,000. He showed experimentally that uric acid when

[1] *Op. cit.*, p. 317.

[2] "Croonian Lectures," *Brit. Med. Journ.*, June 18, 25, July 25, 1893.

first added to blood serum unites with sodium carbonate, producing a quadriurate, but subsequently it takes another molecule of the base, and the much less soluble biurate results, which precipitates in the fine needle-shaped crystals found in the gouty joints.

The fact must not be overlooked that the blood may contain small quantities of uric acid in health. The proportion of uric acid to urea may then reach 2.8 per cent., whereas in gout it may exceed 3.1 (Kammerer.[1]) Halliburton[2] finds much greater variation, and says that the percentage of uric acid in the blood of the gouty may range from 0.004 to 0.175.

Oxalic acid, so closely allied to uric acid, is also sometimes found in the blood, but there are cases even of an advanced type, with abundant tophi, in which the presence of neither acid can be detected.

The blood does not become so rapidly anæmic as in acute rheumatism, and it is mainly in chronic gout that this symptom is demonstrable. The coagulability of the blood is somewhat increased, and it is rich in fibrinogen.

The Joints.—The most typical joint changes are hyperæmia and swelling of the ligaments, capsule, and synovial membrane, with inflammatory exudation into the joint.

These lesions have been carefully studied by many observers, notably by Ebstein. He believes that the first changes are caused by the excess of urates circulating in the blood, which become arrested in the less vascular tissues of the joints, causing by irritation local coagulation necrosis. This is most marked in those tissues, like the cartilages, in which the blood current is feeble and the reaction of the parts involved is acid. The veins about the joints become swollen and congested, and a serous exudate, thickened with acid-urate crystals, accumulates in the joint cavity, in its serous membranes, and often in the bursæ or tendon sheaths of the part. Sometimes the periosteum is infiltrated. Œdema extends in the neighborhood, and if the great toe be first affected, the entire dorsum of the foot and the ankle may share in the œdema, and give rise to "pitting" on pressure. The articular cartilages are first involved, showing either irregular necrotic foci or a uniform distribution of the process. The urate infiltration is interstitial, covered at first by a thin layer of cartilage, which disappears as the deposit thickens, successive layers being formed in renewed attacks. The deposit is usually most dense in parts where the circulation is poorly developed. The fibro-cartilage everywhere and the ligaments soon become included in the process, and the tophi or chalk concretions ("chalkstones") may be felt in them. The ligaments become relaxed and much thickened, and true exostoses may form, restricting the motion of the joint, or in some cases causing permanent ankylosis.

This latter statement has been denied by eminent authorities. Fagge,[3] for instance, said that "bony ankylosis is unknown," and "it is very rare to find the cartilage gone in a gouty joint." Nevertheless, ankylosis must be admitted as present in some advanced cases, and such instances have been cited by Cornil and Ranvier.

Frederick Taylor[4] says: "The cartilage may eventually get quite

[1] *Deut. med. Woch.*, Mar. 5, 12, 1892.
[2] *Loc. cit.*, p. 307.
[3] Fagge: *Principles and Prac. of Med.*, vol. ii. p. 799.
[4] *Practice of Medicine*, p. 848.

destroyed and eroded down to the bone," and William Ewart[1] writes: "The late changes are the lipping of the rim and the not infrequent synostosis of the articular surfaces of the bones, the bony nature of the marginal outgrowths, the bony ankylosis, and the absence of the eburnation special to osteoarthritis, which are characteristic skeletal features of gout, and enable us to identify the disease from the mere inspection of bones long buried. The occurrence of total ankylosis is facilitated by the incrustation and stiffening of the ligaments—conditions which yet more often lead to a spurious ankylosis."

The joint cavities themselves never suppurate, but, according to Fagge and Garrod, bursal abscess near the articular cartilages has been seen. The bursa over the olecranon may become enlarged.

Tophi (from τόψος, a rough, crumbling volcanic rock, *tufa*) is the name given to the gouty concretions when they become sufficiently large to be perceptible. They are composed in the main of sodium biurate in crystalline and granular masses, but other salts may be present. Next in amount are calcium urate and sodium chloride. Traces of calcium phosphate have been found (Lehmann), possibly derived from fragments of bone, although Halliburton[2] suggests that "sodium urate may set up inflammation and become infiltrated with calcium phosphate, as tubercular matter often does. Wurzer found a trace of potassium chloride.

The tophi crumble easily and can be made to rub off a white chalk-like mark. They dissolve readily in nitric acid, and then yield the murexide test for uric acid. This test is performed by evaporating the nitric-acid solution to one third the original bulk, when, upon the addition of ammonia, a brilliant purple hue appears.

Acetic acid also dissolves the sodium biurate deposits and precipitates uric-acid crystals, recognizable with the microscope.

Microscopically, the tophi appear as opaque clusters of radiating crystals or forming a network of needles in the joints.

The number and distribution of the tophi vary. For the most part, they are found at the sides of the affected joints, in the integument about them, in the ligaments, in the tendon sheaths, and finally, when they grow large and numerous, in the joint cavities. They are also quite common in the helix of the ear, where they appear first as small soft, pultaceous masses, hardening in a few months to shot-like nodules or "pearls," which may be both felt and seen as yellowish masses beneath the integument. They may be at the border or in the centre of the helix, and one or two, or a couple of dozen may be present in either ear. They are easily distinguishable from enlarged sebaceous follicles, and are very pathognomonic. Their presence in the ear is promoted, according to Wade, by pressure in sleeping.

Tophi occur exceptionally in other situations. Fagge[3] saw them in the skin of the mid-thigh and the arms, and they have been observed in the tarsal cartilages at the angles of the eyes, in the eyelids and sclerotic layer of the eye (Garrod), in the skin of the penis (Charcot), and in the epiglottis (E. Grawitz). Hayem reported as a rarity the presence of sodium-biurate crystals in the intestinal villi in a bad case of advanced gout (Norman). They have been seen in the trachea, and the

[1] *Op. cit.*, p. 115.
[2] *Text-book of Physiolog. and Patholog. Chemistry*, p. 510.
[3] *Op. cit.*, vol. ii. p. 800.

crystals have been found in the perspiration (Henry). Norman Moore found them once upon the meninges of the cerebellum, and they have been recognized by Garrod in the pericardial and pleuritic fluids. Charcot found them in cephalo-rhachidian fluid, and Bird in the secretions from eczema, and Ollivier on the external dura mater of the spinal cord and sheaths of the spinal nerves, and in the prostate gland.

Tophi have been found in the membrana tympanum. Virchow has reported tophi in the right vocal cord, Garrod in the arytenoids, Norman Moore in both vocal cords, Litten in the crico-arytenoid ligaments and joints, and van der Kolk on the valves of the veins.

The tophi are at first more or less movable, and the skin over them is non-adherent, but later adhesions form and protrusion of the masses takes place through the integument. In this manner they may be extruded and cast off. "Then you have chalkstones like crab's eyes exposed to view, and you may turn them out with a needle" (Sydenham [1]).

They often soften, and discharge for a long time a creamy substance containing urate crystals. Portions of the tophi remain and dry, or they grow and fuse together until all joint structure disappears and the masses attain the size of chestnuts or even of eggs. They are usually rough and nodular. More rarely they set up a local inflammatory process of active ulceration.

The escape of emboli from tophi is exceptionally rare.

The Bones.—The bones themselves are seldom involved in gout beyond the occasional lipping and ankylosis above referred to (page 1004), but urates have rarely been found deposited in the extremities of the long bones near their articular surfaces (Cruveilhier), and Sir Dyce Duckworth [2] has found them in the bones independently of cartilaginous deposit. E. Pfeiffer [3] claims that a true increase in osseous tissue may occur, and cites a case with illustration. The periosteum may be infiltrated with biurates though the bones escape.

The *kidneys* in advanced cases present the lesions of the granular contracted type of nephritis or of sclerosis, but the so-called "gouty kidney" is not pathognomonic, and the urates which are sometimes found deposited in the papillæ are not peculiar to this disease.

The Heart and Bloodvessels.—Arterio-sclerosis is one of the commonest of gouty lesions outside of the joints. In acquired gout it may develop after several acute attacks, but in the hereditary form it is often one of the earliest manifestations. It often reaches a high grade, and by increasing the peripheral resistance leads to cardiac hypertrophy, which is quite common, unaccompanied by valvular lesion. Gouty phlebitis has been described (van der Kolk) with malnutrition of the wall of the vein, with consequent thickening and dilatation. Thrombosis with a firm œdema, pain, and tenderness may follow. Sir Andrew Clarke [4] has described the occurrence of hæmoptysis in elderly persons, which he regarded as of gouty origin.

[1] *Loc. cit.* [2] *A Treatise on Gout.* [3] *Lancet*, April 11, 1892.
[4] *Trans. Med. Soc.*, London, 1890, vol. xiii. p. 9.

ACUTE GOUT.

ETIOLOGY.—The general etiology of gout has been discussed on page 994. Acute attacks are more often precipitated by dietetic errors than in any other way. A single grape or a few sips of sherry or port may excite an attack. Many noted writers upon gout, like Sydenham, Gull, Garrod, and Haig have been themselves sufferers from the malady, and have devoted much attention to the exciting causes of acute gout. The results of dietetic indiscretion are usually remarkably prompt, and the transgressor from dietetic rules may feel the twinges of the approaching paroxysm before he rises from the table.

Among other exciting causes of acute attacks are mentioned venereal excesses, pregnancy, mental anxiety, worry, fatigue, overwork, violent bursts of temper. Local injury to a joint, while not in itself primarily a cause of gout, may determine the eruption of the impending disease in the particular joint which has been bruised or sprained.

Acute gout may occur without warning in a patient as a first experience, or it may occur as a sudden intensification of a chronic case.

SYMPTOMS.—The distinctive symptoms of acute gout are the local joint manifestations of pain, swelling, redness, and tenderness, accompanied by more or less constitutional disturbance. These symptoms usually occur simultaneously, or at least are present together at some time during an acute attack, but one or more may sometimes be absent. Other symptoms may occasionally appear, such as disorders of the mucous membranes, especially of the stomach and bowels, catarrh of the respiratory passages, alterations in the composition of the urine, and various forms of irritation of the nervous system and skin. These other symptoms, however, together with arterio-sclerosis, may be met with in chronic gout or in the gouty diathesis.

Typical Case.—The symptoms of a typical case begin as follows: Premonitory symptoms often appear, especially in patients who have had previous attacks, or they may precede a first attack. There is usually more or less digestive disturbance at the onset of acute gout. Often symptoms of "biliousness" precede the attack by several days. The patient has anorexia, a heavily coated tongue, offensive breath, and gastric dyspepsia. The stools are not natural, and may be either too loose or constipated.

Restlessness, nervousness, insomnia, and irritability of temper are noted. There may be pharyngitis, and asthmatics are apt to have an attack of asthma. In a majority of cases the condition of the urine is a good indication of an impending seizure. It becomes somewhat scanty, high colored, of high specific gravity (1030–1033), is loaded with urates, the normal acidity is increased, the uric acid is markedly lessened, transient albuminuria is common (Garrod), and in some cases, especially in obese subjects, transient glycosuria[1] is also observed.

When premonitory symptoms are wanting, the patient, who perhaps has been feeling exceptionally well, experiences a very sudden severe

[1] The occurrence of gouty glycosuria and of gout with diabetes will be found fully discussed by Warren Coleman in the article upon Diabetes, in Vol. III. p. 834, and the coincidence of gout, glycosuria, and diabetes is described by the writer in the article upon Obesity, Vol. IV. pp. 1035, 1036.

twinge of pain in the great toe. This pain often comes on at night, or it may awaken him from sleep during the early morning hours. It is characterized by dull constant aching with frequent exacerbations, "twinges" of a lancinating character, causing the patient to cry out in agony as if a knife were being thrust into the joint, wearing him out with restlessness and suffering. No matter how severe, it remains localized in the joint affected.

Bearing the weight upon the foot greatly intensifies the pain, but the latter recurs with increasing severity, independently of pressure or motion, until the agony becomes insupportable. Besides the pain in the toe, ball or arch of the foot, there are often muscular cramps in the leg. In a short time, two or three hours, swelling of the metatarso-phalangeal joint is observed, accompanied by an area of redness.

The entire region about the joint is hot (2–4° F. above the normal), shining, slightly œdematous, and immediately over the sides of the joints are lines of exquisite tenderness.

The redness is intense, circumscribed, and corresponds with the area of swelling. The swelling varies in degree; it may be slight in proportion to the pain, or it may be considerable, so that the joint appears double or even treble the ordinary size.

The local increase in heat in the joint is frequently quite perceptible to the touch, and may be felt by the patient himself. The tenderness is extreme, both superficial and deep, and the dread of pressure is very great; jarring of the room by the slamming of a door or a heavy tread may excite intense suffering.

The veins about the joint and often in the leg appear swollen, blue, and prominent.

There may be slight rigors and nausea. Constipation is often present. Anorexia usually accompanies the fit. Nausea and vomiting are less common. The tongue is heavily coated, the breath is offensive, and thirst is complained of. The temperature rises early, but is seldom very high (101°–103° F.), and the pulse rate is somewhat quickened (80–100). The signs of fever subside in the morning but return again at night, and the temperature is commensurate with the degree of local inflammation. Salicylic acid and quinine are alike without effect upon the temperature. It is characteristic of the pain to abate or be absent during the daytime and recur at night. With the abatement the patient may perspire gently and recover the sleep of which the pain deprived him. The tenderness and swelling, however, remain constant until the gradual subsidence of the attack, which lasts from three or four days to a full week. A very violent acute attack is often of shorter duration than one less severe.

Lécorche and others have proved that the uric acid excreted remains deficient before and during the early part of the seizure, then suddenly increases after two days, and again gradually diminishes. Immediately after the subsidence of the "fit," desquamation accompanied by itching takes place over the inflamed joint. Exceptionally, loss of the toe-nail may follow.

The patient promptly recovers, and often feels that his general health has been improved by the attack, but soreness and tenderness may remain in and about the joint for weeks after the subsidence of acute symptoms.

SPECIAL SYMPTOMS.—*Pain.*—The above account applies to a typical case. The attack is sometimes prolonged for a fortnight, or in elderly persons for a month or more, but, on the other hand, in many persons a first attack is much less severe, and all degrees of intensity occur from a feeling of unusual tightness of the boot, or the persistence of a slight sprain, to attacks in which the agonized victim can only describe the pain as a feeling as if the great toe were being wrenched from him with red-hot pincers or were being gnawed by a wild beast. Extreme restlessness characterizes such pain, the patient vainly seeking positions of ease, and yet being intolerant of motion in the joint itself, and even of jarring of the bed or room. Naturally, pain of such acuteness begets loss of sleep and great irritability of temper, and an otherwise amiable person may lose all self-control in this regard. Sydenham, who endured the disease himself for thirty-four years, wrote that "a fit of gout is a fit of bad temper."

Swelling.—While the metatarso-phalangeal joint of the great toe, and in three fourths of all cases that of the right toe, is commonly first involved, sometimes both great toes are implicated in a first attack, or, especially if there has been a preceding local sprain or injury, the symptoms may begin in the knee, ankle, in the metacarpo-phalangeal articulation of the forefinger, or more rarely still a large number of different joints may be simultaneously affected. In the latter case constitutional symptoms are more severe, and some difficulty in diagnosis from acute rheumatism may arise, although the swelling is rarely so great as in the worst cases of the latter disease. Sometimes the swelling of the joint precedes the pain; sometimes the occurrence of the swelling relieves a previously intense pain.

Muscle pains sometimes are complained of in the lumbar region, neck, calves of the legs, etc. The pains are worse in the early morning hours, and may be due to the fever or the gout itself.

The *temperature*, which, as a rule, is intermittent, is proportionate to the duration and severity of the local acute inflammation, and moderate cases may be almost afebrile, which is in striking contrast with rheumatism. Cases of hyperpyrexia are from time to time reported (Montagnon),[1] but are much less common than in rheumatism. As in other local inflammations, the temperature may be slightly elevated in the affected joint, which may feel hot to the touch.

The *color of the skin* over the affected joint is noticeable. It is of a darker red or purplish hue than in rheumatism, with a tendency to shading toward a darker central focus. Pressure momentarily removes the color, but it at once returns afterward, and with it is an intensification of the pain, as the swollen vessels are again distended. The intensity of the discoloration keeps pace with the development, severity, and decline of the other symptoms, although it usually takes twenty-four to thirty-six hours for it to reach a maximum.

The *urine* becomes hyperacid in gout from excess of its normal acidifying salt, the acid phosphate of sodium. This salt is known to antagonize the solution of urates. Roberts[2] finds that the highly soluble quadriurates are the normal salts of healthy fluids of the body. In gout these salts, by lessened alkalinity of the blood and greater acidity

[1] *La Loire médicale*, July 15, 1891.

[2] *Lancet*, June 18 and 25, 1892.

of the urine, are hindered from ready solution and excretion, and become converted into the very insoluble uric-acid salts, the biurates which find lodgement in the joints and various tissues. This author says:[1] "The conditions of the urine which tend to accelerate the precipitation of uric acid are high acidity, poverty in salines, low pigmentation, and high percentage of uric acid." He regards the increased acidity as the most potent of these factors.

Albumin is not necessarily present in gouty urine, but Garrod regards transient albuminuria as of frequent occurrence during an acute attack.

By experimental injection in rabbits it has been shown that the general toxicity of the urine is actually lessened during a fit of gout.

The quantity of the urine may be lessened during the most acute part of the gouty paroxysm, as it often is with any fever, but later, and especially in chronic cases, it may be increased above the normal, especially at night. Modifications in the quantity will naturally depend also upon existing interstitial nephritis and upon the dietetic and other treatment of the case.

Uric Acid.—In the urine this acid is undoubtedly diminished in acute gout (Garrod, Cantani, E. Pfeiffer,[2] Lécorche (page 1000), Haig, and many others). There has been some conflict regarding this point, and it is important in every instance to determine the ratio of uric acid both to urea (the normal ratio is uric acid 1, urea 35) and to the total quantity of urine excreted. When this is done over a considerable period of time, Haig has shown that the uric acid-urea ratio remains for the whole period nearly normal. The diminution in uric-acid excretion occurs either immediately before the fit, or within the first forty-eight hours of it, and this being later followed by an increase above the normal, the total deviation from the normal during the entire attack may not be in any way remarkable. Hence it is easy to over-emphasize the importance of uric-acid tests in gout, and allowance must always be made for an excessive nitrogenous diet and for existing advanced nephritis.

Uric acid is not only demonstrable in the blood in acute attacks of gout, as above described (page 1003), but it may be detected also in lymph, serous fluids, and exudations, and even in the moist discharge from an eczematous surface. It has been found in the perspiration in severe cases. Garrod by quantitative research showed that the uric-acid crystals when plentiful in such fluids as those just mentioned are in the proportion of 1 : 6000 or 1 : 7000, and they are not obtainable at all in less proportion than 1 : 65,000.

E. Pfeiffer[3] endeavored to demonstrate that by filtering the urine of a gouty patient through filter-paper on which had been placed crystals of pure uric acid, some idea would be formed of its capability of dissolving additional uric acid. Roberts[4] pointed out certain fallacies in the method, which was in turn defended by Pfeiffer, but the test has not proved of practical value.

Relapse.—The fit having subsided, the patient may be so fortunate

[1] *Loc. cit.*
[2] *Berl. klin. Woch.*, Nos. 16, 17, 19, 20, 22, 1892.
[3] *Trans. Seventh Congress of German Phys.*, at Weisbaden, 1888.
[4] *Brit. Med. Journ.*, June 14, 1891.

as to escape a repetition for two or three years, though in bad cases it will return in a year's time or less. It is very certain to return at some future time, especially in hereditary cases or in those in which patients pay little heed to prophylaxis. As the seizures recur they usually tend to become somewhat more severe, to recur at shorter intervals, and to involve additional joints. These repeated attacks may originate the anatomical changes of chronic gout, causing permanent swelling and distortion of the affected joints, with palpable accretions of sodium urates.

In some instances patients find themselves anæmic and debilitated after an attack, with flabby toneless muscles, and several months may be consumed in establishing convalescence.

Prognosis.—A first attack of acute gout is rarely ever fatal, and in fact it is seldom that the attack in itself, however often repeated, is so, death being caused either by the various compli tions—especially renal, cardiac, or vascular—of the malady, or by intercurrent diseases which act with greater energy upon an enfeebled gouty constitution. So long as the exacerbations of the disease are confined to the periphery, and to one or two joints, as that of the great toe, the patient is in less danger. Much depends upon heredity, willingness to adopt a more rational mode of life, and no doubt also upon strength of constitution. There are those who, in spite of leading most exemplary lives and availing themselves of every aid which the most intelligent treatment can afford them, have repeated and severe attacks, which they survive but two or three years. Such patients are usually the victims of strong gouty heredity or of enfeebled constitutions. On the other hand, it must be admitted that one meets with others who disregard every warning, both moral and physical, and despite acute attacks recurring every year or two, live on in comparative vigor until a somewhat premature old age.

In any given case the prognosis may be made favorable if the attack is mild, short, yields well to treatment, does not involve the viscera, and does not recur within a year, provided always that the patient will shun that which for him is evil. But pain is soon forgotten, and in many cases the patient feels so well after an attack that his good intentions vanish, and he can no more refrain from gluttony than can the alcoholic from his bottle. Finally, when the lessons of penance have been often enough repeated, it is too late, and the patient cannot be saved from the enduring miseries of chronic gout, permanently invalided, saturated with urates, and possessed with kidneys and a heart which will suddenly fail him if the slightest additional strain be put upon them. Among particularly discouraging indications are lessening intervals between acute attacks, the progressive involvement of different joints, and failure to wholly recover the general health between the seizures.

Diagnosis.—The diagnosis of acute gout is based upon the localized lancinating pain, redness, tenderness, and swelling in the affected joint or joints, the common occurrence of the pain at night, or in the early morning hours; the distended veins and tense shiny skin over the joint, the predilection for the great toe, and the disproportion of the moderate fever to the intense local inflammation.

Acute articular rheumatism must be differentiated from gout. In the former there are apt to be several joints involved simultaneously or in

rapid succession, whereas in gout the disease shows special predilection for the tarso-phalangeal joint of the great toe, and even when other joints are involved there is less tendency for the local symptoms to be migratory. Rheumatism elects the larger joints, especially in first attacks, and although the fingers may be involved, it is much less common in the toes than gout. Moreover, it often involves joints like those of the shoulder or hip, which are seldom affected by gout. Occasionally, however, acute gout will attack a number of joints simultaneously, and give rise to considerable difficulty in diagnosis, but in these and other cases the effects of treatment are radically different. Rheumatism may yield to the salicylates, whereas gout does not, but is improved by colchicum, which in turn is without effect upon rheumatism. As the inflammation of the rheumatic joint subsides, the latter rapidly assumes its normal appearance, whereas in gout the joints may become permanently deformed by repeated attacks, and deposits of sodium urate are often to be felt about them. There are no tophi in the lobes of the ears in rheumatism. Acute rheumatism is much more common than acute gout, especially in women and in the young; it is more apt than gout to be excited by cold, damp, and changes in the weather, and less apt to be excited by errors in eating and drinking, and lack of exercise.

Abscess or a localized sepsis in the great toe would rarely be mistaken for acute gout, although such errors have been made. In gout true fluctuation and the constitutional symptoms of sepsis, chills, sweating, and irregular high temperature are absent. In abscess the pain is more of a throbbing, less of a lancinating, character, and it does not typically appear at night.

Treatment.—The treatment of acute gout is (1) prophylactic; (2) local; (3) constitutional; (4) dietetic and hygienic.

(1) *Prophylaxis.*—In those who inherit gouty tendencies, or who have had previous attacks of gout, much may be done by way of prophylaxis by regarding dietetic rules and living regular and systematic lives. The avoidance of dyspepsia and the maintenance of activity of the skin by frequent bathing and friction, and of the kidneys by drinking abundant simple fluids, such as mineral and effervescing waters, should be the chief attainments. A sedentary life must be abandoned, and systematic outdoor exercise designed especially to increase the breathing capacity and promote more active oxidation processes is desirable. The control of the diet is easily understood from the discussion under Etiology of Gout (page 998) or the influence of diet and alcohol. Further considerations applicable to prophylaxis are given under Treatment of Goutiness (page 1025).

(2) *Local Treatment.*—Acute gout is a brief and self-limited disease. Cullen expressed the opinion that most cases are cured by "patience and flannel." Local treatment usually accomplishes even less than in the case of rheumatism. Moreover, if the pain in the toe-joint be very agonizing and the tenderness extreme, local applications may not be tolerated at all or may prove of no avail.

The affected joint is to be placed in any position most comfortable to the patient, elevated upon pillows, protected by a cradle from the weight of bed clothing which is intolerable, and guarded against all jarring of the bed or room.

Alkalies.—One of the most soothing of local applications is a warm solution of sodium bicarbonate, ℥j to the pint of water. To this ℥j of laudanum may be added if desired. Yeo prescribes carbonate of sodium ℥ss, laudanum ʒiij, in ℥x of water, to be added when used to an equal quantity of hot water. The joint is packed with lint steeped in the solution, wrapped in cotton-wool, and covered with oilsilk.

Cold is usually less grateful than moderate warmth to the joint, although in some cases it is more comforting. Wade, in accordance with his neuritis theory (see page 1002), pencils the branches of the peroneal nerve with sharp pieces of ice.

Hot applications are used by many, and they may be made by means of cloths dipped in very hot water, by steaming the joint for ten minutes, or by hot air. The latter method is especially useful where tenderness is very great, and various forms of portable apparatus are readily adjusted about any joint. The warmth and moisture seem to relieve vascular tension, and the hot air (200° F. or more) causes intense local sweating, which may deplete the joint of some of its inflammatory products.

Ointments, paints, etc. of all kinds have been tried. Solutions may be painted over the joint with a cotton swab or camel's-hair brush, and ointments should be gently smeared and not rubbed in. Ransom applies extract of belladonna ʒj, with glycerine ℥j, over the inflamed joint, which is then encased in cotton batting and oilsilk. Duckworth recommends atropine and morphine dissolved in oleic acid for the same purpose. The tinctures of opium and belladonna in equal parts may be similarly applied. A solution which sometimes gives relief is composed of menthol and chloral āā ʒj, with camphor water and alcohol āā ℥j. Ichthyol ʒj to vaseline ℥j may afford relief. One of the best of recent topical applications is a mixture in equal parts of guaiacol and glycerine, applied with a brush twice a day. Aconitia may be soothing, but is too dangerous for common use.

(3) *Constitutional Treatment.*—As Ewart[1] writes: "Gout itself is curable, but some of its worst results are not." The only remedy which for many years has been regarded as specific is colchicum, but there are many other considerations to be regarded besides its routine prescription.

The medicinal treatment should be inaugurated by active purgation to relieve the overloaded bowels and overworked liver. To robust subjects, 5 grains of blue mass or 5 to 10 grains of calomel, with an equal quantity of sodium bicarbonate, are to be given at once.

If the stools are somewhat loose, smaller doses of calomel (gr. ½) may be several times repeated. Carlsbad salts are much in vogue in all stages of gout, but in the acute attack under consideration nothing is so good as the mercury, which not only rids the system of much offending matter, but favors the use of smaller doses of other remedies.

Colchicum often produces a magical effect within a few hours by reducing temperature and causing subsidence of the pain and other local symptoms. It acts as a diuretic and laxative, both of which effects are most desirable, and it stimulates the liver. It should be remembered that the tincture made from the seeds is about one third stronger as an

[1] *Loc. cit.*

aperient than the wine. Moreover, colchicum may be cumulative in action, unless care be taken to promote its elimination by the use of carbonated alkalies or mineral waters. As a rule, owing to the quicker action desired, the fluid preparations are preferable for acute gout to pills of the extract or acetic extract, and it is well to commence with full doses and taper off, or pursue the intermittent plan of dosage. Ewart advises the use of ʒss to ʒj of the wine as an initial dose, which, however, must not be maintained at this rate. For the milder cases ♏xii or xv will suffice, and it is well to add magnesium carbonate (ʒss) with spirits of chloroform (♏xx) or spirits of peppermint (♏v), or some other carminative. If a stronger diuretic effect is desired, the citrate and acetate of potassium may be given. As the symptoms improve the colchicum dosage should be confined to ♏x or xv two or three times a day. While this remedy has proved the chief reliance in the treatment of gout for many years, nothing having yet appeared which will wholly replace it, if given in larger doses than those mentioned, or if too long continued, it is very apt to irritate the kidneys and alimentary canal, producing strangury and hæmoglobinuria on the one hand, and on the other nausea, vomiting, epigastric pain, and severe diarrhœa, with perhaps melæna. The diarrhœa may occur without apparent gastric disorder. I have seen an almost fatal diarrhœa produced on several occasions in elderly persons to whom the drug had been carelessly administered. When such symptoms supervene the drug must be suspended for several days, when its use may be resumed with great caution.

The practice initiated by Garrod, which is a very good one in some cases, is that of giving a saline cathartic with each dose of colchicum, the idea being that the latter remedy may be used in much smaller doses and is rendered less irritant by the combination.

Colchicin, the active principle of colchicum, was introduced several years ago with the hope that it would prove less irritating, and it is preferred by many clinicians on the ground that the dosage is more easily controlled. It has been given hypodermically in doses of gr. $\frac{1}{50}$ to $\frac{1}{20}$. It can be given per os (gr. $\frac{1}{24}$) when colchicum itself has proved intolerable, but it is fully as irritating.

Some few patients possess great idiosyncrasy against colchicum in any form, being severely purged by small doses, or made otherwise violently ill by it.

In such cases relief of the pain is to be sought in other ways. Morphine may be given hypodermically, though with great caution if renal inadequacy exist or a granular kidney be suspected. A dose of phenocoll (gr. v) or of phenacetine (gr. x) in soda-water may afford relief.

Sodium salicylate is a remedy preferred by Haig in as large doses as those in which it is given for rheumatism. Haig's views of the common etiology of gout and rheumatism have been cited (page 1000). He claims that salicylic acid removes the biurates as soluble salicylurate. Granular kidneys and a disordered stomach are contraindications for this remedy. H. C. Wood recommends the ammonium and strontium salts of salicylic acid for acute cases as less liable to upset gastric digestion.

Piperazin, an alkaloid of the pyridine group, is a drug which has met recently with some favor in the treatment of gout. It is non-poisonous

and non-irritating, and is given in doses of 15 grains three times a day. Being very deliquescent it is necessary to prescribe it in water. It possesses remarkable solvent power over urates. Given internally, it is said to lessen urates and increase urea (Vogt) by promoting oxidation and tissue change. It is somewhat slower in action than colchicin, but many recent writers have spoken in its favor (Schweininger, Blanc,[1] and others). Biesenthal found piperazin would prevent in animals the development of gout which may be produced artificially by chromic-acid injections. Piperazin may also be applied locally by means of lint dipped in a 2 per cent. aqueous solution. R. W. Wilcox[2] has found the internal use of piperazin much enhanced by the addition of phenocoll.

Lysidin is a new alkaloid of similar solvent and neutralizing power with piperazin, but said to be five times more powerful in dissolving uric-acid deposits. It is a hygroscopic reddish-white soluble powder, and the dose should be from 1 to 5 grains in twenty-four hours. It is recommended by E. Grawitz[3] of Berlin. Lycetol is a similar preparation, being a derivative of piperazin (Wittzach[4]).

Intestinal antiseptics may be required if there is much indigestion, though, as a rule, the purgation and the use of colchicum, which stimulates the liver and gives green stools, accomplish the desired result. Such remedies as resorcin, creasote, or salol may be prescribed in these cases. Sulphur has been highly recommended by Ewart. With gastralgia and in myalgic and neuralgic types guaiacum has proved of service, as well as the common coal-tar products, phenacetine and antipyrine.

Gouty patients are apt to be nervous and restless. It is irksome for them to keep quiet, and yet the least jar or movement of the affected joint causes agony. The constant severe pain makes the temper irritable, so much so that a "fit of the gout" is a popular synonym for irascibility. It is therefore sometimes necessary to give a hypodermic injection of morphine—gr. $\frac{1}{8}$ to $\frac{1}{4}$—but as in all cases of recurrent disease, as well as for the special reason stated above (page 1014), this is to be avoided if possible, and the patient should not be allowed to know what is given him, much less to take opium in any form himself. The restlessness may be relieved by codeine—gr. $\frac{1}{2}$ t. i. d.—and by moral support and encouragement, the patient being assured that his sufferings will soon yield to treatment.

(4) *Dietetic and Hygienic Treatment.*—Throughout the stage of acute inflammation it is necessary to keep the patient upon a very light diet, the basis of which should be chicken, mutton, or beef broth, farinaceous gruels, such as barley or arrowroot, and equal parts of milk and Vichy, or junket, or whey. Milk toast, toasted crackers, and soft-cooked eggs may be allowed. As beverages, weak tea or coffee and koumyss may be taken, but it is best to withhold all alcoholic drinks. It is most desirable for the patient to drink abundantly of fluids, and as the fluid diet above mentioned is not in itself sufficient, a daily allowance of 3 or 4 tumblerfuls of water or of some ærated water, such as carbonic water or Apollinaris, must be ordered besides.

[1] *Rev. de Therapeut. Med.-chirurg.*, Paris, Nov. 3, 1894.
[2] *Med. News*, Nov. 27, 1897.
[3] *Deut. med. Woch.*, 1894, vol. xx. p. 786.
[4] *Therap. Monatschrift*, 1895, p. 387.

As the acute symptoms subside more latitude may be permitted, and a little broiled fresh fish, bread, milk, rice-pudding, blanc-mange, or baked custard may be eaten. Afterward, chicken, a chop or a bit of tender steak and baked potato are in order, but the patient should not eat heartily until again able to take outdoor exercise. An exclusive meat diet is not at all desirable; in fact, some authorities like Alexander Haig[1] recommend vegetarianism on the ground that meat introduces uric acid into the body. Haig's reasoning is based in great part on experiments made upon himself while a victim to the malady. On the contrary, William H. Draper of New York has taught for many years that gouty patients do best when given a fair proportion of proteid food judiciously combined with carbohydrates, and this view is representative of the experience of others who have seen most of the disease as it is encountered in this country.

That diet is therefore best which is not too monotonous, and from which all saccharine food and alcohol are excluded. The eating of fats should also be restricted. Elderly patients do not bear well a too sudden and radical change in their diet. Farinaceous food if not too much sweetened is usually well borne, as are fresh green vegetables. Authorities differ in recommending fruits. Sir Andrew Clarke prohibited them. Usually stewed or baked apples are permissible, and a little sodium bicarbonate may be added in lieu of sugar in cooking any fruit which is too acid.

When the patient is sufficiently recovered he should take daily morning cold baths with friction, and robust subjects are benefitted by an occasional Russian bath. Energetic exercise by walking, riding, or driving should be prescribed, and the patient should live as much as possible an outdoor life, for lack of exercise combined with overeating is most apt to bring a repetition of the attack. If digestive tonics are needed, strychnine or nux vomica with compound tincture of cinchona are among the best.

CHRONIC GOUT.

ETIOLOGY.—The general etiology of gout has been discussed upon page 994.

Chronic gout develops in one of two ways—(*a*) as a result of repeated acute attacks; (*b*) insidiously.

(*a*) When the result of repeated acute attacks, the fits appear with increasing frequency, but the pain is less, the swelling and œdema are more extensive and persistent, often lasting for weeks or months, and the redness gives place to pallor.

(*b*) When insidious, the patient may acquire considerable local deformity of the joints, without ever having an acute attack. Such cases are noted particularly among the aged and those in whom the disease is hereditary.

SYMPTOMS.—The symptoms of chronic gout vary much with the constitution of the patient. The fairly robust may sustain repeated attacks of acute gout for twenty or thirty years without ever developing chronic gout, or if the disease becomes chronic it remains comparatively

[1] *Brit. Med. Journ.*, Oct. 3, 1896.

mild, being largely confined to certain joints without secondary visceral disturbances. In others with less power of resistance the disease progresses rapidly, and within a year or two they may become anæmic and debilitated and remain permanently invalided until death from exhaustion, toxæmia, or visceral complications overtakes them.

Local Symptoms.—In a typical case the progress of the malady is as follows: If the disease has begun in one great toe, other joints will soon become involved. These may be other joints of the same foot or of the opposite foot, the joints of the ankle and knees, the terminal phalanges of the fingers, and sometimes the shoulders or elbows. Even the spinal articulations have been known to be affected. Many joints may be simultaneously involved or several will be so, while others previously affected may remain quiescent. Whichever joints are involved, the pathological process and sequence is practically the same in all. The disease often progresses from the smaller to the larger joints, and the several joints may present lesions of many different degrees of pathological evolution. Thus one joint may be found presenting all the appearances of an acute attack (page 1007), another will exhibit simply swelling and œdema, another large recent concretions and tophi, while another may show advanced deformity with softening and sloughing over the concretions.

The joints, less painful than in the acute attack, may be often moved freely, and in advanced cases, especially in elderly women, distinct crepitation will be obtained. A cracking sound may be heard at a little distance as the fingers are manipulated. The toes and fingers are much distorted, being pressed aside by accumulating concretions or permanently bent by yielding to least resistance. The finger-joints may be alternately flexed and extended. Usually the swelling is chiefly about the articulations, and is more diffuse than in the acute form, but sometimes a whole finger will be uniformly thickened and stiff. The thumb-joints are singularly exempt. Tophi accumulate at the sides of the joints and in neighboring fibrous tissues. They also appear in remote situations, as the lobe of the ear, where it is very common to find imbedded two or three or more small white or yellow masses the size of small shot or peas. Tophi are also found along the sides of the fingers, over tendon sheaths, on the nose, in the palms of the hands, and on the forearms. The tophi even about the joints are often quite movable, and being usually painless they can be picked up between the fingers. The skin over them is movable, thin, and shining, but is not reddened, neither does it desquamate, as in acute gout. The smaller tophi may feel smooth, but the larger ones are often very rough, and may appear firmly imbedded and hard, like osteophytes. As the case progresses the deposits in and about a joint may fuse, become exceedingly large, destroying all the joint structure, and finally find their way through the skin.

There is an old joke about a gouty man being able to chalk a billiard cue with his knuckles, and in some cases this is literally true. I have seen a subject of hereditary gout with a concretion as large as a lemon, starting from the great toe-joint and involving much of the dorsum of the foot and three metacarpo-phalangeal articulations of the lesser toes. The mass eroded its way through the skin, partly softened, and so far impeded locomotion that the patient finally had it successfully removed

by surgical operation. When such masses soften, fluctuation may be felt beneath the skin, as in an abscess, and if incised, they discharge a pultaceous, sanguineous, or creamy material with a gritty feel, and composed chiefly of granules and crystalline deposits of biurates. Entire tophi may be discharged in this way, leaving a stellate cicatrix (as in the palm), or a portion only is lost, and the remaining concretion dries and forms a focus for fresh deposit of urates. When the tophi are large the neighboring veins are often very prominent. It occasionally happens during an acute exacerbation that a tophus is absorbed, to be deposited again elsewhere (see page 1006).

Constitutional Symptoms.—The subjects of chronic gout may still show periodicity in the advance of the symptoms, but if they have acute exacerbations, they react less and less promptly and completely, and become weak, anæmic, debilitated, and cachectic. They suffer much from anorexia, constipation, and digestive disturbances, which are aggravated by their inability to exercise freely, and sooner or later they become bedridden, either from helpless deformity, failing nutrition, or asthenia. They become mentally irritable and dull and are often somnolent. The disease in its chronic form is afebrile, and the occurrence of fever either belongs to an acute exacerbation or suggests the onset of a complication.

Complications.—As might be expected in so chronic and severe a disease, the complications are numerous and serious. Some appearances which might be described under this heading are of such common occurrence as to fairly rank as symptoms, and have been so described, but it is an unimportant detail in which aspect they are regarded. The most serious of the complications are those affecting the vascular and renal systems. Gouty lesions of the heart and kidneys, or bloodvessels and kidneys very often coexist and are mutually excitant, so that it may be difficult to determine which organ was primarily affected, but it is believed that in many cases the kidneys are first involved. Arterio-sclerosis, cardiac hypertrophy, and dilatation, granular contracted kidney, chronic gastritis, chronic asthma, and bronchitis, cerebral and other thromboses, sciatica, various eye lesions and skin diseases, diabetes, obesity, cholelithiasis, gravel, vesical calculi, etc., may all occur in different cases.

Prognosis.—The prognosis for ultimate recovery is distinctly bad, but with careful living life may be prolonged for many years. Much depends upon the patient's ability to take care of himself and upon his means to secure proper care and dietetic, hygienic, and climatic treatment. Naturally in the hereditary cases the prognosis is the least favorable.

Diagnosis.—As Fagge[1] said, "If a single deposit of urate of soda can be found, it settles the question."

The only two diseases with which chronic gout may be confounded are chronic rheumatism and rheumatoid arthritis. It will save repetition to refer the differential diagnosis to consideration under those two headings (see page 988).

The majority of cases are easily diagnosed from the appearance of the joints or a study of the visceral symptoms.

Treatment.—The general indications for treatment of chronic gout

[1] *Principles and Practice of Medicine*, vol. ii. p. 805.

are to promote elimination of waste from the body by increasing the functional activity of the skin, the bowels, and the urine, as well as by regulation of the diet and exercise and other hygienic measures. If the symptoms are decided, or if the gouty deposits are upon the increase, it is well to prescribe wine of colchicum (♏x) and potassium iodide (gr. x), which on the whole prove safer and more efficient remedies than any others of the long list which have been exploited for the purpose, such as the salicylates, benzoates, guaiacum, sulphur, etc. The colchicum acts as a cholagogue and vascular sedative, and the iodide is believed to promote absorption and benefit tissue-change in general. The treatment is continued for two or three weeks. Larger doses are usually not required, especially if the bowels are kept active. For this purpose 5 or 10 grains of calomel should be given at night, two or three times a week, followed by a dose of bitter water, Rochelle salts, or a seidlitz powder in the morning. In other cases it is preferable to give Carlsbad salts ʒj three or four times a day.

Diuretics.—The best diuretic for these cases is water, but most persons, and the gouty especially, drink less than they need, and it is difficult to have one's directions obeyed in this matter. Well-to-do patients will pay large sums for bottled mineral waters because they imagine a specific value, which it must be owned consists largely in the attractiveness of the label or the elaborateness of the chemist's analysis of the ingredients, and if they will not drink ordinary water, but will take the titled waters, there is usually no harm done. There are two devices to which I have long resorted to get such patients to take water which are in common use by others: one is to order a 5-grain effervescing tablet of lithium citrate as a placebo to be taken in a tumblerful of water three or four times a day; the other is to order a tumblerful of hot water before each meal and at bedtime. Lithium has been much extolled as a solvent of uric acid, and so it is in the laboratory, but in the blood it promptly forms an insoluble compound with sodium phosphate, which renders it of little or no value.

The sthenic cases with much acidity are benefited by taking potassium citrate or acetate with the bicarbonate three times daily before meals. These remedies are useful for their antacid, diuretic, and alterative effects. Sodium and magnesium carbonates are also employed with advantage.

The *local treatment* consists mainly in the judicious use of massage, either alone or combined with a stream of thermal water, as applied at Aix, for example, passive movements, hot-air applications (see page 990), and the application to the affected joints from time to time of iodine tincture, turpentine stupes, compound camphor liniment. A useful lotion is piperazin in 2 per cent. solution applied at night and left until morning in contact with the joint, which is wrapped in cotton and oilsilk. Recently the application of electrolysis to the concretions in the joints has seemed productive of benefit in some cases. Surgical operation is not to be thought of, except in most extreme cases like the one mentioned above (page 1017), for it is rarely successful and often productive of much harm.

Sufferers from chronic gout should live a wholly rational life, retiring early, avoiding stimulants, excessive use of tobacco, sexual excesses, and

all excitement. If alcoholic stimulants cannot be wholly dispensed with, owing to long-continued habituation to their use, or to the special need on account of weakness, the best form, and in fact the only form in which they can be safely employed, is that of much-diluted whiskey (preferably Scotch) taken with meals.

Spas.—Countless European spas have claimed specific virtue in the treatment of chronic gout. Those patients who are fairly strong or plethoric do best at the sulphate springs of Carlsbad, Marienbad, or Brides-les-Bains, and those having acid dyspepsia improve at Vichy or Vals. Kissingen and Homburg also have special reputation for the relief of the chronic gastric catarrh of these cases, and Neuenahr is particularly good. Patients suffering from gravel or stone of gouty origin may find relief in the diuretic and solvent waters of Vittel or Contréxeville. In England many cases of chronic gout are benefited at Harrowgate and Bath, and in the United States at the Hot Springs of Virginia or Arkansas, or the sulphur springs of Richfield in New York, and of Glenwood in Colorado. It is impossible within the limits of this work to further discuss the merits of all the mud baths, sand baths, and other varieties which are recommended for treatment. While not denying the local benefit to be derived in some cases from alkaline and sulphur baths, it is the belief of the writer that in most instances the improvement is mainly attributable to the systematic living, careful dieting, good air, and freedom from cares and responsibilities, which are obtained at the noted spas, rather than from any specific properties of their waters. Many of the waters in this country are as good chemically as those of foreign springs, but speaking generally, patients derive more benefit abroad because they find better system in the routine of the spas, and more diversion in the trip, besides having the added benefit of the double sea voyage.

The mode of giving the baths varies much at different health resorts. In general, a course of daily baths continued for three weeks is sufficient. The duration of each bath is from five to eight minutes, the temperature varies from 98° to 106° F., and fully half an hour's rest should follow. Massage is often given with advantage in connection with the bathing.

Patients who are much enfeebled or whose respiratory mucous membranes are especially irritable do well when possible to spend their winters in a warm and equable climate, such as that of Southern California, Egypt, or Algiers.

THE GOUTY DIATHESIS, OR "GOUTINESS."

Definition.—This diathesis, called also "irregular gout," "abarticular gout," "atypical gout," "latent gout," "the gouty vice," "constitutional gout," "masked gout," etc., represents the underlying condition of the system which is inherited by those who come of gouty stock, or which is acquired by *bon-vivants* prior to the characteristic arthritic manifestations of the disease. "Suppressed," "retrocedent," and "metastatic" gout are expressions used to denote cases in which visceral or vascular symptoms predominate, the joint symptoms for a time being held in abeyance. "Goutiness" is the term adopted by William Ewart to describe the condition under discussion in his classical

work upon "Gout and Goutiness,"[1] and his views on this point are of so much value that they are herewith quoted at length:

"Instead of a diathesis which needs 'eradicating,' we conceive of a faulty habit [in cellular activities] slowly acquired, which needs replacing by a *healthy habit*, the growth of which must also be slow. This consideration must guide our treatment; it also encourages a belief in the *curability* of *goutiness*, though not of the late results of articular gout.

"*Objectively*, we deal with two states—gout and goutiness; and these names fairly express the relations and the differences existing between them. One state may never pass into the other. More commonly goutiness inclines almost fatally toward declared gout; and, with few exceptions, those once attacked with declared gout lapse sooner or later into goutiness.

"'Goutiness' is applicable to all the conditions in which the *constitutional change is manifest*, though declared gout may be altogether absent or present only at long intervals. The evidence of goutiness may be *partly structural*, as in the delicacy of the tissues of those who inherit the infection; but in all cases it is also functional, and is made up of the gouty visceral manifestations.

"The *functional change*, as it affects the juices, is *abnormal acidity;* as it affects the tissues, *increased irritability* and *lowered resistance*.

"Indeed, goutiness is in varying degrees the basis of all articular gout, but most manifestly of that which is inherited."[2]

The term lithæmic diathesis, or lithæmia as it was originally called by Murchison, is used by some writers as synonymous with gouty diathesis, but this is somewhat inaccurate, as lithæmia is a condition believed by Murchison and his followers to be primarily due to functional hepatic derangement (although even this is denied by Bouchard) or other causes, and it will be separately described (see page 1027).

Furthermore, it is not absolutely proven that "lithic" acid or uric acid is really the cause of lithiasis, whereas it is known to be one of the chief agents in producing gout. The gouty diathesis may therefore be defined as the antecedent condition of defective proteid metabolism which leads to gout in either its acute or chronic form. Persons who inherit this peculiar diathesis may, by good fortune or good care, live to a ripe age without ever being the victims of true gout, or they may die of the complications which the diathesis begets before arthritic symptoms have had time to develop.

The condition of latent gout is declared by W. H. Draper and F. C. Shattuck to be of much more frequent occurrence in women than in men.

Severe and sudden attacks of acute gout are now far less prevalent even in its home in England than a generation ago, and they seem to be replaced by an increase in the number of cases of goutiness (Ewart[3]). This fact was observed some years ago by Milner Fothergill.[4]

Goutiness varies greatly in type even in the same individual, who may exhibit alternation of symptoms, now manifest chiefly in the respiratory, now in the vascular, or now in the digestive, system.

[1] *Gout and Goutiness*, London, 1896.
[2] *Loc. cit.*, pp. 310–312, 314.
[3] *Loc. cit.*, p. 21.
[4] *Gout in its Protean Forms.*

Symptoms.—*Vascular System.*—One of the earliest, most important, and characteristic symptoms of the gouty diathesis is arterio-sclerosis, manifested by perceptible thickening of the arterial walls and a uniformly high arterial tension. Atheromatous deposits may form at the root of the aorta or upon the aortic valve cusps, in the latter case sometimes giving rise to a bruit. As a further serious outcome of this condition aneurysm may develop, and cerebral apoplexy is by no means rare. Gouty phlebitis is well recognized.

Secondary hypertrophy of the left ventricle of the heart is produced by the arterio-sclerosis, and the latter process, by invading the coronary arteries, may interfere with the nutrition of the heart and lead to a myocarditis. Pericarditis is not common.

"Gout of the heart," as it is popularly termed, is a phrase used to describe any gouty cardiac complication, but particularly such conjoined symptoms as sudden attacks of præcordial pain, dyspnœa, syncope, vertigo, cyanosis, and palpitation. The heart sounds become distant and the pulse is very feeble.

In a patient under my observation at the present time repeated attacks of vertigo have characterized the gouty diathesis for more than three years.

More or less anæmia and pallor are present in some patients, while others are distinctly plethoric.

Respiratory System.—Three varieties of pulmonary disease are noted in the gouty diathesis: (*a*) asthma; (*b*) emphysema; (*c*) repeated obstinate attacks of bronchitis. Elderly persons who have long been "gouty" are exceedingly apt to suffer from emphysema with frequent asthmatic seizures.

Sometimes asthmatic attacks are the first and only manifestations of the gouty vice, and may so remain for years before the development of true gout. In other cases the youth of gouty heritage shows marked irritability of the mucous membranes, and suffers perhaps from gastro-intestinal catarrhs, but particularly from severe and protracted attacks of bronchial catarrh, which yield less easily than usual to treatment. In elderly subjects the bronchitis may become chronic. Uric-acid crystals have been found in the sputum by J. W. Moore and Grube.

Pleurisy, though less common, is sometimes observed. Epistaxis occasionally arises. Gouty subjects take cold easily, and attacks of coryza, laryngitis, or chronic granular pharyngitis are often seen.

Genito-urinary System.—Patients having the gouty diathesis tend to the early development of nephritis. The urine may be at first high colored, of high specific gravity, loaded with urates and uric and oxalic acids, and somewhat lessened in quantity, but later it becomes more abundant, pale, and contains little organic matter. Rendu insists upon the persistent hyperacidity of gouty urine. Albumin may be present in moderate quantity together with hyaline and granular casts. The subjects of gouty nephritis are apt to have œdema, general anasarca, much dyspnœa, and pulmonary œdema, and they may die in uræmia.

Others have "showers" of "gravel" or uric-acid sediment in the urine, and renal and vesical calculi, though less common than might be supposed, are sometimes present. Attacks of frequent painful micturition, hæmaturia, and even non-infectious urethritis have been reported.

Oxaluria and glycosuria may appear.

The testicles have been exceptionally the seat of gouty inflammation. Urethritis is not rare, which Ebstein refers to prostatorrhœa. Disorders of menstruation are common, such as menorrhagia, as well as various uterine and ovarian ailments, which have been ascribed to goutiness by Mabboux of Paris, Rendu, and others.

The Nervous System.—Gouty headaches are well known, hæmicrania and migraine being common forms. They occur through a variety of causes. They may be due to the frequent attacks of indigestion, or to the high arterial tension. A patient who was a great sufferer from them for years I was able to relieve only by the constant employment of nitroglycerine to reduce the extremely high tension of a strongly hereditary gouty diathesis.

Neuralgias and vague shooting pains appear in all parts of the body. Sciatica is common. Darting pains in various parts of the body and severe cramps in the legs, soreness of muscles, lumbago, dysæsthesia or paræsthesia, and stiffness of joints or bursitis may be complained of by middle-aged patients. The heels are often sore. An irascible temper is characteristic, but it belongs more particularly to the acute form of gout or to advanced degrees of chronic gout. Patients may suffer from the gouty diathesis or even from chronic gout through long lives and yet retain their full intellectual vigor until the end. In few cases comparatively do they become dull, though often churlish. The occurrence of apoplexy has been referred to as not unexpected (page 1022), and basilar meningitis is of very rare development. Ewart refers to exceptional forms of encephalopathy; hemiplegia, aphasia, and convulsions may be of gouty origin, and cerebral thrombosis may occur.

The condition of neurasthenia, so prevalent in parts of this country at the present time, especially in crowded cities, where the bustle and excitement of modern life readily overtaxes the nervous system, sometimes, no doubt, is of gouty origin. R. W. Wilcox[1] expresses the opinion that many neurasthenic cases are cases of neurotic lithæmia.

The *digestive system* in the gouty is apt to be deranged, although some patients never suffer in this way. An acid dyspepsia with flatulency, heartburn, and sour eructations is rather characteristic of gout in all its forms. Curiously some patients are relieved of their dyspepsia by an intervening fit of gout. The tongue in gouty dyspepsia may be in the condition of glossitis migrans or leucoplakia. It is commonly red and thin. In a woman whom I have lately seen it was so deeply cracked, fissured, and eroded as to give rise to great pain and thirst, making it difficult to take enough food to maintain nutrition.

Peridental inflammation or gingivitis may occur. The uvula is sometimes enlarged, and acute suppurative tonsillitis (quinsy) may appear in connection with goutiness.

Severe "bilious" headaches and intense gastralgia may recur periodically for years. Chronic gastritis is not rare, and hepatic congestion with hemorrhoids is often present. Colitis is not uncommon, and some patients suffer greatly from violent intestinal colic. I have known four sons of the same family whose father died of chronic gout, and who each possessed the gouty diathesis in marked degree. One died of a

[1] *Med. News*, Nov. 27, 1897.

gouty kidney; a second was a great sufferer from severe bronchial catarrhs, and finally died of pneumonia; the third had intense bronchial catarrhs—several attacks every winter—and sudden seizures of purely intestinal colic, so severe as to momentarily deprive him of reason; and the fourth is abnormally obese. Cases of appendicitis have been ascribed to goutiness, but there is little if any foundation for belief in such etiology.

General Nutrition.—The general nutrition of those having the gouty diathesis may or may not suffer. In some persons almost robust vigor of body and mind is retained for years, while others are always ailing, are thin if not emaciated, and are anæmic and sallow, with weak, flabby muscles. A certain proportion of patients having good appetites, fair digestion, and gluttonous tastes, and being disinclined to exercise, tend, as they pass middle life, to rapidly gain in weight, and passing the limits of stoutness acquire extreme obesity (see page 1035). Their bulk adds to their inability to exercise, their weight injures their gouty joints, and they tend to acquire glycosuria, myocarditis, and other serious complications.

The Skin.—The skin, which in tophaceous gout may be smooth and possibly atrophic, is often harsh and dry with gouty diathesis, and many patients develop patches of dry scaly eczema, which come and go and are easily excited by dietetic errors and slight modifications in the general health. Suard has found urates in eczematous serum. Pruritus appears in various parts of the body, sometimes in the soles of the feet, and patients complain of sensations of burning in the feet and elsewhere, especially at night. Itching of the eyelids may be troublesome. Erythema, urticaria, acne, pityriasis, boils, carbuncles, herpes, and exfoliative dermatitis are among the other common skin lesions. An acrid irritant perspiration is often troublesome. The nails exhibit longitudinal striæ and become thin and brittle, and the hair is apt to turn gray early in life, or baldness appears.

The Eyes.—A variety of eye lesions have been described in connection with gout and the gouty diathesis. Of these iritis and glaucoma are the most common, conjunctivitis being present less often than in acute rheumatism. Other lesions noted are hemorrhagic retinitis, suppurative panophthalmitis, keratitis, and irido-choroiditis. Charles Stedman Bull[1] describes a variety of "lesions of the retinal vessels, retina, and optic nerves associated with gout."

Prognosis.—The prognosis of the gouty diathesis depends upon several factors. In cases of marked heredity, where both parents have been gouty, or where the disease in its severer forms has prevailed through several generations, it is distinctly bad as regards ultimate recovery. Such patients, if they pass thirty years of age, are apt to have the manifestations of goutiness become more and more frequent and pronounced, and being prone to the development of arterio-sclerosis and the small granular kidney, are apt to possess enfeebled resisting power against acute infections, the inroads of alcoholism, or of cardiac and hepatic derangements, etc. In one family of my acquaintance, both parents having been great sufferers from gout, which proved fatal before they reached fifty years of age, each of their four children possessed the gouty diath-

[1] *Med. News*, May 8, 1897.

esis to an extraordinary degree, and all died before attaining the age of thirty-five years: one in an acute attack of gout which involved the heart, two of uræmia from gouty kidney, and the other from an attack of grippe preceded by remarkably high arterial tension.

When the diathesis is acquired much will depend upon its early recognition and the ability and willingness of the patient to abandon once for all those habits of life which have been distinctly shown to beget it. By leading a strictly temperate existence in every application of the phrase, such patients may live on in comparative comfort and safety for twenty or thirty years or even more beyond middle life, but having once demonstrated their weakness in this direction, they can never allow themselves indulgences without great risk of establishing permanent pathological conditions in the kidneys, vessels, heart, or other organs, which may cost them a decade or two of life. The practical experience of large insurance companies is a good criterion of such cases, and by them the gouty are always classed as "extra hazardous" risks. No doubt many persons having exceptional vigor of constitution can tolerate the continuance of a once-established gouty diathesis, but at best premature senility is in store for them.

DIAGNOSIS.—The diagnosis of the gouty diathesis and of hidden or latent gout must be largely based upon careful inquiry into possible heredity, the habits of life of the patient, and the occurrence of previous attacks of the acute form of the disease. In all cases of severe and prolonged headache, neuralgia, asthma, repeated obstinate catarrhs of the respiratory or alimentary mucous membranes, eczema, arterio-sclerosis, or "gravel," which are not to be accounted for upon other grounds, the possibility of a gouty element should be carefully investigated along the lines already suggested (see Diagnosis of Acute Gout, page 1011).

The diagnosis of the true gouty diathesis from lithæmia will be considered under the latter heading (page 1029).

TREATMENT.—The treatment of goutiness must be largely hygienic and dietetic. Patients in fairly good health are much better off if they let medicines severely alone, and especially the numerous "tonics" and cure-alls which their friends usually press upon them.

Exercise, systematic and vigorous but not fatiguing, should be studiously practised, and definite rules should be laid down as to its form and duration adapted to each case. Boxing, rowing, bicycling, horseback riding, golf playing, etc. are all beneficial at times, the three former for younger subjects, the two latter especially for those past middle age. It should be observed that more good is often got from five or ten minutes of brisk walking or other exercise in the open air than by an hour or two of idle sauntering. Patients unable to take active exercise should spend as much time as possible in the open air by driving, or sitting on a veranda. It is especially desirable to avoid taking cold after exercise. Patients should dry perspiration carefully, wear woollen underclothing, and avoid exposure and fatigue of all kinds. Keeping the skin active by daily cool bathing and vigorous friction, and the bowels active by medicine, massage, dieting, etc., are further undoubted preventives of "taking cold."

Medicated baths, or preferably the natural mineral spring baths, prove of much service when the treatment is not overdone. This sub-

ject has been detailed by the writer under the Treatment of Chronic Gout, page 1018, and the rules for goutiness should be the same. Often great benefit is derived from a fortnight's sea-bathing in summer.

Diet.—Much can be done for goutiness by attention to diet. The patient should be warned on the one hand against overeating, and on the other against following for too long time a diet which, like the "meat and hot water" treatment, is so impoverishing as to dangerously reduce his strength. Usually three meals suffice, and these should be taken at regular hours, without intervening lunches, and patients must not overeat. Meat of any kind (excepting pork), game, fowl, or fresh fish may be eaten once or twice a day. Eggs, lean bacon or ham, oysters, sweetbreads, may be taken at other meals. Oatmeal, cornmeal, hominy, wheatena, or cracked wheat, stale bread, and crackers constitute the most desirable cereal foods. For vegetables the patient may eat baked or mashed white potatoes, fresh pease, string beans, young green corn, spinach, cauliflower, celery, tomatoes, onions, and such fruits as apples, oranges, or grapes.

Cream cheese or good American cheese furnishes additional proteid, like the legumes and coarser cereals, and makes it unnecessary to eat very largely of meat. Soups and broths are allowed, also butter in moderation, tea and coffee, and desserts may consist of custards, gelatine-preparations, such as blanc-mange, wine, jelly, etc.

To be avoided are rich foods of all sorts, pastry, cake, sweets, griddle cakes, pickles, vinegar, spices, curry, fried foods, sausages, etc.

Alkaline mineral waters, such as Vichy or plain water should be drunk freely before meals and at bedtime. Alcohol is usually unnecessary. A good claret or dilute whiskey and water are the only forms in which it is permissible when needed. Malt liquors of all sorts are forbidden.

(The reader should also consult the section upon the Diet in Lithæmia, page 1030.)

When anæmia is present in neurasthenic cases or others in which nervous symptoms predominate, iron, Fowler's solution, and cod-liver oil are all beneficial. Tincture of nux vomica (♏v) with a drachm of some simple bitter, such as tincture of gentian or compound tincture of cinchona, should be given before meals in cases of feeble digestion. Many patients, especially those who suffer from gastric catarrh, constipation, and high vascular tension, are benefited by Carlsbad salts, or by taking ʒj of sodium sulphate in half a tumblerful of hot water two or three times a day (or in smaller dose if this proves too laxative), an hour or so before meals. Continued or violent catharsis is alike unnecessary and undesirable, but a daily free evacuation of the bowels should be insisted upon, and an occasional mercurial cathartic may be needed for plethoric cases. If arterial tension remains persistently high, an effort should be made to reduce it by nitroglycerine, gr. $\frac{1}{100}$ every three hours, or 5 grains of chloral hydrate three or four times a day. I have had patients continue the use of nitroglycerine for two or three years almost continuously with advantage, their symptoms, such as high tension, headache, nausea, and diminished urine, recurring whenever it was temporarily omitted.

LITHÆMIA.

By W. GILMAN THOMPSON, M. D.

Synonyms and Definition.—"Lithæmia" is a somewhat vague phrase. The derivation of the word implies the presence of lithic (*i. e.* uric) acid in the blood in excess. Lithuria, or excess of lithic acid in the urine, is sometimes used synonymously, and so is the phrase lithic acid diathesis, by which is meant a morbid tendency toward the accumulation of excessive uric acid in the blood and secretions of the body, thereby developing a peculiar complex of symptoms. Uricacidæmia and uricæmia are also employed as synonyms. Of late years the word lithæmia has been made to include a great variety of conditions from neurasthenia, to biliousness, renal calculi, and gout, and there is some doubt in the mind of the writer as to the desirability of retaining the phrase and insisting upon a special lithæmic diathesis.[1] Many of the nervous and other symptoms heretofore attributed to excessive accumulation of uric acid in the system have been lately shown to occur quite independently of it, and on the other hand, such accumulation may form aside from the production of typical symptoms.

Etiology.—Whatever view one may take of the pathology of lithæmia, any one who sees much of a certain class of patients, principally living in our larger cities of the North and West, who are overworked or who live in a whirl of social excitement, and who eat too much food, drink too little fluid, and exercise little or none, must admit that these causes tend to produce a fairly typical group of symptoms different from biliousness, and which, for lack of a better name, are called lithæmia. I have come to regard mental strain, worry, and anxiety as altogether the most potent influence in producing these symptoms. When the business crisis is successfully adjusted, or the domestic difficulty is suitably overcome, or the strain of caring for illness in another is passed, how often the lithæmic symptoms disappear of their own accord, and the patient rebounds to good health again.

Excessive indulgence at the table, especially in nitrogenous food, with a disproportion of cereals and fresh vegetables, is another cause. In some persons excessive use of tobacco and abuse of alcohol seem to be excitant causes, although the condition is often observed among women to whom these statements do not apply.

Heredity is probably not without influence in many cases.

Climate, if cold, favors the development of lithæmia. Many patients are worse during the winter months. Cold, by lessening the

[1] So high an authority as Strümpell does not give the subject separate consideration in the last edition of his text-book.

cutaneous circulation and checking perspiration, seems to exert some influence.

A neurotic temperament, such as is very often met with in this country, especially among overworked or overexcited city residents, is an important factor. Such persons often resort to stimulants of various kinds to tide themselves over emergencies, and this makes matters rapidly worse.

Pathology.—Murchison, who coined the term lithæmia, applied it to a form of functional disorder of the liver whereby proteid metabolism was impaired, but his original conception of the condition has been expanded by subsequent writers to cover a far wider field. It is definitely known that lithæmia is not only often present as an antecedent of gout, but uric acid is found in excess in the blood in leucæmia, pneumonia, some varieties of nephritis, various neuroses, and other diseases, besides being greatly influenced by animal diet. Nor is it certain that the liver is primarily deranged, or that uric acid is the sole agent which produces the symptoms. Other substances may act in combination with it or independently to produce like results. Oxaluria is often associated with lithæmia and lithuria, and various other excrementitous products as well as ptomaines may be present. Da Costa, in calling attention to these facts, defines lithæmia broadly as a "morbid state where the income of nutriment is in excess of the output of waste," and in common with Savory, Shoemaker, Osler,[1] and others, attributes the origin of much of the trouble to defective oxidation. Others lay more stress upon defective elimination, but both conditions are often combined in such manner that it is difficult to determine the relative share of responsibility, or which condition was the first to operate. Again, there is the question of overproduction of uric acid, but this view has met with less favor except in the sense that the blood being supplied with excessive proteid food, more uric acid becomes apparent only because less urea is formed than would represent all the proteid food waste. Bouchard is one of the chief opponents of the original Murchison theory, and maintains that the liver is not at all responsible for the condition of lithæmia.

A large part of the pathology of lithæmia is related to the etiology of gout and goutiness (in fact, lithæmia has been called "American gout"), and under those headings the writer has already fully discussed the prevalent theories of uric-acid formation, deposition, and elimination. (See Pathogenesis of Gout, pages 999–1003; Uric Acid Theory, page 999; Urine in Acute Gout, pages 1009, 1010; Uric Acid, page 1010; Symptoms of Goutiness, pages 1022–1024.) It is not intended to imply that the uric-acid and gouty diatheses are identical, as will be appreciated by referring to the above sections, for, as William Ewart[2] writes, "The two diatheses, though they largely overlap, cannot be fused into one. They are both distinct and well constituted diatheses." Perhaps the best proof of this is the fact that in some diseases as, for example, leucæmia, a considerable excess of uric acid may be constantly present without producing a single symptom of gout, or, for that matter, of the lithæmic state.

Pathological Anatomy.—In addition to the lessened alkalinity

[1] *Practice of Medicine*, p. 738. [2] *Gout and Goutiness*, p. 9.

of the blood and the possible increase of uric acid therein, the characteristic changes developed by prolonged lithæmia are scleroses (renal, vascular, and hepatic) and localized inflammations, chiefly in the serous membranes—the synovia, pleura, etc.

It is believed that the uric acid or some toxin irritates the capillaries, causing their contraction, thereby raising arterial tension, and by this twofold toxic and mechanical irritation of the arterioles arterio-sclerosis is developed. This in turn leads to cardiac hypertrophy and to further sclerotic changes in the kidney (chronic interstitial nephritis of sclerotic type) and vascular cirrhosis of the liver (Strümpell).[1]

SYMPTOMS.—The chief symptoms are exemplified in the nervous and digestive systems. In addition, the circulation, skin, and genito-urinary system may be more or less affected.

Many vague nervous symptoms are complained of, such as more or less vertigo, tinnitus aurium, hebetude, insomnia, restlessness, burning sensations in the palms or soles, flushing of the face, hemicrania, or diffuse frontal or basilar headache, darting pains in the extremities or elsewhere. Hypochondriasis is very common.

There is more or less gastro-intestinal disorder. The appetite is absent or capricious, the tongue is coated, or sometimes red and dry. Thirst and a metallic taste in the mouth are complained of. Delayed gastric digestion causes pyrosis, hiccough, sensations of weight and oppression in the epigastrium, and sometimes nausea or vomiting. There may be genuine gastralgia. Flatulence is annoying, and the bowels are constipated, or irregular dark-colored offensive frothy stools are passed. There may be hepatic tenderness on pressure. Hemorrhoids are often present. Palpitation from flatulence occurs either an hour or two after meals or at irregular times. A patient now under observation has been for some weeks regularly awakened by it at night. The arterial tension is usually increased. Varicose veins are sometimes developed.

Pruritus, eczema, urticaria, and lichen are quite common.

The urine of lithæmia may be normal, but it is more often somewhat typical of the perverted proteid metabolism, when it has the following characteristics: great acidity, high specific gravity (1028–1032), a dark, dull-red color, a tendency to deposit on cooling a heavy, "brick-dust" sediment composed of uric-acid crystals, but largely of acid sodium quadriurates. Temporary albuminuria is occasionally present. The passage of urine may cause burning pain in the urethra.

Anders emphasizes "the broad clinical fact that the urethral and genital mucous membranes often become inflamed on slight provocation, producing urethritis, cystitis, orchitis, epididymitis, vaginitis, endometritis. These conditions resist treatment obstinately."[2]

Other patients present the clinical picture of neurasthenia (see pages 556, 561). They become anæmic and suffer much from languor and muscular and mental fatigue.

DIAGNOSIS.—The diagnosis can usually be made upon discovering any considerable number of the above-described symptoms. The main difficulty lies in determining whether or not cases are of gouty origin. Careful inquiry should be made in regard to possible gouty inheritance,

[1] *Text-book of Medicine*, p. 987.

[2] *Practice of Medicine*, 1897, p. 401.

and the patient should be examined for tophi or distorted joints. A history of an acute attack of gout with localized symptoms may be obtainable. It must be admitted that many cases of severity so closely resemble goutiness, or so merge into that condition, that diagnosis is impossible (see Symptoms of Goutiness, page 1022).

"*Biliousness*" is accompanied by more pronounced gastro-intestinal disturbance, or by a symptom climax ending in bilious vomiting. In this condition the stools are light-colored, the urine often shows traces of bile pigment, the liver is often somewhat enlarged and tender, and a slight jaundice or a muddy complexion is common. None of these symptoms are typical of lithæmia.

Prognosis.—The prognosis is good, provided the patient is able to change his habits of life and is willing to carry out the treatment. Otherwise, more grave conditions may follow after a few years, such as arteriosclerosis or chronic interstitial nephritis.

Treatment is usually satisfactory, although some cases prove very obstinate. The best results are obtainable from diet and other hygiene.

Diet.—When the lithæmic symptoms become acute, it is necessary to simplify the diet by reducing the total quantity of food eaten, and especially that of meat. I am accustomed, for a few days at least, to withhold meat altogether, putting the patient upon a diet of cereals (oatmeal, hominy, wheatena, etc., macaroni, rice, and bread), fresh green vegetables (string beans, celery, asparagus, spinach, lettuce, etc.), and fruits (oranges, apples, etc.). Later, meat is allowed once a day, eggs, crisp bacon, fresh fish, shellfish, sweetbread, etc. being substituted for it at other meals. It is neither necessary nor desirable to enforce strict and continued vegetarianism. Many lithæmic patients are anæmic or neurasthenic and need meat in some form, but a large number of them bring on the condition originally by eating more nitrogenous food than they need or can digest, and then they drink so little fluid and take so little exercise that they fail to eliminate the waste, and a drachm or so of proteid residue accumulated each day, in a few weeks makes a very considerable bulk of irritant material in the system. Large quantities of plain water or of aerated waters must be taken each day, preferably on first rising, an hour before meals, and upon retiring. Six or eight tumblerfuls may be taken daily with advantage, but the patient will usually require some urging to do this, and it should be explained to him that his system needs a good "flushing." Under this simple treatment of reducing proteid income and increasing the output of waste—for more water in the urine usually means more solid matter as well—the patient promptly improves, arterial tension becomes reduced, the headache and neuralgic pains abate, and digestion becomes natural.

Eating of sugars and sweets of every kind must be temporarily forbidden, and cheese, fats, cream, butter, etc. must be taken sparingly. This is the practice at Carlsbad also, and it materially lessens the work of the liver, besides reducing the acid (lactic, butyric, etc.) fermentation in the stomach.

As a guide to dietetic measures, it is well to test with litmus the acidity of the urine when voided, and to aim at rendering it temporarily neutral or even slightly alkaline. This can be done by the adaptation of food and drink above suggested. Animal food, except milk, has a tendency to make the urine acid, whereas milk and vegetable food favor

alkalinity. In reference to the lithic acid diathesis in children, Fothergill wrote that "lean meat, raw meat minced, and beef tea are so much poison." Peptonizing such foods makes matters even worse, for the dyspepsia of lithæmia is in part a conservative process, checking digestion and absorption, whereas predigested foods are much more promptly conveyed to the overburdened liver or overloaded blood. It is not meant to imply that a meat diet is the sole cause of lithæmia, for the races of men (Eskimos, some eastern tribes, and others) who live upon meat exclusively do not suffer from this condition, and in some patients fermenting sweets will be equally harmful; but it certainly makes worse an acute attack. Some lithæmic patients can never tolerate much meat in hot weather, although they can do so at other seasons.

In severe cases, with much digestive, circulatory, or nervous disturbance, it is sometimes best to prescribe a diet of two or two and a half quarts of milk daily for two or three days, with nothing else except water. It is a good rule to forbid *in toto* all "made dishes," twice-cooked meats, highly-seasoned foods, fried foods, sauces, rich gravies, pickles, and condiments. Coffee and tea should be taken in great moderation only, and quite dilute. Between the more acute attacks it is undesirable to restrict the dietary too much, or patients will lose appetite and strength, and unintelligent cooking in many cases does far more harm than the nature of the food itself.

An exception to the use of fats is to be made in the case of strumous or rachitic children after three or five years of age. They often need butter, cream, cod-liver oil, etc., and farinaceous porridge or pudding, but should not be tempted with sweets.

Alcohol is not needed. For a further consideration of the subjects of beverages, the treatment at spas, and general hygienic treatment the reader is referred to the article upon Goutiness, page 1025. Habitual smokers do well to stop the use of tobacco entirely until they recover.

Hygienic measures consist in the prescription of abundant exercise, out-door life, sea-bathing or baths at the alkaline spas for robust patients. The more anæmic, or those suffering from neurasthenia, may require a period of rest in bed at the beginning of treatment, with a milk diet, massage, and strict mental as well as physical rest. Later, a change of climate and scene is usually curative. A sea voyage is excellent, or a brief residence among the mountains. The nature of the climate is often of less importance than the fact that the change diverts the patient's mind from cares and worries.

Medicinal treatment is usually of minor importance, and is chiefly confined to diuretics and laxatives. Sodium phosphate and salicylic acid are highly recommended for their respective action upon the liver and in promoting the excretion of urea (Haig), but the much vaunted lithium preparations, "lithia waters," etc. are useless except in so far as they make the patient take fluid. Podophyllin, cascara and calomel are all occasionally of value.

Temporary relief from the headache and neuralgias is often obtained by phenacetine. An excellent combination is phenacetine gr. iij, salol and caffeine *āā* gr. ij, to be given in capsules three or four times a day. Nux vomica is always a good tonic, and if the nutrition is poor, gr. $\frac{1}{20}$ of arsenious acid with gr. $\frac{1}{30}$ of strychnine may be given t. i. d.

OBESITY.

By W. GILMAN THOMPSON, M. D.

Synonyms.—Polysarcia; Corpulencia morbosa; Lipomatosis universalis.

Definition.—Obesity, called also corpulency or abnormal stoutness, is a condition characterized by excessive deposit of fat in many parts of the body, with consequent enormous increase in body-weight. It is regarded primarily as a form of disordered nutrition caused by the excessive consumption of food in general or the disproportional consumption of certain classes of foods, which is not counterbalanced by complete oxidation and elimination. Obesity may be either inherited or acquired.

It is impossible to set a limit by weight at which ordinary stoutness merges into abnormal obesity, as much depends upon the height of the individual, the relative development of the bones and muscles, etc. For an average man of a height of 5 feet 8 inches, a weight of 165 pounds may be considered normal, and 30 or 40 or even 50 additional pounds may be carried without inconvenience by much taller men. A weight above this—*i. e.* a total of 200 pounds and more might well constitute obesity, although for a man of large frame and of height above 6 feet, 225 pounds may not be fairly classed as obesity. A patient of mine who is 6 feet 6 inches tall carries a weight of 235 pounds easily, without the slightest appearance of obesity. A weight of 300 pounds is almost always a grave condition of obesity (although I know a man weighing 336 pounds who is quite agile and even dances well), and one of 400 pounds or more usually renders the individual almost helpless and beyond the possibility of cure. The famous Daniel Lambert, at twenty-three years of age, weighed 448 pounds, and eventually attained, it is said, to a weight of 739 pounds (Duckworth). A youth is at present being exhibited in New York who weighs 712 pounds and has a girth of 92 inches.

Since the earliest historic times obesity has been associated in the vulgar mind with plenty, and with ideas of self-indulgence, as exemplified in mythology, in the images of Bacchus and of many oriental gods, both male and female. Egyptain women cultivate corpulency, and among certain savage tribes obesity is regarded as a most desirable attainment, which they seek by special modes of feeding, and its possession in exaggerated form is looked upon with reverence. Among the Hottentots, for example, the excessive development of fat in certain parts of the body, as the buttocks and breasts, is held in special admiration.

Etiology.—*Sex.*—Obesity may affect either sex, but is more common in women than in men—in nearly the proportion of ten to one. In women it occurs principally after the menopause, and is much dreaded

by them as they approach the climacteric. It may, however, appear much earlier.

Age.—Obesity, when inherited, may develop earlier than the acquired form, and obesity in childhood or youth must be regarded as hereditary. The children of obese parents may become quite stout soon after their second dentition, and so remain until the period of puberty, when they may become excessively corpulent, although their bodies are not unwieldy. Examples of early development of hereditary obesity will be cited on page 1035.

Acquired obesity does not always become marked in either sex until the fifth decade of life. At this time, as pointed out in the case of gout (page 995), the disproportion between exercise and eating is often most pronounced. Exercise is much less cultivated than in earlier years, while the appetite still remains undiminished, and increasing affluence often provides luxuries of the table and permits habits of ease which are highly conducive to corpulency.

Obesity is not common in old age. The tendencies which lead to it are usually operative before sixty years of age, if at all; and as excessive obesity lessens the resisting powers of the body toward disease, the octogenarian is rarely corpulent, and the nonogenarian is never so.

Oertel[1] points out the curious changes which the disposition of fat undergoes with advancing years. Infants who present inherited obesity may become relatively less corpulent during early childhood, re-exhibit the tendency at puberty (especially if females), lose it again, and re-develop it after the menopause or in middle life. In the newborn the omentum, mesentery, and subcutaneous cellular tissue contain but little fat, and this remains true while the child is growing in height. In middle life, however, fat elects these situations especially, and, as old age approaches, it disappears only from the skin, remaining much longer in the common sites of internal accumulation (see page 1040).

Climate possesses doubtful influence upon the development of obesity if the many other factors are eliminated. Europeans living in hot climates, such as that of India, which encourage inertia, may, if well fed, become stouter, yet the Hindoos are much more prone to obesity than Mohammedans living in the same climate. In colder climates exercise is more indulged in, and oxidation is believed to be more active, and obesity is less easily developed, although a conspicuous adiposity is not incompatible with extreme cold. Dr. Frederick Cook of the Peary Arctic expedition told me that the most northern of all Eskimos have an exceedingly well-developed subcutaneous adipose layer. This he noticed particularly upon cutting through the skin in the performance of some minor surgical operations. In several Eskimos whom Lieutenant Peary lately brought to this country, four of whom I had an opportunity to examine in Bellevue Hospital, this was a very noticeable feature, all the bony outlines being very smoothly covered by integument much more uniformly fat than is usual in the Caucasian race. Some of the most northern Eskimos become almost corpulent, and yet their diet consists exclusively of fats and proteids, their only vegetable food being lichens occasionally taken from the stomach of a deer that has licked them off the rocks.

[1] *Twentieth Century Practice of Medicine*, vol. ii. p. 637.

Some importance must be attached to the fact that in hot climates there is less demand for fat combustion to maintain the body heat, and this circumstance may promote its storage in the tissues.

Season.—The season of the year has no definite effect upon obesity. Many persons gain weight in summer and lose it in winter, maintaining an average equilibrium, but with others the exact reverse occurs. As such persons pass middle life they may fail to lose in one season all that they gained in another, and in this way go on increasing their fat deposit from year to year. Many of the cases in which season apparently plays some part are really more influenced by the variations in diet, exercise, or occupation which are incident to different times of year than they are by the season itself.

Race.—It cannot be said that race exerts any very definite predisposing influence upon obesity when other factors such as general prosperity and mode of life are eliminated. Laziness, indolence, abundant food, and life in hot climates will affect the different tribes and races of man in this regard more than the influence of race itself. Obesity is in this country somewhat more common among middle-aged Hebrew women and Germans. Negroes are prone to it when well fed and not overworked, possibly because they exercise comparatively little restraint over their natural appetites.

The typical American has very little tendency to obesity (although ordinary stoutness is common enough), and the exaggerated cases of it found in this country are usually among those of foreign birth or parentage.

Heredity.—The tendency to obesity is undoubtedly transmitted, although the diseased condition may not develop until middle life. Among the obese fully 50 per cent. possess this heredity (Oertel). The inheritance in some cases may become manifest in early childhood, even in infancy, and it is somewhat more pronounced in the female sex. Oertel[1] of Munich reports the case of a girl of 7 years who weighed 124 lbs., and refers to notable cases recorded by others, as, for example, a boy of 5 years, weighing 189 lbs. (Weinberger), a Hindoo child of 10 years weighing 266 lbs. (Don); a girl of 4 years weighing 137 lbs.; a boy of 15 months weighing 35 lbs. (Barkhausen); and a new-born infant weighing over 18 lbs. (Wolf). Similar phenomena of hereditary transmission of obesity have been noted by breeders of domestic animals, especially swine. Young infants, wrongly fed upon prepared starchy foods, often become extremely stout, a condition which should not be mistaken for hereditary obesity as it disappears as they grow older and receive proper diet.

Temperament is not without influence upon obesity. The nervous, active, high-strung organization is rarely one which tends to store up fat, whereas the lazy, indolent, phlegmatic temperament distinctly favors the process.

Gout is perhaps more often than any other disease associated with obesity, and children of gouty parentage may show a decided tendency to obesity in adolescence. In such cases obesity may be the only manifestation of goutiness, or true gout may be later associated with it, the obesity usually developing first.

[1] *Twentieth Century Practice of Medicine*, vol. ii. p. 633.

Glycosuria is of very common occurrence among the obese if past forty years of age, and in such cases a considerable amount of sugar may be tolerated in the urine without the appearance of symptoms of true diabetes mellitus. Moderate glycosuria in a man past fifty years of age who is obese may not prove harmful, and some clinicians go so far as to regard its appearance as somewhat conservative in that it may alarm the patient into following more closely those dietetic principles which are alike beneficial to the two conditions. I have recently seen a patient, a man sixty years of age, who weighed 310 lbs., and who had 6 per cent. of sugar in the urine. He had the early symptoms of locomotor ataxia, tubercular consolidation of the apex of the right lung, and with occasional hæmoptysis, yet he had none of the symptoms of diabetes except the glycosuria, and complained chiefly of pulmonary hemorrhage and a slightly ataxic gait. He was fairly strong and able to work.

Many theories have been advanced to explain the coincidence of glycosuria and obesity, but as the real nature of diabetes is as yet imperfectly understood, none are satisfactory. Van Noörden believes that in cases of "diabetogenous obesity" (which others term "lipogenous") the excess of sugar present in the blood fails of elimination and is stored as fat among the tissues. Thus the diabetes must have preceded the obesity. Further discussion of this topic has been given by Coleman in the article upon Diabetes (Vol. III., p. 836).

A recent theory of Kisch is that the muscles of the obese become too overburdened by fat accumulation to carry on their normal function of glycogen consumption, and hence diabetes results.

The triple combination of gout or goutiness, diabetes (or glycosuria), and obesity is often recognized. R. Schmitz,[1] in an analysis of 600 diabetics, found 35 of this type.

Anæmia of advanced grade by reducing oxidation processes sometimes leads to obesity. Chlorotic girls are often stout, and very anæmic women who suffer much from languor and inability to take ordinary exercise may accumulate excessive fat.

Various other diseases either predispose to obesity or are oftentimes associated with it. Recovery from severe and protracted illness, such as neurasthenia, enteric fever, or pneumonia, will sometimes cause patients who were previously always thin to gain weight with such rapidity that it becomes cumbersome within six months. I have recently seen such an event follow puerperal septicæmia in a young woman of twenty-six years. A full mercurial course given in treatment of syphilis may develop obesity. The exhaustion which in some women succeeds the bearing of several children in too rapid succession often leaves them in a neurasthenic state, which after a long period of slow reaction may be followed by corpulency.

Acephalic monsters are often born obese, and cretinous dwarfs are often corpulent (see Starr's illustrations of Cretinism, Vol. III., pp. 695, 696, and Plate I.). The same holds good in many cases of myxœdema and among scrofulous and rachitic children.

Excessive anæmia and hemorrhage are sometimes precursors of obesity, whereas on the contrary in wasting diseases, such as carcinoma, fat is lost early.

[1] *Lancet*, Nov. 4, 1882.

Injuries, such as fractures which are of a more or less permanently disabling nature, so as to cut short a career of active exercise, will strongly favor the development of corpulency in one otherwise disposed to it. I have seen a naturally stout man of forty become obese after fracturing both patellæ, which accident restricted him to sedentary life. A congenital club-foot may similarly favor the early development of hereditary obesity.

Castration is sometimes a factor in producing obesity. It is well known that castrated animals of all sorts grow larger and stouter than the unoperated male. The chief illustration of this curious physiological phenomenon is found in the eunuchs of the oriental harems, who are proverbially obese, but it should be observed that they are also well fed and live very indolent lives. Removal of both ovaries from woman does not necessarily lead to obesity ; it may even prevent it by improving the general health and enabling the patient to take more exercise. Glavecke[1] states that 42 per cent. of women in whom the menopause has been artificially induced become obese. In such statistics, however, the difficulty is apparent of establishing any definite limit at which obesity begins, and a stoutness which is natural to the individual may follow any operation relieving a chronic malady, although it may never have been attained before. Excessive obesity is certainly not so common as this percentage would imply. Sterility in women consequent upon chronic but non-malignant uterine or ovarian disease is sometimes, but not always, followed by obesity as is the natural menopause, and religious male celibates, if well fed and indolent, are apt to grow corpulent.

Overeating is a far more common fault than excessive drinking, and is undoubtedly an all-important factor in producing obesity. Obesity never develops upon a starvation diet. Yet many subjects of this malady deny that they eat too much, and when the condition is once established it may be still maintained upon a somewhat frugal diet. In such cases, however, upon careful investigation it will usually be found that the common dietetic error has existed at an earlier period or else there is a strong hereditary element present. Sir Henry Thompson has emphasized the fact that whereas overeating in the first half of life may be relieved by occasional "bilious" attacks which enable the body to cast off accumulated waste, later "the unemployed material may be relegated in the form of fat to be stored on the external surface of the body, or be packed among the internal organs, and thus he or she may become corpulent and heavy."[2]

"When the growth of the body has been completed, there follows a period of approximate equilibrium, lasting through two or three decades, in which the waste of any excessive quantity of food eaten is still readily eliminated through the emunctories, kept active by exercise and work. After middle age activity is considerably diminished, while the habit of fully satisfying a vigorous appetite may be continued, resulting in the consumption of a much larger amount of food than is required for maintaining the vital functions. It is extremely difficult for persons who feel in robust health and whose mental is proportionately greater than their physical activity to realize that they not only require less food than they did ten, fifteen, or twenty years earlier in life, but that

[1] *Arch. für Gynäkol.*, xxxv. i. [2] *Diet in Relation to Age and Activity*, p. 26.

eating as they do may be productive of positive harm by overloading the excretory organs. It is for this reason that many persons accumulate excessive weight after reaching a certain advanced period of life."[1]

In conclusion, it must be emphasized that large eating does not of itself produce obesity, for many persons of spare habit eat more than the obese, and although they endeavor to gain weight by eating the so-called fat-producing foods, from some opposite perversion of nutrition, often of nervous origin, they remain lean all their lives. Moreover, there are those who accumulate fat abnormally despite the absence of any discoverable faults in either eating or drinking. Such cases are hereditary or else the patients suffer from defective processes of oxidation and elimination.

Improper Diet.—The dietetic error quite often may not consist in taking food which is excessive in aggregate bulk, but consists rather in an ill-balanced dietary in which certain classes of foods wrongly predominate. In this connection Bauer writes: "The fat stored up in the body acts in like manner with the fat contained in the food, since it likewise lessens the waste of tissue and secondarily the oxidation. Thus, we understand why abstinence can be longer borne by organisms rich in fat than by those poorly furnished with it, the former consuming less of the albumin of their organs. The stock of fat stored up in the body is, moreover, the cause why corpulent individuals frequently continue to gain in bulk, although they are not in the habit of indulging in food immoderately.

"If an increase of albumin be desired without a considerable addition to the store of fat, a liberal allowance of albumin with relatively small quantities of carbohydrates must be provided. If, on the other hand, a substantial addition to the fat appear desirable, the food must contain less albumin and more carbohydrates, with a fair proportion of fats."

Liebig first showed clearly that the carbohydrate foods, starches, and sugars are the chief fat producers in the body, rather than fat itself. Pettenkofer and Voit then demonstrated that decomposition of proteid food resulted finally in fat formation, and claimed that the action of the carbohydrates was indirect, or, in other words, carbohydrates, by their complete and ready conversion into carbon dioxide and water, spare the fat of food and the fat produced by proteid food from complete combustion and allow it to accumulate within the body. Further recent experimental evidence proves, however, that the carbohydrates are also capable of directly producing fats, and the conclusion is reached that all three classes of food—proteids, fats, and carbohydrates—are capable of forming fat, while it is an undoubted clinical fact that the carbohydrates, if allowed to predominate in any dietary, will favor corpulency. These facts are of obvious importance in directing the diet of the obese, and the modern dietetic regimens which are often most successful are based not upon the total exclusion of any one of these three primitive classes of foods, but their judicious combination in proper proportions. (See the section upon Treatment, page 1046.)

Alcohol.—The constant use of alcohol, even in moderation, favors obesity in some persons, and its excessive consumption, when it does

[1] W. Gilman Thompson: *Practical Dietetics*, 1898, p. 599.

not wholly disorder the digestive organs, or cause cirrhotic changes, is often productive of the same condition. This applies particularly to the fermented malt liquors, heavy beers, porter, ale, and stout. Malsters and beer brewers are proverbially stout. Rich sugar-laden wines, port, sherry, madeira, etc., have the same tendency if habitually drunk, and in some persons whiskey-drinking predisposes to obesity. Alcohol alone may not have this effect, but its combination with overeating in the *bon-viveur* is particularly disastrous; and the various malt liquors drunk to excess add so much dextrine, maltose, and other fat-forming carbohydrates with the alcohol, that even those whose occupations require much exercise are unable to completely oxidize all their ingesta.[1]

Deficient Oxidation and Elimination.—For a complete discussion of the problems of fat metamorphosis and deficient oxidation and elimination with consequent accumulation of food products, reference must be had to such admirable works upon physiological chemistry as those of Gamgee,[2] Bunge,[3] and Halliburton.[4] It can only be observed here, in connection with the clinical aspect of the subject, that lack of due exercise, too prolonged rest and sleep, entire freedom from cares, responsibilities, or excitements of every kind, all distinctly promote the rapid development of obesity in those in whom other conditions are favorable, such as those of diet, heredity, etc.

The lack of exercise fails to excite deep breathing and active oxidation and lessens the functional activity of all the emunctories, and the resultant accumulation of fat diminishes the desire or capability for exercise and maintains a vicious circle.

Occupation exerts some influence upon the acquirement of obesity or at least of moderate corpulency. Soldiers and sailors are usually free from it if actively employed, whereas it is notoriously common among beer brewers and malsters (who drink much beer), butchers (who live well), coachmen (if they do not groom their own horses, and living an outdoor sedentary life explains their tendency), monks (who live indolent lives and eat less meat than starchy food).

Ingestion of Fluids.—How often is the physician asked by the patient seeking relief from threatening obesity, "Will water make me fat?" Unquestionably the ingestion of large amounts of fluid, whether as soups, broths, beverages, such as dilute tea and coffee, or milk, or plain water itself, greatly favors the development of obesity. Oertel of Munich, who has especially studied this portion of the subject, attributes the baneful influence of fluids to circulatory and oxidation disturbances. It may not be that the fluid originates the habit of corpulency, for it may contain but little real nutriment, but this habit being once established and fat having begun to accumulate, the fluid tends to produce visceral congestions, dilutes the secretions and other fluids of the body, promotes the decomposition of the circulating albumin, and, as Voit and J. Munk have shown, the fat-forming elements of the food are converted into tissue fat in larger quantities.

PATHOGENESIS.—The exact mode of formation of fat within the body has been the subject of several theories. Toldt regards the fat cells as

[1] See Oertel's *Manuel der Therap. der Kreislaufstörungen.*
[2] *Physiolog. Chem. of the Animal Body.*
[3] *Physiolog. and Patholog. Chem.*
[4] *Text-book of Chem. Physiol. and Pathol.*

wholly distinct from connective tissue, having different embryological origin, and being perpetuated and increased only from pre-existing fat cells; yet he admits that under some conditions connective-tissue cells may both produce fat and store it, or be converted into it.

Virchow and Fleming, on the other hand, regard adipose and loose connective tissue as of like origin, and by them fat cells are believed to be derived directly from those connective-tissue cells which happen to receive a favorable blood supply.

Both theories agree upon the necessity of well-developed vascularity, with some distention of the vessels and retardation of the blood stream.

Cohnheim ascribes fat accumulation to lessened oxidation in the tissue cells—*i. e.* to diminished combustion activity, a condition possibly induced by impoverished red blood cells, which contain too little hæmoglobin. It is certainly true that some forms of anæmia are apt to be accompanied by corpulency, but it is equally true of such cases that with marked improvement under treatment for the anæmia, corpulency may increase.

Unna expounds a more elaborate theory, which, however, as yet lacks substantial confirmation. He regards some fat as formed in the muscles, some in the skin by action of the adipose glands, and some in the mesentery by action of blood corpuscles.

It must be admitted that up to the present time the pathogenesis of obesity is most imperfectly understood, and there is still grave doubt as to whether it has any claim at all to be regarded as a disease entity, and is not merely a morbid phenomenon, the outgrowth of many different disturbances of function and nutrition.

It is generally believed that fat deposits, however dense, are constantly being decomposed and reformed—a fact which lends some encouragement to treatment.

Pathological Anatomy.—There is much greater variation in the proportion of fat in the healthy body than in that of proteid substance; thus, according to Moleschott, while the latter averages 15 to 16 per cent. of the total body weight, the former may vary between 9 and 23 per cent. within physiological limits. Women are apt to have relatively more fat than men in health by about 2.5 per cent. The conditions which effect these fluctuations in fat storage in health are mainly age, sex, the quality and quantity of food eaten, the amount of sleep and exercise indulged in, season and climate, heredity, occupation, race and individual peculiarities. It is largely modifications in one or more of these conditions that will determine an increase of fat storage beyond all ordinary limits.

The situations in which fat is found in obesity correspond with those in which normally more or less fat is present, and the fat of obesity does not exceed these limitations. If Toldt's theory be correct of the origin of fat from previous embryonal cells, perpetuating their kind through adipose and not connective tissue—in the latter case occurring only as a secondary transformation—there is no reason why fat in excess should be formed away from such tissues. Fat is accordingly absent from the auricles, the eyelids, the scrotum, nymphæ, penis, clitoris, and intestinal muscle, although several of these organs, like the penis, are highly vascular.

Fat accumulates (*a*) internally in and around various organs, and (*b*) externally in the subcutaneous adipose tissue.

(*a*) Internally fat is found stored in the mediastinum, over the surface and about the base of the heart, in the mesentery, greater omentum, and appendicæ epiploicæ of the intestines, in the cancellous tissue, and especially in the yellow marrow of bones, the adipose capsule surrounding the kidneys (suet), and the intermuscular sheath of voluntary muscles.

(*b*) Externally fat is prominently stored in the malar region of the face, the neck, the breasts, abdominal wall, especially its lower portion, the pubic region, gluteal region, soles of the feet, and palms, about the shoulders, arms, and thighs, (flexures of joints), orbits.

There is a notable absence of fat within the cranium and spinal canal.

In some cases of obesity, localized deposits of fat are observed which amount to separate tumors. Such fatty accumulations have been noticed occurring above the occipital regions by Sir Dyce Duckworth and others.

Microscopically, in the plethoric forms the fat cells of the adipose tissue are large, and the excess of oil which they contain obscures their nuclei, membranes, and intermediate mucoid substance. The cells are elongated and made irregular by distention and compression. In the anæmic form the fat cells are somewhat paler, their oil globules, which are sometimes several in number, are smaller, and the nuclei and other elements are more distinct.

Several competent microscopists have found fat granules free in the blood serum and in the muscles in extreme cases, especially among the alcoholic obese. The blood in the plethoric cases may contain, according to Oertel, 106 per cent. of hæmoglobin (Fleischl's scale), and its specific gravity is also increased.

The fat within the body penetrates in advanced cases into such organs as are supplied with adipose or connective tissue. Masses of fat overlie the heart, crowd between the heart and pericardium, and in the auriculo-ventricular and coronary grooves. The heart action becomes more or less embarrassed in consequence, and fat may penetrate between the fibres of the heart muscle in the interstitial connective tissue, or the former may undergo true fatty degeneration. Hypertrophy followed by dilatation, or dilatation alone, results.

The lungs present evidences of chronic catarrh and congestion. The liver cells are overloaded with fat, and the liver itself is usually enlarged and hyperæmic. In cases of obesity which have originated through habits of gourmandizing, the stomach will be found considerably enlarged, often with a catarrhal mucosa.

The kidneys are not necessarily affected, and the urine varies much in different cases, being sometimes condensed, or the reverse in instances of polyuria, which are common in the plethoric type.

Hernial protrusions of omental fat have been known to be forced through interstices in the linea alba, between the umbilicus and ensiform cartilage. Misplacements of the viscera, such as the uterus, have resulted from the pressure upon them of the mass of fat.

SYMPTOMS.—In the early stages of obesity it is customary to distin-

guish two types, the (*a*) plethoric, and the (*b*) anæmic; but later, when excessive accumulation of fat embarrasses the organism in many ways, the two types merge more and more into a common hydræmic form, in which the original distinctions no longer obtain.

In the early history of any case of obesity the patient's symptoms arise principally from mechanical disturbances of functional activity, locomotion, etc., but in the late stage of the condition they become those of degenerative changes in the viscera, heart, liver, etc., and hydræmic blood. Fat being a poor conductor of heat, the normal heat radiation of the body is reduced.

(*a*) *Plethoric Type.*—In the plethoric form, such as is often seen in robust beer brewers, butchers, and men of similar occupations, the albuminous as well as the adipose tissues of the body are increased, and the individual feels in good health, strong, vigorous, and active. The muscles are well developed, the blood is rich in cellular elements and hæmoglobin, the heart is often slightly enlarged, its action is strong and the circulation is brisk. The appetite is good, and the patient often drinks much beer or other fluid, which in turn increases the quantity of urine and perspiration. The skin is smooth and soft, perhaps unctuous, and the color is normal, or the face is often flushed. The girth increases materially, and the fulness of the neck is prominent. The thickness of the abdominal wall in fat may attain three or four inches.

In this condition the patient may remain stationary for several years, or he may gain steadily in weight, 10, 15, or 20 pounds in a year. Feeling so well, he is often proud of his size, and regards with complacency the good-humored comments of his friends. Unless the process is checked, sooner or later the fat becomes a burden, and the patient's whole condition changes. He becomes grotesquely deformed and clumsy. The normal folds of the skin are obliterated and replaced by deeper folds of layers of subcutaneous fat. A double or triple chin appears, the back of the neck is wrinkled, and the pendulous abdomen folds over upon itself. Palpation of the abdomen can be made to excite a series of waves, which have sometimes been mistaken for ascitic fluid. On having an assistant press the edge of the hand or of a book vertically down upon the median abdominal line, the wave is cut off and does not pass to the opposite side, like the fluid wave, and with a little care its superficial character can always be demonstrated. The protruding or pendulous abdomen may make coitus an impossibility, and males having such deformity are often sterile, their semen containing but few if any spermatozoa. The omental fat is much thickened and partial circulation is obstructed.

The facial expression is changed and capable of less variation than normal. In females the breasts become greatly enlarged, elongated, and pendulous, and the creases formed beneath them and in the other folds of the surface often become reddened, excoriated, and emit an offensive odor from the difficulty of maintaining cleanliness and the formation of fatty acids from the increased and decomposing sebaceous secretion. Pruritus, erythema, eczema, and other localized skin affections appear in and about the folds of the scrotum, groin, axilla, breasts, labia, etc. Acne is often present, as are comedones and intertrigo. The abdomen continues to increase in size and becomes truly enormous, rendering physical examination of the viscera wholly impossible. The umbilicus

PLATE VII.

Excessive Obesity, Plethoric Type: Weight, 410 pounds.

is deeply sunken, and the folds of the belly hang down over the genitalia and thighs. The patient walks slowly, with a straddling gait, and all sudden movements are impossible. Walking may become altogether impossible from clumsiness, dyspnœa, or from pains in the back and legs produced from the weight of fat and the effort to maintain a changed equilibrium. A woman lately in my wards at Bellevue Hospital, who weighed over 400 pounds, seemed literally to flow over the narrow hospital cot as she lay upon it, and, lying upon her side, she so completely filled the bed that she was unable to turn without falling out, and had to be helped out of the bed and turned about when she wished a change of position. Such patients cannot breathe lying flat upon the back, for the heavy abdomen, crowding upon the fat-laden viscera, presses the diaphragm high up into the chest, dangerously impeding respiratory movements. The pulse becomes weak and slightly increased in frequency or irregular, and the heart sounds may be quite inaudible, in part from the cushion of fat which covers the chest, and in part from feebleness of the heart itself, even though it does not present the appearance of true fatty degeneration at autopsy. The hands and feet remain relatively small. True œdema of the feet and ankles is not always present, but it may be observed, and the feeble circulation augments the poor nutrition of the extremities, so that varicose veins may develop and ulcerate. Such ulcers or abrasions occurring from injury heal imperfectly, although bed-sores are not common. The patient depicted in the accompanying illustration (Plate VII.), a man of middle age, who weighed 410 lbs., died in Bellevue Hospital from septicæmia caused by infection through ulcers of the leg, which he had been unable to keep clean.

(*b*) *Anæmic Type.*—While the larger number of patients of the plethoric type are males, the majority of the anæmic type are chlorotic women. At the start they present a wholly different clinical type. They are pale, anæmic, feeble, and disinclined to exertion. The muscles are ill-developed and flabby, the superficial adipose is much less firm and elastic than in the other type, and the skin may even become wrinkled, especially as the disease advances. The surface of the abdomen often presents a large number of small nodules, of the size of a split pea or bean, which are irregular subcutaneous deposits of fat. The least exertion produces dyspnœa, and if the patient rests long in one position, leaning backward or upon the side, there is apt to be hypostatic congestion at the most dependent portion of the lungs. The pulse is feeble, small, accelerated, often dicrotic, and the heart sounds are indistinct. The appetite is often very poor, and constipation, with coated tongue and offensive breath, is common. These patients often have an aversion for meat and prefer carbohydrate foods. The temperature is nearly always subnormal, a fact which Oertel [1] attributes to the diminution in hæmoglobin and its lessened power of absorption of oxygen, as well as to the imperfect tissue metamorphosis and lessened muscular activity. The blood examination presents the ordinary picture of a secondary anæmia. While the patients of this type become excessively corpulent, they rarely reach the truly colossal proportions of the worst cases of the plethoric form, for they are more apt to die sooner from exhaustion or complications. Oertel explains very wisely that these patients have

[1] *Loc. cit.*, p. 653.

become obese, not, as their plethoric opposites, by ingestion of enormous quantities of tissue-forming food and drink, but through deficient oxidation and faulty nutrition consequent upon an anæmic and debilitated state of the system. Frequent attacks of palpitation, brought on by very slight causes, are characteristic. The heart becomes dilated as the diseased condition progresses, and if the sounds can be distinctly heard at all they are usually accompanied by anæmic bruits or those due to relaxation. The enfeebled circulation promotes an hydræmic state, arterial pressure is low, and the quantity of urine voided is small. Œdema of the feet and ankles is much more common than in the plethoric form. The anæmic obese bear attacks of intercurrent febrile disease very poorly. Not only is this due to the feeble circulation and poor nutrition, but Liebermeister showed that as the body surface becomes greatly disproportionate to the bulk, and is so much covered with fat that the heat-regulating mechanism is most imperfect, the heat of fever tends to accumulate within the body and still further weaken the heart.

For the same reason antipyretics may throw these patients into most critical collapse. In general they convalesce slowly from acute diseases, and have very little resisting power against infections, succumbing easily to attacks of pneumonia, enteric fever, tuberculosis, etc.

Among minor nutritional disturbances in connection with obesity are variations in the amount of hair, which may become very thin or absent in some situations, as about the genitals. On the other hand, in obese women hair often grows quite prominently in unwonted situations, as upon the upper lip, chin, thighs, or back, etc.

Complications.—Menstruation is frequently disturbed in obesity, as it is in other profound nutritional disorders, such as excessive anæmia, diabetes, etc. The most common condition following the sudden acquisition of corpulency is a simple omission for one or two periods of the menstrual flow, or it becomes less frequent than usual, occurring only at intervals of six or eight weeks, etc. It is often scanty and painful. If not actually sterile, such women usually become so after bearing one or two children. Early abortion is common among the obese.

Currier[1] says: "A woman under thirty years of age, who becomes obese from whatever cause, will usually suffer with amenorrhœa or oligo-menorrhœa, the menstrual flow, if present at all, being accompanied with pain, though prior to the accumulation of fat it may have been painless. If such women marry they are usually sterile." In obesity the circulation is poor, the muscles and viscera are relatively inactive in function, and the writer just quoted attributes much of the menstrual disorder to enlargement of the uterus by infiltration with fat.

The liability to excoriations of the skin and the difficulty of keeping them clean renders these patients subject to septic infection, which may become fatal, as in the case referred to on page 1043. Subjects of obesity often develop boils and carbuncles which may prove serious.

Arterio-sclerosis is of common association with obesity. The process may begin in the coronary vessels of a fat heart, or as a general endarteritis. Definite etiological relationship between these two disorders is not established, but their concurrence is often observed and is of grave

[1] *The Menopause*, p. 118, 1897.

import, for the rigid vessel walls interfere still further with nutrition and embarrass the feeble heart.

Cardiac asthma, occurring in paroxysmal night attacks, is another common complication. It usually indicates decided weakness of the heart muscle. Attacks of cardiac angina of great intensity, accompanied by palpitation and a very feeble, rapid pulse, are also present in many instances. Such attacks may appear as a result of sudden dilatation, or be apparently unprovoked. Hemorrhagic infarctions and thromboses occasionally occur. Gall stones are sometimes present with obesity.

Prognosis.—Obesity, once established, continues to increase unless promptly treated, and the benefit to be expected from treatment depends much upon the non-involvement of internal organs in the process and the remaining vigor of the patient's circulation. In general, prognosis is better for the plethoric than the anæmic cases. In many cases improvement will follow treatment up to a certain point, when the condition remains in equilibrium, which is easily disturbed by a return to former habits of life. Even though an apparent complete cure be secured in an early case, the patient is always liable to relapse. This is especially true of the hereditary cases. Very extreme cases, in which a weight of 350, 400, or more pounds is added to a body of ordinary skeletal development, have a distinctly bad prognosis. Patients may survive the attainment of such obesity for three or four or even ten years, but they are liable to die suddenly at any time from cardiac asthenia, from intercurrent diseases, or from one of the more serious complications already described (page 1044). A favorable prognosis in a given case will largely depend upon the promptness with which the treatment operates and the ability of the patient to endure reducing measures without alarming weakness. The hereditary cases are much less amenable to treatment than are the acquired. The presence of gout or advanced arterio-sclerosis with obesity renders the prognosis much more grave, but the concurrence of moderate glycosuria is not so much to be dreaded, although decided diabetes is of course bad. Oertel[1] proposes a practical test for the prognosis of advanced cases in which a feeble circulation with hydræmia and possibly œdema and serous accumulations are present. For two days the patient eats and drinks as usual, but the quantity of food eaten, the fluid drunk, and the urine excreted is all carefully measured. Then for two days the fluid ingested is restricted to 700 to 1000 c.c. (depending upon the weight of the patient), and the urine is again carefully measured. If the quantity of urine is found to be proportionately greater than when more fluid was being ingested, it indicates that the heart still has some rallying power and the circulation is able to carry off through the kidneys some of the superfluous fluid stored in the body, making the prognosis much more favorable than it otherwise would be.

Should a serious surgical operation become necessary in an obese subject, the prognosis at once becomes grave. Sir James Paget[2] wrote many years ago:

"I know no operations in which I more nearly despair of doing good than in those for umbilical hernia or for compound fractures in people that are over-fat. Nothing short of the clearest evidence of

[1] *Loc. cit.*, p. 690.

[2] *Clinical Lectures and Essays*, 1879, p. 14.

necessity or of great probable good should lead you to advise cutting operations in people of this kind." Life insurance companies usually regard obese patients among "extra hazardous" risks. Death occurs from various causes, notably cardiac syncope, apoplexy, uræmia, cardiac rupture (rare), cerebral thrombosis, pneumonia, etc.

DIAGNOSIS.—The diagnosis of obesity is readily made from the patient's appearance and history, and no one is likely to confound it with general anasarca or œdema, although its presence often masks the diagnosis of other diseases from the physical impossibility of reaching the viscera or tumors by the ordinary methods of physical examination. The plethoric and anæmic types of the disease are easily distinguished from each other by attention to the symptoms above described.

TREATMENT.—The first requisite in the treatment of obesity is to determine the original cause of the malady, and to ascertain to what extent the visible fat accumulation may be accompanied by visceral interstitial deposits or fatty degeneration. In all cases there are two objects to be attained by treatment—first, to enable the patient to fully oxidize and eliminate the excess of fat already stored; and, secondly, to prevent its reaccumulation.

It will be inferred from the section upon the varied etiology (page 1033) of obesity that no one method of treatment can be made applicable to all cases. Much discretion must be exercised, and while under treatment the patient should be subjected to daily observation of the condition of the heart action, the circulation, the general muscular strength, the respiration, the urine, etc., for by such means only can one be assured that the patient, while perhaps rapidly losing flesh, is not also losing strength and vitality to a degree which may easily render recovery impossible. The patient also should be systematically weighed once a week.

The prophylaxis of hereditary obesity should consist in withholding from infants, as much as possible, saccharine and proprietary starchy preparations, malted foods, etc. Older children should not be allowed to form habits of overeating or excessive indulgence in candies, preserves, sweets, or syrups, and adults should avoid especially sweets of all kinds, malt liquors, and excess of carbohydrates of any other sort. Sedentary lives and habits of indolence must be discouraged.

In all cases of obesity among adults the possible occurrence of glycosuria must be borne in mind, as well as of gout. The bowels must be kept active with salines, such as Carlsbad or Rochelle salts, and feeble heart action must be strengthened by strychnine or digitalis. All things tending to restrict oxidation, such as tight lacing, sedentary habits with cramped positions, close air, and overdistention of the stomach, must be carefully avoided. Anæmia should be corrected, if possible, with iron and arsenic, and the skin should be kept clean and active by frequent bathing. For the more robust patients the daily morning cold bath, followed by vigorous friction, is most desirable.

Exercise must be as carefully prescribed as diet, and vigorous patients derive especial benefit from horseback riding and sometimes from wheeling, although the greater danger and difficulty attending the latter mode of exercise prohibits it for very heavy patients unless a tricycle is used. Patients who are unable to take these more violent

forms of exercise should practice walking over prescribed distances, or be given massage or passive movements.

Meals should be taken at regular intervals, and the dietary should be written out in full and carefully adhered to. Any dietary becomes monotonous in time, and many obese patients starve themselves into dangerous weakness by taking the advice of too many well-meaning friends, and by eliminating one article of food after another they finally reach a condition of prostration. Patients cannot be got to weigh their food regularly, and practically that is unnecessary, for common sense rules are usually sufficient, and too minute attention directed to matters of diet destroys all appetite and defeats the best ends. A too long continued diet of meat often causes as much dyspepsia as saccharine food, besides being very constipating, and monotony in diet should be avoided.

Relapses are apt to occur under any system of treatment, and in the plethoric cases in the earlier stage sometimes the very exercise and hygienic measures recommended increase the appetite and digestive powers so much that the patient eats voraciously and goes on gaining in weight despite treatment.

"Anti-fat" remedies are much sought after by a credulous public, who support the sale of countless quack nostrums offering delusive hope of relief. The entire lot of "reduction pills," concentrated salts and purges, extracts of phytolacca-berries (*Fucus vesiculosus*), and other so-called "specifics" for reduction of obesity, must be strongly condemned as absolutely unscientific and often harmful or dangerous, as many of them unquestionably are.

There is no medicinal specific for obesity, and the treatment beyond that of the complications is almost wholly dietetic. Whatever favor reducing remedies may from time to time have seemed to possess is due either to the fact that they destroy the appetite and disorder digestion, or else the patient who has faith in them is induced to accompany their use with dietetic restrictions which he would not otherwise be persuaded are for his advantage.

Many different dietetic systems have been put in practice from time to time for the treatment of obesity with more or less success, some of which appear diametrically opposed, but the following statements will, I think, be generally accepted without question:

1. No single article of diet can be held responsible for obesity, nor will the withdrawal of any single article of food, such as sugar, control it.

2. The carbohydrates as a class must be reduced in amount, and sweets of all kinds must be positively forbidden.

3. The ingestion of fluids must be restricted to as nearly 30 ounces per diem as possible.

4. In the plethoric cases with good appetites and digestion, a reduction in the total quantity of food eaten is necessary.

5. In serious cases it is undesirable to make too sudden or too radical alterations in the dietary, lest the patient's strength be critically impaired.

6. It is highly important not to weaken the patient by starvation or too rigid enforcement of any dietetic system, or too long continuance of such system, if improvement does not follow.

7. If such improvement be not apparent under dietetic treatment within two or three weeks, it is doubtful whether it can be attained at all, especially in hereditary cases.

8. As a rule, the longer the case has existed, or the more advanced it has become, the less likelihood is there of permanent relief.

9. It is best, whenever possible, to forbid the use of alcohol in any form, but especially the use of malt liquors.

It would be easy to supplement this article with elaborate dietetic tables and calculations of food ingredients and calories, but while such observations are of scientific interest, I have never found them of practical application, for the peculiarities of each case must be studied by themselves. Von Noörden recognized this when he found that patients accustomed to eating large quantities of carbohydrates and less fat often did best when the fat was reduced instead of the carbohydrates, and *vice versâ*.

In early cases of commencing acquired or hereditary plethoric obesity the following regimen is usually sufficient to effect improvement or equilibrium, if not cure. The patient should take three meals a day, moderate in amount and at regular intervals. Meat should be eaten but twice a day, no soups should be eaten, and as little fluid as possible should be taken with meals. A small tumblerful of water (6 ounces), either hot or cold, should be taken three quarters of an hour before each meal, and again on rising, and on retiring at night. This will give about 30 ounces of fluid per diem, a low standard, which should be maintained for ten days or a fortnight, if possible, and subsequently slightly increased. If weak tea or black coffee is drunk, the quantity of water may be correspondingly reduced.

Saccharine or diabetine may be substituted for sugar in beverages or in cooking, although many patients refuse such makeshifts on account of their taste. Acid fruit may be cooked with a little soda to render less sugar necessary.

Breakfast should be fairly substantial, and should consist of a stale roll or a couple of slices of dry toast with a minimum of butter, and such meat as lamb or mutton chop, beefsteak, broiled chicken, lean ham, or lean crisp bacon, or if preferred, any variety of broiled fresh fish may be substituted. Such fruit may be added as oranges, grapefruit, or sour apples, raw or stewed. Coffee may be drunk either black or with a little skimmed milk, but without sugar.

Luncheon may comprise such food as raw oysters or clams, crabs or lobster, eggs, soft "boiled," poached, or in omelette, liver, stale bread, dry toast, crackers, unsweetened wafers, celery or lettuce, with a little old cheese.

Dinner must consist mainly of meat or fish, and vegetables must be eaten sparingly, not more than two kinds at any one meal. Soups and desserts had better be omitted entirely. Any variety of fresh fish or shell fish may be eaten if not fried. Roast lean beef, veal, mutton, lamb, chicken, turkey (without "dressing" or sauces made with flour), or game of any kind may be allowed the patient, and one or two of such vegetables as stewed tomatoes, spinach, string beans, beet-tops, Brussels sprouts, asparagus, green peppers, celery, squash, oyster plant, cucumbers; as relishes, pickles, horseradish, or radishes. Fruits, as

specified for breakfast, may be again eaten, or a few almonds are allowable.

If alcoholic beverages are indicated by long continued habit or otherwise, they should be restricted to a small glass or two of one of the following: Scotch whiskey and water, a thin claret, or dry Moselle diluted with Apollinaris.

The above dietary, while adapted to the more plethoric cases, may not be best for anæmic, debilitated subjects or those whose poor appetite prevents them from getting enough nourishment from a regimen which does not appeal to their palate. Such patients, while keeping as near as possible to the standard above given, need supplementary feeding in the form of light lunches in the middle of the forenoon and afternoon, and perhaps just before retiring.

For this purpose they are to take such articles as a cup of bouillon or of broth with a plain biscuit, an egg-nogg, a thin scraped-beef sandwich, a cup of unsweetened cocoa, etc.

While the writer is of the belief that it is wholly unnecessary to exalt the treatment of obesity to the exclusiveness of a medical "specialty," it would be unfair to the reader to omit brief mention of the chief systems of treatment which have been long practised successfully in Europe, and to some extent imitated in this country.[1] Of these systems the best known and most successful to-day are those of Oertel and Ebstein, the true Banting system, introduced originally by Dr. W. Harvey, having proved too rigid. Schweininger's "cure" is practically that of Oertel, modified by withholding all fluid from the meals and until fully two hours have elapsed after them. Schleicher's system is also similar to Oertel's.

The Banting system, by which Mr. Banting in 1862 succeeded in himself losing 46 pounds of superfluous weight within a year, was a starvation regimen on which patients were allowed only between 21 and 27 ounces of food per diem, about one half of which was meat, only 2 ounces being bread, the balance being composed of fresh fruits and the lighter kinds of green vegetables. This dietary has been found too restricted for any but the most robust or plethoric subjects, and has in consequence been practically abandoned.

The Ebstein treatment, as originally practised in Germany, has met with more success. Fat is allowed in this dietary upon the theory that it does not promote fat storage in the body, and by contributing to the sense of satiety, makes it easier to eat less food. Ebstein, therefore, allows cream, butter, fat meat, and fatty soups, and proscribes the use of sugar, potatoes, and all forms of starchy foods except $3\frac{1}{2}$ ounces of bread per diem. Among vegetable foods he includes asparagus, spinach, cabbage, pease, beans, and salads. Meat of any kind may be eaten, together with fresh fish or eggs.

The *Oertel treatment* for obesity consists of the application of two principles: (1) the dietetic, and (2) the mechanical. His dietary includes almost twice as much animal food as that of Ebstein, double the quantity of carbohydrates, and less than half the quantity of fats. Fluid is withheld as much as possible, and that prescribed consists of a moderate

[1] For a full account of these various systems of treatment the reader is referred to the writer's work on *Practical Dietetics*, New York, 1898, pp. 599–615.

cup (six ounces) of coffee, weak tea, or milk twice a day, with twelve ounces of wine diluted with an equal quantity of water, which may be taken at dinner. From four to six ounces of bread are given per diem, and, while meats make up the bulk of the menu, eggs, fresh fish, fruits, salads, and a few fresh green vegetables are admitted.

The second or "mechanical" part of the Oertel treatment for obesity consists in carefully regulated exercise by hill climbing, which is prescribed as definitely as a medicine. The idea of this treatment by open-air exercise in favorable climatic resorts is to promote the formation of blood and strengthen the muscular tissue, especially of the enfeebled heart wall, and to increase oxidation, thereby enabling the patient, who is at the same time dieting, to consume the superfluous fat of his own body, eliminate the superfluous fluid through the skin and kidneys, and oxidize his food so thoroughly that further fat deposit ceases.

The Oertel treatment has attained great reputation in Europe, and special sanitaria or "Terrain Curorte" for its administration have been established in Germany, Austria, and Switzerland. At these sanitaria "health paths" are laid out, partly level and in part winding about the hillside. They are divided into four different grades, varying in slope from 5° to 20°. They are marked with colored sign-boards, stating distances and elevations. The patient begins on the lesser grade, and walks a prescribed distance, no matter how long it takes him, stopping as he wills for breath. Each day the distance is increased, or the grade prescribed is steeper, until considerable effort can be made and the patient walks for three or four hours without dyspnœa or fatigue. By this system many excellent results have been obtained with patients who for years had taken practically no exercise, because what they first undertook was too violent or ill-adapted, and resulted in harm.

Voit's explanation of the value of a meat diet in reducing corpulency is that albumin while circulating in the blood is more readily oxidized than either fats or carbohydrates: the fat deposited in the tissues acts like free circulating fat, not primarily by saving proteids from oxidation, but by causing a greater proportion of them to enter into the organized tissues, thus saving tissue waste. The tissues use up the circulating albumin, but not being exhausted in the process, proceed to oxidize the fat besides, and thus reduce it. An exclusively lean-meat diet increases the circulating albumin, and if proteid waste be also freely eliminated, fat does not accumulate in the body. If fat be now added to the dietary, then it becomes deposited in the tissues.

The carbohydrates, like ingested fat, may similarly protect circulating albumin from destruction and aid its transformation to organic albumin, but it is not proved that they themselves *make* fat, as at first supposed, for they are very completely oxidized even when eaten in considerable excess. The fat accumulates rather from their protection of other foods from final oxidation. Meat and carbohydrate diet alone and free from fats may increase the tissue fat production, for the fat which originates from the splitting up of albumin is spared further metabolism by the carbohydrates.

In such manner is explained the fact that fat metabolism within the body is quite independent of fat ingestion.

Fat ingested as food is less easily oxidized than are carbohydrates,

and it has less tendency to hamper the final metabolism of proteids, and is therefore less apt than carbohydrates to cause obesity. Since fat, weight for weight, has more potential energy than carbohydrates, the latter furnish body heat quicker than fat, and the carbohydrates should enter into the normal dietary in from four to five times greater amount than proteids. In the normal diet from 1 to 2.5 ounces of fat is sufficient to maintain good health.

The urea eliminated represents less carbon proportionately than it does nitrogen as compared with the amount of these two elements ingested with the food, hence it seems quite possible for the carbon of the food not accounted for in the urea to unite in fat formation, provided it is not all eliminated as carbon dioxide. Michael Foster calculates that 100 grammes of proteid food might be made to yield 42 grammes of fat.

Germain Sée, contrary to most observers, declares that the use of abundant drink is distinctly beneficial in obesity. His diet consists mainly of proteids and fat, withholding carbohydrates and alcohol, but the patients are made to drink hot weak tea and water in large quantity.

Weir Mitchell's system is based mainly upon rest in bed with massage, Swedish movements, and a diet composed either wholly or in great part of skimmed milk. The treatment is carried out for a month or six weeks.

The Yeo diet admits the giving of fats in moderation. Meat is eaten but once a day (six ounces at a time). Eggs, fish, game, fresh green vegetables, and fruits are included. Hot water and hot aromatic drinks are prescribed freely between meals, especially for gouty subjects.

Dujardin-Beaumetz believes that the Ebstein and Oertel systems are too restricted to support normal nutrition and activity, and he prescribes water or light wines with meals.

Bouchard bases his treatment upon the results of frequent observation of the urine, reducing proteid food whenever the nitrogenous waste in the urine reaches a certain maximum.

The meat and hot water treatment is practically a starvation diet, by which the patient's strength is too greatly reduced for safety, and it has never been generally received with favor by the profession.

Sir Dyce Duckworth[1] furnishes the following excellent summary of an appropriate diet for a few weeks' treatment:

"For an adult in early and middle life the relative quantities of food required should average 12 to 14 ounces of meat, 6 to 8 ounces of toasted bread, rusk or gluten, and almond biscuit, 4 to 5 ounces of green vegetables, 1 to 1½ ounces of butter and fat, and 30 to 35 ounces of fluid, including wine, tea, and water."

Thyroid Extract.—Quite recently attention has been called to the value of the thyroid extract in the treatment of excessive obesity. The extraordinary influence which this remedy possesses in modifying nutrition in cretinism and myxœdema is well known (see Vol. III. pp. 697, 710), and in obesity it sometimes acts favorably. A loss of weight may occur under its administration down to a certain point, a loss, say of 30 pounds, but after this its effects are disappointing, and danger from en-

[1] Allbutt: *A System of Medicine*, vol. v. p. 619.

suing weakness may result. P. Jervis,[1] reports two cases treated with benefit without dieting by giving tabloids of thyroid extract. One of these patients lost 14 pounds in six weeks. The remedy should be given in 5-grain tabloids administered after meals three times a day, but the patients should be carefully watched from day to day, and identical treatment and precautions should be carried out with those described by Starr in the article upon Myxœdema, Vol. III. p. 710.

Attempts have been made to reduce corpulency by hydrotherapy upon the hypothesis that cold bathing favorably influences the heat-regulating functions of the body and modifies nutrition, but the results have proved disappointing, except in so far as the treatment acts as an adjuvant to other methods.

The systems of treatment in vogue at many of the foreign mineral springs are of benefit to cases of moderate severity, notably the hot alkaline sodium-sulphate baths. The plethoric cases do well at Carlsbad or Marienbad under more bracing treatment, and the more feeble patients do better at the milder courses of Ems, Kissingen, or Homberg. In this country sea-bathing in summer is beneficial for plethoric cases in the early stages, and in winter such patients may take one or two Russian baths a week with benefit, provided the circulation is still normal.

Ever since the close of the last century surgical removal of excessive fat has been occasionally practised. A pendulous accumulation in the lower abdomen may hang down over the pubes and impede locomotion, so that its excision is justifiable. Such operations are mainly of service where the accumulation is more or less localized and are naturally not of frequent application.

Several writers have reported the successful removal of cumbersome fat breasts. One such breast weighed 52 pounds (Portalupi), others $30\frac{1}{2}$ and $20\frac{1}{2}$ pounds respectively (Robert and Annesat).

[1] *Brit. Med. Journ.*, Oct. 2, 1897, p. 904.

SCURVY.

By W. GILMAN THOMPSON, M. D.

Synonym.—Scorbutus.

Definition.—Scurvy is a subacute or chronic disease, characterized by inanition, anæmia, a tendency to swelling and bleeding of the gums, and to purpura, with marked prostration.

Infantile scurvy has certain characteristics which make it desirable to consider it under a separate subheading (page 1060). The disease has long been recognized, and was described by Jean Eichtius in 1541 (Petit).

Etiology.—Scurvy is variously classed by different authors as a disease of the blood, of disordered nutrition, of infectious origin.

Frequency.—In past times and up to the middle of this century the disease was very much more common than at present. Vasco da Gama lost 160 sailors by it when upon one of his voyages around the Cape of Good Hope (Stengel). War, pestilence, and famine have always had more or less scurvy associated. To-day, owing to the shorter voyages, more wholesome food, improved hygiene, and better understanding of the disease, it is comparatively rare, except in the far East, where hygiene is still rudimentary.

The occurrence of scurvy on shipboard in the merchant marine formerly became such a public scandal that boards of trade have been compelled to require that antiscorbutic foods and remedies should be carried on all vessels undertaking long voyages, and the development of scurvy at sea, unless in case of shipwreck, or on voyages prolonged beyond all expected limit, may subject the captains or owners to indictment for criminal negligence. An English law early required that lime-juice should be carried upon long voyages, and this formerly earned the nickname of "lime-juicers" for British sailors. Many almshouses have similar regulations. An ounce a day should be served with grog after the first ten days at sea.

Epidemicity.—Scurvy has long been held to be of epidemic origin, and the recent suggestion that it is an infection lends some support to this view. It has often prevailed very extensively in armies, on overcrowded, ill-ventilated ships during long voyages, in almshouses, prisons, etc., but before true epidemicity is acknowledged it must be shown that the common dietetic and hygienic factors have not been present to simultaneously affect a large number of persons. In the United States at least the disease is now rarely seen except in sporadic instances, and the records of the largest metropolitan hospitals may report no cases for a year or two at a time.

Food.—Improper diet is unquestionably the exciting cause of scurvy

in nearly all, if not all, cases; but in attempting to define wherein the dietetic error consists, one is met by very conflicting facts, and the conclusion must be reached that neither the presence nor absence of any one food or any special class of foods is invariably productive of scurvy.

Among the chief dietetic errors which have been believed to produce it are: excess of salt meat and fish, exclusive meat diet, tainted food, badly cooked food, too much fat, lack of fresh vegetables and fruits, a too monotonous diet, and, in infants, absence of fresh milk.

Of these causes, excepting the last named, the absence of fresh vegetable food is admittedly the most important factor. Mere deficiency of proper food may sometimes cause scurvy, although professional fasters do not become scorbutic. I have seen several mild cases in anæmic convalescents from protracted enteric fever who had been too long kept upon an exclusive milk diet. Berthensen has reported six cases having similar origin.

In seeking the dietetic origin of scurvy one should review thoroughly the dietetic habits of the different tribes and races of man, or false emphasis will be placed on isolated observations. For example, the most northern Eskimos and the Indians of the Alaskan Archipelago live on an exclusive diet of meat, fish, eggs, etc., without ever tasting vegetable food, which is inaccessible to them. Yet they are not subject to scurvy, and are uncommonly healthy. Moreover, they have decided preference for stale and putrescent food. The Congo African, on the other hand, subsists chiefly upon plantains, rarely having any proteid food, and he is similarly free from scurvy, and nothing can surpass the monotony of diet among these two extremes of race. It must be admitted from these and similar observations that food which may produce scurvy in one group of men may not in another, for while, as just stated, the Eskimo escapes the disease, like his Mongolian brother who lives upon pure rice, the earlier American and European expeditions to the Eskimo's land found scurvy the most serious malady encountered. In the British Arctic expedition of 1875–1876 over 48 per cent. of the men suffered from scurvy.

When the potato crop failed in Ireland in 1846 scurvy became very prevalent, and in the Crimean war 23,000 cases occurred among the French troops alone.

Scurvy contributed over 15 per cent. to the death rate in the late Civil War in this country, and Woodruff,[1] referring to scurvy in the United States army, writes: "If transportation is so deficient that only bacon, hard-tack, and coffee can be carried, actual scurvy is the result. The company commander must secure something else for his men. The lack of fresh vegetables and fresh meat is the chief fault. Why fresh things are needed is not known, but it is believed to be due to the fact that the body thus receives certain salts and unknown substances necessary as stimulants or tonics to the tissues, which salts and substances are destroyed by the usual methods of preservation.

"It is not to be denied that men may live for many years without tasting such articles of diet, though it is rare for a man to be denied all three—meats, fresh vegetables, and fresh fruits. If they are so denied,

[1] *Journ. Amer. Med. Assoc.*, Dec. 3, 1892.

they are not possessed of that health which permits of the highest mental and physical development."

Scurvy has often occurred both among children and adults in asylums, reformatories, and similar institutions, where the food furnished was in itself wholesome enough, but was either spoiled or made too monotonous in the cooking.

Bad drinking-water has been said to cause scurvy in camps and garrisons, even when the food was wholesome.

Age.—Scurvy may develop at any age. It was originally supposed that infants were exempt, and so they are when breast-fed, but within the past ten years it has been proved that the proprietary infant foods, given to the exclusion of good fresh milk, beget a fairly acute type of the disease. For this reason scurvy is at present almost as common among infants as among adults in this country. (See Scurvy in Infants, page 1060.) It occasionally occurs among the aged poor, who from loss of teeth cannot masticate, and who cannot afford appropriate diet.

Sex.—The disease is comparatively rare among women who are not subject to many of the conditions which cause it in men. Under circumstances of famine, siege of cities, etc., women are alike affected with men, suffering identical privations. The protracted sieges of Paris and Metz, during the Franco-Prussian war, gave abundant illustrations of this fact.

Other diseases predispose somewhat to scurvy, notably those attended by emaciation, anæmia, and purpura. It is found in connection with dysentery and other chronic intestinal maladies, causing failure of food absorption; malarial cachexia, advanced syphilis, and alcoholism. The decay of the teeth is important by preventing mastication. Nervousness, worry, anxiety, and notably home-sickness, all may contribute to the predisposition.

Other Causes.—Heredity is probably without influence. Occupation is also without influence, except in so far as it leads men to undertake voyages and expeditions of hardship where fresh food is unobtainable.

Season and climate have been thought to influence the disease, but it is difficult to formulate rules regarding them. It can only be said that the prevalence of scurvy has been much greater in some years than in others, irrespective of famine. Cold seasons, which drive people indoors and thereby subject them to bad hygiene, such as damp, dirty, ill-ventilated quarters, lack of food and other hardships, produce more scurvy, and after such experience the disease prevails in the early spring months, disappearing rapidly thereafter (Hermann-Amberger). The geographical distribution of the disease is universal.

PATHOGENESIS.—The earlier theories of scurvy have been gradually disproved, and definite knowledge of its pathogeny is lacking.

Excess of sodium chloride in the blood from consumption of salt meats and fish has been found in scurvy, but also in anæmias of all forms, and moreover scurvy occurs without any such change.

Garrod advanced the theory that a lack of potassium, which is ordinarily supplied by potatoes and other vegetables or by fruits, is the cause of scurvy. He found a diminution of potassium salts in the blood in this disease, and Duchek reported an increase in their elimination by the urine, but further research has shown the alleged excess in

the urine to be merely relative, and scurvy may occur despite a normal supply of potassium salts in the food. Moreover, similar conditions are observed in anæmia without scurvy. Immermann believed that a lack of these salts caused trophic disorders, which persist for some time after the deficiency has been made good; and Bauer explained the late development of scurvy on the ground that there is always some supply of salts on hand which must be first used. Ralfe found explanation of the development of scurvy in the absence of common salts of vegetable acids—the citrates, lactates, malates, etc., which by their conversion into carbonates in the blood aid in maintaining its alkalinity—hence the blood is more acid in scurvy. But this theory has also failed of general acceptance.

Infection.—Stengel,[1] with Babes[2] and others, is inclined to regard scurvy as an infection, although admitting the difficulty of proving this theory. He argues thus from the facts that scurvy may be epidemic, that it tends to produce purpuric and other hemorrhages, and that several experimenters have transferred the purpura from man to animals by blood inoculation (this, however, might have been septicæmic). Müller, and later Babes in 1893, reported the finding of a specific elongated bacillus, together with streptococci, in the superficial and deeper layers of scorbutic gums. Inoculation of this tissue in rabbits causes purpura and ecchymoses; but Babes regards this bacillus as of common occurrence in the mouth, and not therefore the sole cause of scurvy, although it may be stimulated to activity when the health is debilitated. Boruträger[3] inclines to somewhat similar views after finding a special coccus in hemorrhagic areas in the spleen of one dead of scurvy. Berthensen,[4] after studying an epidemic of 225 cases in St. Petersburg, argues that the disease is an infection on the following grounds: it is epidemic, apparently infectious after distinct incubation (in several cases), and second attacks are rare. Relapses do occur, however.

W. Koch[5] stands almost alone in the belief that all hemorrhagic diseases have common origin in scurvy, which is due rather to certain tissue changes and not to bacteria.

Pathological Anatomy.—Post-mortem examination of the body of a patient dead from scurvy at once suggests profound changes in the composition of the blood, and yet chemical and microscopical examinations alike fail to reveal the exact nature of these changes. Ecchymoses, extravasations of blood and serum, and œdema are common, and the blood is deficient in potassium salts, while those of sodium are increased and alkalinity is lessened. These chemical alterations are by no means characteristic, however, and the microscopical examination shows no peculiarities in the anæmia which is often present. The red corpuscles may be normal in number or diminished by one third, or even one half in severe cases, and the hæmoglobin may be somewhat disproportionately decreased. Similarly, the white corpuscles may be normal in number, or together with the eosinophiles considerably increased. Penzoldt

[1] *Twentieth Century Practice of Medicine*, vol. viii. p. 496.
[2] *New York Med. Record*, May 19, 1894.
[3] *Vierteljahreschrift für gericht. Med. und Sanitätswesen*, Berlin, B. 6, H. 2, 1893.
[4] *Deutsch. Arch. für klin. Med.*, Bd. xl. H. 2-5.
[5] *Jahrbuch für Kinderheilkunde*, B. 32, H. 1, 2.

describes numerous small, granular, refractive cells. All of these modifications of the blood are from time to time found in varieties of anæmia or other conditions in no wise associated with scurvy.

In scurvy also the blood coagulates poorly or not at all, and rigor mortis is ill-defined.

The ecchymoses appear as purpuric eruptions of varying size in the skin, in the serous membranes, notably the pleura, pericardium, cerebro-spinal meninges, and synovial linings of the joints, and in the mucous membranes of the mouth, stomach, intestines, and bronchi. Larger extravasations, hæmatomata, sometimes softened, suppurating, or often partially organized, are observed in the larger muscles like the gluteus and gastrocnemius, and in the vicinities of the joints. The latter contain increased synovia, sometimes hemorrhagic, and pseudo-crepitus and pseudo-ankylosis (Stengel) may be detected from fibrous thickening about the clots. The congested serous surfaces of the membranes above mentioned pour out increased exudation, which is apt to be hemorrhagic, especially in the pleural cavities. These changes appear to be congestive rather than inflammatory, and cell proliferation is absent.

Owing to the anæmia, vascular weakness, and altered composition of the blood œdema is common, both in the lungs and in the subcutaneous tissues, especially about the feet and legs.

The heart presents parenchymatous and fatty changes, myocarditis being the prevailing condition. Dilatation ensues, and ecchymoses are found both upon the epi- and endocardium (Stengel).[1] The liver is anæmic, often fatty, and like the enlarged spleen and the kidneys, may show hemorrhagic infarcts and areas of softening. Several pathologists describe lymphoid proliferation and hemorrhagic infiltration of the bone marrow, and still more rarely of the mesenteric glands. The bones may be fractured, carious, or present separation of epiphyses. The gum lesions are typical, almost pathognomonic. The extent of the lesions varies from the portion between the upper and lower incisors to the entire gum surfaces. There is much swelling, with redness, fibrous thickening of the deeper layers, which may cause distinct protrusions, with degeneration, necrosis, and erosion of the superficial layers. The bloodvessels may become thickened and to some extent occluded, or they may be eroded by ulceration.

Symptoms.—The symptoms develop very insidiously. In some cases the early symptoms being overlooked, a sudden, extensive, internal hemorrhage first attracts attention, but it is always the result of a gradual onset of the disease.

The patient acquires decided pallor, and complains of great weakness and fatigue on slight exertion. The skin is dry, cool, and blanched, and there may be a subicteric hue in skin and conjunctivæ. The muscles are flaccid. Protracted cases acquire a strikingly hollow visage and considerable general emaciation, but the latter is not always an early appearance.

The first symptom noticed by the patient may be bleeding from the gums occurring on brushing the teeth or biting hard substances. There is more or less stomatitis ulcerosa. The tongue is at first swollen, and later becomes dry, hardened, and encrusted.

[1] *Loc. cit.*, p. 499.

The gums are at first red and swollen, and bleed easily upon the slightest touch. Later, they may become pale and are irregularly enlarged, or somewhat fungous and friable, protruding between the incisors and other teeth, and bulging up over their surfaces. They are usually quite tender to the touch, but may not be otherwise painful. The buccal surfaces may present superficial ulcers. There is often quite serious hemorrhage from the mouth, and more or less oozing of blood is constantly present. This blood, by decomposing, adds to the fetor of the breath, for owing to the soreness of the mouth and weakness of the patient the tongue becomes extremely foul, and the odor of the breath from bad cases is almost unendurable.

The patient early complains of anorexia and a "bad taste in the mouth." There is often thirst and unnatural craving for sour foods and strong condiments. The stomach becomes irritable, nausea and vomiting are common, and there may be hæmatemesis. Early constipation may be succeeded by diarrhœa, offensive and sometimes watery hemorrhagic stools, which latter have been described as dysenteric. Somewhat late in each case hemorrhages appear. They may be visible in the form of petechiæ or larger purpuric spots on the extremities and trunk, or invisible in the serous cavities (pericardium, pleura, joint cavities, etc.). Ecchymoses are most common on the feet and legs, least so upon the face. They occur both around the sweat glands and hair follicles (Stengel). Considerable extravasations of blood may occur in the subcutaneous tissue and in the connective tissue between the muscles. Superficial blebs and ulcers may form over the areas of extravasation. Hemorrhage also takes place from the mucous surfaces, but less often than in hemorrhagic purpura. Blood may come from the bladder, rectum, uterus, and nose, as well as mouth. Hæmaturia has been observed. The major joints, especially those of the lower extremities, may become swollen, stiff, and tender. Bruises are ill borne, and wounds do not heal readily.

The circulation and respiration present no peculiarities beyond those naturally caused by loss of blood, anæmia, and weakness. The pulse, in severe cases, becomes quite rapid and feeble; the heart sounds are weakened, possibly accompanied by a hæmic murmur; attacks of syncope and dyspnœa occur. Congestion of the lungs may be present. The temperature usually remains normal or slightly subnormal. The urine is lessened in quantity, and of deeper color and higher specific gravity than normal. The acidity is diminished, as are also the alkaline phosphates. Peptonuria, acetonuria, and albuminuria have all been observed, as well as hæmoglobinuria, but evidence of severe nephritis is not common.

Ocular symptoms may be present. Night-blindness has been observed among sailors. Hemorrhages may take place in the conjunctiva or in any of the structures of the eye itself, with more or less corresponding visual disturbance.

The mental symptoms are characterized by deep depression, verging upon melancholia. Muttering delirium and coma precede dissolution. Meningeal hemorrhage may give rise to convulsions and localized symptoms, such as hemiplegia.

Complications and Sequelæ.—The stomach is often exceedingly

irritable, and ulcerative enteritis and dysentery may develop. Lobar and broncho-pneumonia, and pleurisy, or rather a hemorrhagic hydrothorax, are very common and often fatal. Stiffening and swelling of the joints may persist for months, and even ankylosis may ensue. In cases in which the gums have been badly affected, permanent loss of their substance may result, with formation of cicatrices and subsequent caries of the teeth. Desquamation may accompany convalescence. Nephritis is rare.

Diagnosis.—Scurvy, especially if the mouth does not happen to be very sore, is often overlooked until the patient has become dangerously weak, and even then it may be mistaken for the cachexia of malignant disease—purpura, general neurasthenia, marasmus, etc. The essential diagnostic features are the fungous and hyperæmic condition of the gums, the hemorrhages from the gums and other mucous surfaces, hemorrhages into the skin, viscera, and serous cavities, a tendency to swelling and soreness of the larger joints, and œdema. In purpura the hemorrhages are usually not visceral or serous, but mainly superficial, and, as in the other diseases mentioned, the gums are not affected. The gums are, however, also not affected in scurvy in the toothless extremes of life—in infants and the very old. In purpura the joint symptoms are often more pronounced than in scurvy, except in children.

The history of the case, the prevalence of an epidemic, and favorable reaction to dietetic treatment must all be considered.

Prognosis.—Cases recognized early and which are favorably situated almost invariably recover completely, usually after an interval of a few weeks. Bad cases are very serious and sometimes fatal. Weakened by anæmia and inanition they may easily succumb to hemorrhages, or die from simple asthenia. Fatal cases have occurred from sudden hemorrhage, and sometimes from septic infection, as well as in the more common way from inanition and asthenia. In an epidemic occurring in 1891 in the Military Hospital of St. Petersburg, of 225 patients, 19, or 8½ per cent., died, and 52 had to be paroled for an entire year on account of anæmia and weakness (Berthensen).[1]

Relapses are very common among those who are unable to maintain the prophylaxis and treatment which might be curative. In this manner the disease may be prolonged for months or several years.

Prophylaxis.—If the rules of cleanliness, good discipline, regularity in hours of eating and sleeping, etc. be enforced among soldiers and sailors, scurvy may be avoided, even if fresh vegetable food is not procurable. Dried legumes are useless preventive foods, but canned and compressed vegetables in seamen's rations have done much to prevent scurvy; but fresh food is always preferable to any other, no matter how it may be preserved.

Treatment.—The treatment of ordinary mild cases of scurvy is almost wholly dietetic, and recovery usually is complete within from two or three days to as many weeks. The more severe cases require tonic and stimulant medication, with absolute rest. The citrate of iron, or of quinine and iron, with nux vomica, are among the best tonics. Iron and arsenic should always be given in some mild form if there is much anæmia. The enfeebled circulation may require strychnine or

[1] *Loc. cit.*

digitalis. Vinegar, acetic acid, potassium citrate, chlorate, and bitartrate have all proved beneficial. During the War of the Rebellion the expressed juice of sorghum was used with some success. For stomatitis and bleeding gums astringent mouth-washes of alum, etc. may be employed, and brushing the gums with a 10 per cent. silver-nitrate solution, or a solution of potassium permanganate or potassium chlorate used as a mouth-wash are to be recommended. Calcium salts have been given to prevent hemorrhage, but it is doubtful if any drug exercises much control in this regard in scurvy.

In mild cases of short duration a few fresh vegetables or oranges, or the juice of two or three limes or lemons, must be eaten daily; and lime-juice has long been regarded as an excellent preventive as well as curative agent. A. E. Wright,[1] in an interesting discussion of blood coagulation, throws disfavor upon fresh lemon- or lime-juice, on the ground that their chief acids, citric and tartaric, when given *per os*, have a decided inhibitory control over intra-vascular coagulation, and hence, in cases with much hemorrhage from the gums, tend to maintain oozing of the blood. Neutral citrates are preferable, as they do not act in this manner. These objections are, however, rather theoretical than practical. The bleeding from the mouth is easily controlled as above described, and lime-juice is unquestionably a good remedy.

In more severe cases, when the mouth becomes too sore to masticate ordinary food, or the patient is too feeble to digest it, fluids must be given. Fresh milk, broths and meat soups thickened with vegetables, fresh vegetable *purées*, and eggs are the best. Fresh orange-, lemon- or lime-juice must be added four or five times daily. At sea and in camps, when the patients become stronger, there may be given, with fresh meat, when obtainable, sauerkraut, well-boiled cabbage, baked potatoes, pickles, salads, and "greens," such as beet-tops, water-cress, fresh mustard, or radishes. Lettuce, spinach, sorrel, potatoes, and apples, as well as limes and lemons, have all been rated as "anti-scorbutic" foods, but no one of these can be specially relied upon.

SCURVY IN INFANTS AND YOUNG CHILDREN.

Etiology.—It is now twenty years since Ingerslev and W. B. Cheadle[2] first called attention to the possibility of scurvy in the very young, and since that time a large number of confirmatory observations have been reported, and to Thomas Barlow[3] belongs the credit of first carefully describing the peculiar pathological conditions of the lower limbs in cases especially associated with rickets. Sutherland,[4] in a report of 71 cases, found rachitic diathesis to be a very strong factor underlying the extensive bone lesions. Jacobi reports 40 cases of association of these two diseases (see Rachitis, page 938).

Among infants the disease is most apt to first appear from the ninth to the fourteenth month, but it may develop at any later period. Rotch,[5]

[1] *Brit. Med. Journ.*, July, 1894, p. 57.
[2] *Lancet*, Nov., 1878.
[3] *Med.-Chir. Trans.*, London, vol. lxvi., 1883.
[4] *Practitioner*, London, Feb., 1894.
[5] *Pediatrics*, 1896, p. 1076.

referring to a personal experience with 60 or 70 cases, says that nearly all occurred between eight and twelve months of age, and that he "has met with no cases later than the first half of the second year, and with none earlier than the first half of the first year."

Northrup[1] and Crandall in 1894 reported an examination into the causes of infantile scurvy, and found it almost invariably due to substitution, for various reasons, of proprietary foods for fresh food. They write that "even fresh milk in small proportions is not sufficient to insure immunity," and "the exact diet is known in 33 cases. We find that 12 of these children (36 per cent.) were fed on proprietary foods exclusively, 6 (18 per cent.) had received an exclusive diet of condensed milk or evaporated cream, while 3 received a combination of these two foods. Over 63 per cent., therefore, were fed upon a diet of proprietary foods and condensed milk. Two children received sterilized milk exclusively, and 3 a weak mixture of milk and water."

Northrup adds: "It is a significant fact that the country which furnishes most of the literature of scorbutus in children is the same which is posted from end to end with advertisements of proprietary foods." He says also that the children of the well-to-do are even more apt to have scurvy than those of the very poor, owing to the ability of the parents to indulge the child in a variety of such foods; and Cheadle called attention to the fact that the children of the poor are usually taken while very young to the family table, where they are often given potatoes and similar foods.

Barlow,[2] in summarizing the histories of numerous cases of infantile scurvy, says: "There is no evidence that any child has developed this group of symptoms whilst being suckled at the breast. Further, the most striking cases are those in which infants have been brought up for several months on artificial foods prepared with water and without any fresh aliment."

Some of those who have recognized scurvy in infants (Heubner, Barlow, and others) state that it may sometimes be caused by the prolonged use of sterilized milk—*i. e.* milk heated to 212° F., but Stengel, Holt, and Rotch dissent from this view. Milk of poor quality or too dilute has been shown to cause it.

PATHOLOGICAL ANATOMY.—The essential differences between scurvy in the adult and in infancy and early childhood consist in the presence in the latter of subperiosteal hemorrhages and thickening of the long bones of the legs, separation of the epiphyses, and excessive local pain and tenderness, with pseudo-paralysis. These changes are most pronounced about the femur, but the tibia and fibula are often involved to a lesser degree. The periosteum is raised by the extravasated blood, producing a fusiform swelling about the shaft, and there may be superficial necrosis of the bone.

There is corresponding general swelling of the skin, which is tense and shining. The tenderness is so great over the swollen limbs that the child shrieks with pain if disturbed, and the occurrence of pseudo-paralysis is explained by the pain caused by any contracture of the muscles attached to the tender periosteum. When the swelling subsides under

[1] *New York Med. Journ.*, May 26, 1894.

[2] *Keating's Cyclopædia of the Diseases of Children*, vol. ii. p. 276.

treatment and the clots are absorbed, it then becomes apparent that the shafts of the long bones are considerably thickened, and their epiphyses are sometimes found to be separated, giving crepitus. A curious case was diagnosed by Henry Ling Taylor,[1] of a girl eleven months old, having localized pain and swelling over the first and second lumbar vertebræ, which had led to a previous diagnosis of Pott's disease by several physicians. The child had pseudo-paralysis, spongy gums, and other typical symptoms, and recovered upon dietetic regimen.

The forearms may be affected in the same manner with the legs, but far less frequently, and the thighs are usually worse than the legs. The scapula, lumbar vertebræ, and orbits have all been affected. The joints usually escape.

Symptoms.—The symptoms, aside from the pain, tenderness, swelling, pseudo-paralysis, and crepitus, with tense shiny skin, which have been referred to in connection with the pathological changes in the leg bones, are practically those already described as occurring in the adult, although in toothless gums there is little if any swelling, and the presence of punctate ecchymoses may be the only symptom in the mouth. Older children who are teething, or who already have teeth, often have a stomatitis ulcerosa, like adults.

The infant with scurvy loses appetite, becomes pallid, often perspires freely, and lies very quietly, dreading all movements which elicit pain. Such a child will scream with terror if it thinks the legs are to be moved. Diarrhœa is common.

As in the adult, but less commonly, general purpura and hemorrhages from mucous membranes or into viscera and serous cavities may be present. Hæmaturia may be the only exhibition of blood extravasation aside from the periosteal swellings. Proptosis, caused by blood extravasation behind the orbit, has been noticed by several clinicians, and Sutherland is disposed to regard œdema of the eyelids as diagnostic.

Diagnosis.—Scorbutus in infancy and childhood must be principally differentiated from acute and subacute rheumatism and rachitis. I have seen several cases recently which had been erroneously treated for rheumatism.

Rheumatism is much more acute, and is rare in very young infants. The pain and swelling are mainly in the joints, whereas in scurvy they are along the shafts of the femur, tibia, and fibula, and crepitus may be sometimes obtained at the epiphyses. The spongy gums, if present, the pseudo-paralysis, and finally the improvement under dietetic treatment, all point to scurvy, rheumatism being affected by salicylates and but little by diet. In rheumatism there are usually local heat and redness about the joints, which are wanting in scurvy.

Scorbutus in infants was originally regarded as acute rachitis, but this was an error, and the diagnosis from rickets can always be made with proper care. Rachitis and scurvy may, however, co-exist, and Rotch[2] says that he has seen a dozen cases in which the two diseases became coincident, and after cure of the scurvy the rachitic symptoms remained.

From rachitis scurvy may be chiefly differentiated by the following appearances: Greater relative involvement of the legs, with thickening

[1] *Amer. Med. Surg. Bull.*, New York, Feb. 1, 1894. [2] *Loc. cit.*

of the shafts of the long bones, and tendency to fracture of their epiphyseal ends; great local tenderness, combined with immobility of the limbs, extensive diffuse swelling of the legs, usually without pitting. In rickets there is gross enlargement of the epiphyses of the long bones, the square forehead, "beading" of the ribs, tendency to bowing of the legs, absence of pain, etc.—symptoms which have no counterpart in scurvy.

The stomatitis ulcerosa of scurvy is not found with rickets.

Purpura in infants lacks the striking bone phenomena of scurvy, as well as the swollen gums.

Hereditary syphilis also lacks the peculiar bone phenomena, and although periostitis rarely may be present, it is not due to hemorrhage, and osteochondritis is far more common. Ecchymoses are absent. Dactylitis, craniotabes, and catarrhal symptoms, common in syphilis, are lacking in scurvy.

Infantile paralysis ought not to be confounded with scurvy, for it can usually be demonstrated in the latter disease that the pseudo-paralysis is merely an appearance due to soreness and dread of pain.

Prognosis.—Not a few cases of infantile scurvy have proved fatal from lack of recognition until too late for treatment. The disease being much better understood than formerly, now offers a better prognosis, and cases which are put early upon a proper diet usually recover in a few weeks, and improvement may be expected within a few days.

Treatment.—The treatment of infantile scurvy is very simple. All prepared or proprietary foods, condensed milk, sterilized milk, etc., must be wholly discarded, and the infant is to be fed upon fresh mother's or cow's milk of good quality and not too much diluted, fresh expressed beef juice ʒij or iij daily, egg albumen, and fresh orange or peach juice. The juice of a whole orange may be daily given to an infant of nine or ten months. Children over one year old may be given a little of the expressed juice of boiled vegetables. Barlow recommends a little well boiled and "sieved" potato with cream. Scorbutic infants often take meat juice and orange juice with an eagerness which amounts to craving. Older children, of three to ten years of age, should be made to take fresh vegetable *purées*, baked mashed potatoes, scraped apple pulp, baked oranges, lemonade, etc., for the voluntary refusal of fresh vegetable foods may have been the chief etiological factor in the disease.

For the painful condition of the legs, rest in the horizontal position is enjoined, and the limbs should be protected in cotton and immobilized by sand bags. Wet compresses may be of service. During convalescence, if anæmia persists, the citrate of iron and quinine should be prescribed, and small doses of Fowler's solution of arsenic.

ADDISON'S DISEASE.

By WARREN COLEMAN, M. D.

DEFINITION.—Addison's disease is a constitutional affection characterized by progressive muscular weakness, loss of cardiac and vascular tone, alimentary disturbances, and generally by a peculiar pigmentation of the skin, due to interruption of the secretion of the suprarenal capsules, and probably also, in some cases, to reflex nerve disturbances.

ETIOLOGY.—Addison's disease is of infrequent occurrence. Lewin gives a total of 800 reported cases. The disease is thought to be more common on the Continent and England than in America. Heredity seems not to be a factor in its development. Andrews,[1] however, has recorded cases in two brothers. All cases hitherto described have been among Caucasians, but recently Kailas Chundra Bose[2] has reported a number of cases from his practice in India. In none of them, however, was the diagnosis confirmed by autopsy. No age is exempt from Addison's disease. It has been met with as early as the seventh day and as late as the eighty-first year. The majority of cases have occurred between the twentieth and fiftieth years. Addison's disease is of more frequent occurrence in males—about 65 per cent. of all cases. Greenhow has called attention to its greater frequency among laboring classes, consequent, as he supposes, upon their greater liability to overexertion and injury. It seems a well-established fact that injuries to the back or abdomen may be the determining factor in the development of the disease, and that without the injury being sufficient to incapacitate the individual. In some instances the injury has led to caries of the vertebræ, to be followed by Addison's disease.

Any or all of the above factors must be regarded as accidental, or at most predisposing, causes.

Addison's disease has been associated with *tuberculosis* of the suprarenal capsules in 80 per cent. of 757 cases.[3] The disease, however, does not seem to be more common in tubercular families. Tuberculosis of the suprarenal capsules is not necessarily associated with tubercular lesions in other parts of the body. The suprarenal capsules may be the only tubercular foci, though, on the other hand, the lesion may be secondary to tuberculosis of the lungs or vertebræ. The rarity of tuberculosis of the suprarenal capsules in pulmonary or general tuberculosis is a noteworthy fact. A systematic examination of the glands,

[1] *St. Bartholomew's Hospital Reports*, vol. xxvii.
[2] *Indian Medical Gazette*, July and Aug., 1888.
[3] W. Gilman Thompson: *Amer. Jour. Med. Sci.*, Oct., 1893, and *Trans. Assoc. Amer. Phys.*, 1893, p. 35.

however, shows the lesion to be more common than hitherto supposed. This rarity of involvement of the suprarenal capsules may be explained in one of two ways: either that when tubercle bacilli gain entrance to the circulation they are not carried to these glands by reason of some circulatory peculiarity, or that the glands are not readily susceptible to tuberculosis. It appears that something more than the mere presence of the bacilli in the glands is needed for their lodgement and development. From some unpublished experiments conducted at the Loomis Laboratory, I am inclined to believe that there is some inherent reason in the suprarenal capsules why tuberculosis is so uncommon in them—a something, perhaps, which is antagonistic to the localization of the lesion. The experiments are briefly as follows: The suprarenal capsules of eleven rabbits were directly inoculated after abdominal section with virulent cultures of tubercle bacilli, the wounds closed, and the rabbits killed after periods varying from six weeks to one year. In only two instances was the inoculation successful, though in all cases tuberculosis was found in other organs of the body. After failure in the first two or three experiments to produce tuberculosis of the suprarenal capsules the glands were injured by the needle before the bacilli were thrown in. In one case of the series a culture of streptococcus erysipelatus of proved virulence was inoculated simultaneously with the tubercle bacilli, yet without result. A legitimate conclusion from these experiments seems to be that there is some protective agency in the suprarenal capsules, at least in rabbits, against tubercular involvement.

As a rule, both capsules are tubercular in Addison's disease, and not only so, but there is almost, if not complete, destruction of the glands. Large portions of the glands, however, have remained uninvolved in other cases. In still other cases the lesion has been unilateral, leaving one gland entirely unaffected.

As stated above, some injury to the back or abdomen is sometimes the immediate predisposing cause to tuberculosis of the suprarenal capsules. Again, the tuberculosis may extend to the suprarenal capsules by direct continuity from some adjacent tubercular focus—*e. g.* caries of the vertebræ.

Of the remaining 20 per cent. of cases in the series above referred to which are not tubercular, 8 per cent. are associated with *some other lesion* of the gland—*e. g.* carcinoma, sarcoma, simple atrophy, etc.

Chronic interstitial inflammation, leading to destruction of the glandular substance, has been found associated with the disease.

Tumors of the suprarenal capsules have also been found, in a certain number of cases of Addison's disease, at the autopsy table.

Simple atrophy of the suprarenal capsules has been described in a limited number of cases as the cause of the disease. The atrophy has reached at times such an extreme grade that the glands could not be found after death. Such atrophy or the disappearance of the glands as the result of tuberculosis is the probable explanation of the 4 cases of Addison's disease reported by Martini, Spender, Fletcher, and Rispal, with congenital absence of the suprarenal capsules. In Rispal's case, for instance, the disease occurred in a man aged twenty-four years, who died ten months after the onset of the symptoms. Without antici-

pating the discussion of the relation of the suprarenal capsules to Addison's disease, in a case of this kind there is but one of two conclusions to be reached: either that the suprarenal capsules have no relation to Addison's disease, or that they had disappeared through atrophy or some other lesion. It is inconceivable, if the suprarenal capsules bear any relation to the disease, that they could have been congenitally absent and their absence not have caused symptoms during the twenty-three years of life previous to the admitted presence of the disease.

Blood cysts and extravasations of blood into the substance of the gland have also been present in cases of the disease. Cases associated with blood cysts and blood extravasations have generally, though not always, occurred in the early period of life.

Birsch-Hirschfeld, Andrews, and Chiperovitch[1] have reported cases of Addison's disease due to *syphilitic lesions* of the suprarenal capsules.

In the remaining 12 per cent. of cases of Addison's disease in the above series, *no lesion* of the suprarenal capsules was found. In some of the cases the abdominal sympathetic ganglia or nerves were described as diseased. In other cases not only was there no lesion of the sympathetic system, but no demonstrable lesion in the body.

Lesions in the *abdominal sympathetic* nervous system have been found in many cases of Addison's disease, generally associated with some lesion of the suprarenal capsules. These lesions have formed the basis of the nervous theory of the disease. It is doubtful, however, if they be a factor in the production of more than a single group of symptoms, and even in this group they may play a subsidiary rôle.

Attacks of malarial fever have occurred in cases which subsequently developed Addison's disease, and the question has been raised as to whether malarial fever bears any relation to the causation of Addison's disease. But a like inquiry into the past history of the patients would undoubtedly reveal the fact that other ordinary diseases had antedated the Addison's disease to the same or an even greater extent. It would appear that the development of Addison's disease in persons who had been previously affected with malarial fever was scarcely more than a coincidence. Indeed, it would be strange had not some of the diseases to which persons are ordinarily subject been recorded as preceding the affection.

PATHOLOGICAL ANATOMY.—The following lesions of the suprarenal capsules have been described in the reported cases of Addison's disease: (1) Tuberculosis; (2) chronic interstitial inflammation; (3) neoplasm; (4) simple atrophy; (5) blood extravasations; (6) syphilis.

Tuberculosis is by far the most frequent lesion of the suprarenal capsules in cases of Addison's disease. This lesion has been described in the past as scrofulous, caseous, and fibro-caseous. Such cases are now generally admitted to have been cases of tuberculosis. Both capsules are generally attacked, but one is frequently invaded sooner than the other, consequently, the two organs often present different stages of the process. Occasionally, a single gland is involved, the other remaining healthy.

[1] *La Bulletin Médicale*, Paris, July 21, 1895.

The tubercular process usually begins, as Virchow first pointed out, in the medulla of the gland as a single miliary tubercle or an aggregation of miliary tubercles. The process never, or but exceptionally, begins in the cortex. Several tubercular foci may start coincidently in the gland. At other times the foci are secondary to a primary deposit in the gland itself. Frequently smaller masses may be seen at the periphery of the primary focus. However this may be, multiplication, growth, and coalition of the tubercles ultimately lead to replacement of the medulla, and finally, by further extension, to obliteration of the organ as a physiological and anatomical entity. As the lesion progresses retrograde changes occur near the centre of the nodules, resulting in caseation. Following upon the caseation, but not with equal rapidity in all cases, a deposit of calcareous particles takes place in the necrotic areas, until finally the suprarenal capsules become converted into large, nodular, distorted masses, in which it may be impossible to demonstrate, even microscopically, the presence of suprarenal cells. When the pathological process has reached the surface of the organ the capsule becomes secondarily involved, and it may increase to several times its normal thickness. A reactive inflammation is often started in the circumjacent structures.

Sometimes the caseous areas undergo liquefaction instead of calcification, and pockets of creamy, yellowish, sometimes greenish material form. The glands in these cases appear to be the seat of abscess formation, but the semiliquid material is not pus. It consists of necrotic or necrosed cells and amorphous and fatty granules. Liquefaction of the necrosed tissue may extend throughout the entire mass and the gland become reduced to a dense fibrous capsule, enclosing a thick, creamy, yellowish material. Later on, when the water has been absorbed, the fibrous capsule contracts and blends with the amorphous material within it, calcification proceeds or begins, and the place of the suprarenal capsule is occupied by a small, irregular fibro-calcareous mass.

It follows, then, that the gross appearance of the capsules in any individual case of Addison's disease, with tuberculosis of the suprarenal capsules, will vary according to the age and extent of the process. The microscopic appearance will also vary. As long as the formed or forming tubercles are present the sections will show typical appearances. Even when the whole or greater part of the organ has become involved tubercules may yet be found in the capsule of the gland. It has been stated that giant cells are not always present; but this would seem to depend upon the stage to which the process had advanced. A time may ultimately be reached when no cellular elements can be found, yet this is not evidence of their previous absence.

Tubercle bacilli have been found sufficiently often, and the lesion is sufficiently characteristic to be pronounced definitely *tuberculosis*.

The lesion in the capsules may be secondary to a lesion elsewhere in the body, notably to caries of the vertebræ, or it may be primary.

Cases of tuberculosis of the suprarenal capsules are not Addison's disease, unless accompanied by symptoms.

Chronic interstitial inflammation of the suprarenal capsules, with subsequent or concomitant atrophy of the suprarenal parenchyma. The process is purely a chronic interstitial inflammation without tubercular

involvement, and is in all respects similar to fibrous processes in other organs; as, for example, in the liver and thyroid.

Neoplasms.—Addison's disease has been associated occasionally with tumors of the suprarenal capsules, such as sarcoma, carcinoma, and echinococcus cysts. In the case of sarcoma and carcinoma the new formations are generally secondary to a growth elsewhere in the body. Primary tumors of the suprarenal capsules do occur, however, and may prove a cause of Addison's disease. One or both of the suprarenal capsules may be affected. Secondary tumors of the suprarenal capsules present the microscopic appearances of the primary growth.

Simple Atrophy.—Partial atrophy of the suprarenal capsules is a normal process of old age.[1] Simple atrophy may occur, however, earlier in life and, reaching an extreme grade, be a cause of Addison's disease. The glands have more than once been reduced to the size of a split pea. In the cases of the Addison's disease described with congenital absence of the suprarenal capsules, it is supposed that the glands disappeared through atrophy or tuberculosis. The abdominal sympathetic nerves and ganglia are generally healthy in these cases.

Blood Cysts.—A few cases of Addison's disease have been associated with blood cysts or extravasations of blood into the substance of the suprarenal capsules. In some of these the extravasation has led to the development of a subsequent fibrosis. The lesions are believed to be due to trauma of the capsules, resulting from injuries received at birth or subsequently. One case has been described in an infant,[2] and Napier[3] has recorded a case in a man aged forty-six, a butcher by occupation. In both instances only one suprarenal capsule was affected.

Syphilitic lesions, gummata and fibrosis, have been found in the suprarenal capsules in several cases of Addison's disease.

Nervous System.—The lesions described in the nervous system in cases of Addison's disease have been chiefly lesions of the abdominal sympathetic nerves and ganglia. Lesions have been found, however, in the central nervous system. The changes in the abdominal sympathetic nerves (exclusive of the ganglia) occur as secondary lesions to the inflammatory process about the capsules. The nerve branches become entangled in the new connective-tissue formations, and suffer not only in their function, but even in their anatomical existence. Microscopically, the axis cylinders may be found in various stages of degeneration. These changes by no means, however, occur in every case of the disease, but only in those cases in which the inflammatory process has extended to the periphery of the organ. Jurgens and Fleiner have described degeneration of the splanchnics in Addison's disease.

Of the abdominal sympathetic ganglia, those of the solar plexus, and in particular the semilunar ganglia, have received most attention. Extended investigation into the condition of these ganglia in cases taken at random in the autopsy amphitheatre has discredited many reports on the diseased state of these structures. Thus, Hale White has shown the ganglion cells to be pigmented and atrophied in cases

[1] Rolleston: *Brit. Med Journ.*, 1895, vol. i.
[2] Cited by Thompson: *Amer. Journ. Med. Sci.*, Oct., 1893.
[3] *Glasgow Med. Journ.*, May 13, 1891.

not Addison's disease—that atrophy of the semilunar ganglia, in fact, is a process of old age. In some of his cases the fibrous tissue of the ganglion was increased in amount, and in others there was round-cell infiltration. Before him Lubimoff,[1] from a series of 250 examinations, had called attention to pigment deposits in the ganglion cells, sclerosis of the ganglion cells, round-cell infiltration of the connective tissue, and changes in the vessel walls. In none of the cases was Addison's disease the cause of death.

On the other hand, von Kahlden[2] has described the following changes in the semilunar ganglia in Addison's disease: atrophy of the ganglion cells and of their nuclei, with extensive pigment deposit in the protoplasm; hyaline degeneration of the vessel walls, with round-cell infiltration and hemorrhage into the external coat.

The changes in the semilunar ganglia so far described in cases of Addison's disease cannot be regarded as characteristic.

Lesions have also been found in the central nervous system. Kalindéro and Babes have described them in the cord and posterior roots in a typical case of Addison's disease. The anterior roots were normal. Certain portions of the anterior and posterior columns in the cervical and dorsal regions presented changes consisting in thickening of the neuroglia, sclerosis of the vessels, with numerous round cells and hyaline globes in the perivascular spaces. The posterior roots were the seat of neuritis, characterized by swelling of the axis cylinders, their interruption in places, and multiplication of cells. In Bourredi's case (cited by Tizzoni) there was hyperæmia of the cord, with perivascular inflammation and degenerative changes in the ganglion cells and nerve fibres.

Hypertrophy of the lymph follicles of the lower alimentary tract, referred to by Greenhow, is the only other lesion worthy of note in Addison's disease. It frequently, but by no means constantly, occurs.

Location of the Pigment.—The pigment granules are found in the lowermost cells of the rete Malpighii. A few pigmented cells, called "carrier cells," from their supposed function of conveying pigment granules to the epidermis, are present in the dermis. Mann states that the rete Malpighii contains no pigment in mucous membranes, but that when these are pigmented the granules lie in the corium, which likewise contains "carrier cells."

Pathogenesis.—Addison originally stated that the disease which now bears his name was due to lesions of the suprarenal capsules. He believed that any destructive lesion might produce the disease. Subsequently he altered his view, admitting a nerve element in its pathogenesis.

Recent investigation has shown the suprarenal capsules to possess an important physiological function. The results obtained have given new impetus to the study of the relation between abrogation of this function and the phenomena of Addison's disease.

The majority of cases of Addison's disease are associated with definite lesion of the suprarenal capsules without other notable pathological changes in the body. Moreover, in the majority of cases, the lesion has

[1] *Virchow's Archiv.*, lxi., S. 145–207.
[2] *Archiv. f. Path. Anat. und Phys.*, B. cxiv., H. 1.

replaced or otherwise rendered functionless the glandular tissue proper. In a minimum number of cases the suprarenal capsules have been found without demonstrable lesion. It remains, however, to be shown that the gland was functionating in these cases.

Successful removal of the suprarenal capsules in animals gives rise to symptoms somewhat analogous to those of Addison's disease,[1] and proves rapidly fatal. Brown-Séquard obtained these results in 1856. Other observers repeated his experiments, but with different results, and stated that the suprarenal capsules were not essential to life. The failures to produce symptoms after attempted removal of the glands have probably resulted from incomplete removal or the presence of accessory bodies. Accessory suprarenal capsules are commonly present in some animals and are sometimes found in men. Stilling[2] has shown by removal of one suprarenal capsule that the gland is capable of hypertrophy. It is possible this hypertrophy may extend to accessory bodies and prevent the incidence of symptoms after removal of the glands or in disease.

Langlois[3] states that if one sixth of the capsule by weight be left in a dog the animal survives.

Ligature of the suprarenal vein produces practically the same symptoms as removal of the gland (Boinet).

The 4 cases reported in which the suprarenal capsules have been stated to be congenitally absent in man, have been cases of Addison's disease.

F. and S. Marino-Zucco[4] inoculated the suprarenal capsules with pseudo-tubercle bacilli, and claim to have obtained the slow development of the symptoms of suprarenal removal and an increase in the pigmentation of the skin and hair of the animals experimented upon.

Aqueous extracts of the suprarenal capsules contain a substance which acts powerfully on the muscular tissues of the circulatory apparatus and skeletal muscles (Schäfer). Schäfer concludes from his experiments that the suprarenal capsules form an internal secretion, and that this substance is contained in it. He failed to find the active substance in the suprarenal capsules of a case of Addison's disease. Abel and Crawford have succeeded in separating a blood-pressure raising constituent from extracts of suprarenal capsules, which they believe belongs to the alkaloid group. It has been stated that animals from whom the suprarenal capsules have been removed, and that cases of Addison's disease, are benefited by hypodermic injections of suprarenal extract.[5]

From the above facts a new theory of the pathogenesis of Addison's disease has been put forward—the theory of *suprarenal inadequacy*. This theory supposes the existence of the suprarenal secretion and a beneficial action of this secretion in the economy. At this point opinion is divided. On the one hand, it is contended that the secretion of the suprarenal capsules has a directly beneficial action on muscular contraction, and perhaps on other functions of the body; on the other hand, that the secretion acts by virtue of antidotal or antagonistic properties,

[1] Abelous and Langlois, Tizzoni, Schäfer. [2] *Revue de médicine*, Paris, Oct., 1891.
[3] *La Bulletin méd.*, Paris, May 3, 1894.
[4] *Schmidt's Jahrbücher*, Leipzig, Aug. 13, 1894.
[5] Zyboulski: *La Presse médicale*, Paris, June 8, 1895.

rendering inert a toxin or toxins developed during metabolism—*the auto-intoxication theory.*

Theory of the Direct Beneficial Action of the Suprarenal Secretion.—This theory is supported by analogy. Schäfer found that injections of suprarenal extract produced marked effects upon the muscular layer of the arteries, the muscular tissue of the heart, and the skeletal muscles. The action is direct, occurring when salt solution is substituted for blood. The muscular layer of the arteries is markedly contracted, causing a rise of blood pressure. When the heart is freed from nervous control its contractions are increased both in frequency and force, still further raising blood pressure. The contraction of skeletal muscles in response to a single stimulus is much prolonged.

Very small doses of suprarenal extract are sufficient to produce marked effects; thus Schäfer states that less than $\frac{1}{13000}$ gramme (one $\frac{1}{800}$ grain) of the desiccated gland is sufficient to produce an effect upon the heart and arteries of an adult man.

Since the suprarenal capsules contain a constituent which acts so powerfully upon the vascular (including heart) and general muscular systems, and since Addison's disease is prominently characterized by loss of vascular and cardiac tone and muscular weakness, it is assumed that Addison's disease is caused, at least in part, by suspension of the suprarenal secretion. The facts of its pathological anatomy are not opposed to this theory. The majority of cases of the disease show partial or complete destruction of the suprarenal capsules as secreting organs. On the contrary, it may be doubted if a case has ever occurred with extensive destruction of both suprarenal capsules of long standing without the symptoms of Addison's disease. If so, it is possible that accessory bodies prevented the incidence of symptoms or that the entire gland had not been destroyed. The production of the disease when the medulla only or chiefly has been involved in the lesion, is explained by the fact that the active principle of the suprarenal capsules is contained only in the medulla of the gland. A lesion which, though small in itself, compresses or occludes the suprarenal vein, might produce the same effects as a more generally destructive lesion.

While this theory may be regarded as a distinct advance toward an explanation of the pathogenesis of Addison's disease, and as resting upon a scientific basis, it cannot be regarded as conclusive. If the theory is correct, blood from the suprarenal vein, when injected into animals, should produce the same effects as injections of suprarenal extract. Zyboulski states that the blood from the suprarenal vein does produce these effects, but Schäfer has failed to confirm his results, though he used as much as 10 c.c. of blood in certain cases. Schäfer states, however, that the converse effects of suprarenal removal and injections of suprarenal extract confirm the theory of internal secretion.

Auto-intoxication Theory.—This theory admits the presence of the suprarenal secretion, but supposes its action to be indirect rather than direct. According to the theory, the suprarenal secretion neutralizes or in some manner renders inert a toxin or toxins developed normally in the body, and in the absence of the secretion a condition of toxæmia is induced.

The theory is based largely upon experiments performed by Brown-Séquard and Abelous and Langlois. They have found that the blood of animals dying from suprarenal removal is toxic for other animals from whom the glands have been removed. Schäfer justly argues that the blood from a moribund animal, whatever the cause, would probably be injurious to another animal suffering from the effects of a capital operation. The experiments are not a sufficient justification of the theory. Against it we have the positive action of injections of suprarenal extracts and the converse condition of Addison's disease.

Objections may be raised to both the suprarenal inadequacy theories. Cases of Addison's disease have occurred without disease of the suprarenal capsules. These cases have been explained on the score of functional inactivity on the part of the glands. But it is difficult to conceive of a functional inactivity extending over the period of time necessary for the development of the average case of Addison's disease. The same objection holds when only one gland has become diseased. A suspension of function must be supposed in the other gland. Entire destruction of one gland is not sufficient to produce the disease, as several of my autopsies prove. Again, it may be argued that extensive destruction of the suprarenal capsules by tumor formations has been observed without the symptoms of Addison's disease. Tumors of the suprarenal capsules, however, are generally secondary, and the primary growth is generally far advanced when the suprarenal metastasis occurs, so that death may occur from the primary growth before the symptoms of Addison's disease have had time to develop; in other words, before destruction of the suprarenal capsule as a secreting organ is complete. Destruction of the suprarenal capsules by secondary tumor formations is generally not as complete as in the case of tuberculosis. The fact that tumors of the suprarenal capsules have been known to produce typical symptoms of Addison's disease, has an important bearing in this connection.

The Nervous Theory.—In 1854, Kölliker expressed the opinion from anatomical studies that the suprarenal capsules consisted of two physiologically distinct portions—the cortex or secreting portion and the medulla or nervous portion.

About the same time, Addison's famous memoir appeared, in which he stated that the disease he described was due to abrogation of the function of the suprarenal capsules.

Brown-Séquard then studied the effects of suprarenal removal, and stated that their removal was invariably and rapidly fatal. Harley, Phillipeaux, Schiff, and others repeated the experiments, but failed to confirm the results, and it was concluded that the suprarenal capsules possessed no function, or, at any rate, that their function was not known.

Furthermore, cases of Addison's disease were now described in which no lesion of the suprarenal capsule had been found, and other cases in which, though the suprarenal capsules were diseased, the ganglia and branches of the abdominal sympathetic nerves were involved in secondary interstitial formations.

Schiff and some others of the earlier experimenters had observed diarrhœa and vascular disturbances as a result of extirpation of the

suprarenal capsules. Similar results followed extirpation of the abdominal sympathetic nerves and ganglia.

Riesel argued that the morbid phenomena were the result of reflex nerve disturbances, the irritation starting in the branches of the abdominal sympathetic nerves. He cited in evidence Goltz's experiment of stopping the heart of the frog by forcibly striking the intestines.

Wilks and Greenhow believed that Addison's disease was of nervous origin. They contended that the morbid phenomena were reflex, due to irritation of the abdominal sympathetic nerves and ganglia entangled in the fibro-caseous masses into which the suprarenal capsules had become converted. Greenhow separated the changes in the nerves into two stages—the stage of irritation and the stage of atrophy. Wilks believed that only one lesion of the suprarenal capsules, the fibro-caseous metamorphosis, was capable of producing the disease.

Arnand and Alexais[1] accept the nervous theory, but limit the morbid action to the ganglia in the capsule of the suprarenals.

To sum up: The supporters of the nervous theory contend that the symptoms of Addison's disease are, in the main, reflex phenomena, resulting from irritation or destruction of the abdominal sympathetic ganglia or nerves. It has also been suggested in cases where there was neither disease of the suprarenal capsules nor of the nerves or ganglia, that Addison's disease has occurred from functional disturbances of the nervous system.

There are several objections to the nervous theory. Numerous cases of Addison's disease have occurred without involvement of the abdominal sympathetic nerves or ganglia, and that, too, where the suprarenal capsules had been destroyed as functionating organs. Such cases are those in which simple atrophy of the suprarenal capsules has been found, and cases in which the medulla only or chiefly has been involved in the pathological process. Moreover, extensive lesions of the abdominal sympathetic nerves have repeatedly occurred without the symptoms of Addison's disease; nor is extirpation of the cœliac plexus or branches of the abdominal sympathetic nerves followed by the disease.

The nervous theory may be regarded as the negative side of the pathogenesis of the disease, it being assumed that the suprarenal capsules possess no function. Yet a nervous element in the pathogenesis of the disease cannot be entirely disregarded. Extirpation of the cœliac plexus and branches of the abdominal sympathetic nerves does produce symptoms which are similar to some of the symptoms of Addison's disease—abdominal vascular disturbances. And in the present state of our knowledge one is inclined to explain the genesis of the disease as partly the result of nerve disturbances and partly the result of interference with the function of the suprarenal capsules. The double origin cannot be claimed in every case. Some cases present chiefly loss of cardiac and vascular tone with muscular weakness, and can be accounted for by the absence of the suprarenal secretion.

The theory that Addison's disease is the result of an interrupted *excretory function* on the part of the suprarenal capsules is untenable.

Symptoms.—*Onset.*—The symptoms of Addison's disease develop slowly. Ease of fatigue and a gradually increasing indisposition to

[1] *Marseille médicale*, Marseilles, Jan. 30, Feb. 28, Mar. 30.

exertion are generally the first symptoms of which the patient complains. The indisposition may have extended back over weeks or months when the patient comes under observation. Anorexia, with more or less gastric disturbance, is frequently an early symptom. There may be actual vomiting. Sometimes pigmentation of the skin precedes the other symptoms. At times, the patient seems to become suddenly ill, and dies after a few weeks, or months, at most. It may be doubted, however, if the disease ever develops suddenly; and in cases where it appears to it is probable that the patient has disregarded such symptoms as have been present—that the apparently acute onset is but an exacerbation in the course of the malady. The usual slow development of the lesion in the capsules lends weight to this belief, and the insidious development of the disease becomes explicable in the light of the suprarenal inadequacy theory.

The *chief symptoms* to be noted in a case of Addison's disease are asthenia, circulatory disturbances, pigmentation of the skin and mucous membranes, and alimentary and nervous disturbances. They are not all equally prominent in every case of Addison's disease, however; thus all cases are not pigmented.

Asthenia is perhaps the most marked feature of the disease in the majority of cases. In common with circulatory disturbances it is probably the most constant symptom. Patients generally come to their physician complaining of a gradual failing of strength extending over many weeks and months. The loss of muscular power has steadily progressed. At first there has been felt only a disinclination to exertion, the patient becoming easily fatigued. Later, when the muscular weakness has become more pronounced, there is total inability for sustained effort.

Circulatory disturbances appear early in nearly every case. There is distinct loss of cardiac and vascular tone. The heart action becomes feeble, and the pulse becomes frequent, small, and easily compressible. Functional cardiac murmurs are sometimes heard. The circulatory disturbances may manifest themselves in many ways. Since there is loss of cardiac and vascular tone, an equal circuit of the blood is not maintained. The brain receives a deficient supply, partly because of the enfeebled heart action, partly because of the loss of tone in the cerebral vessels. This anæmia is so marked and is so easily intensified that patients in the later stages of the disease are not able to get up, or even to sit up in bed, without a tendency to, or actual, syncope. Death has been known to follow immediately upon suddenly sitting up in bed.

Dyspnœa on slight exertion is also a troublesome and distressing symptom. It cannot be accounted for by anæmia, since a diminution in the number of red blood cells may, but does not necessarily, occur. It is due to defective circulation through the lungs. If anæmia be present, and especially if it be marked, the respiratory difficulty will be still further enhanced.

Circulatory disturbances in the alimentary tract lead to imperfect digestion and interference with the regularity of the bowels. Loss of tone in the abdominal vascular area is serious because of the enormous capacity of the abdominal vessels. A rabbit, for example, may be bled

to death in his own abdominal cavity by ligating the portal vein, and that without a drop of blood leaving the vessels.

The cause of the cardiac and vascular disturbances seems to be two-fold, and is probably best accounted for through the combined action of the suprarenal inadequacy and nervous theories. Personally, it is believed that the former is the more potent though not exclusive factor, and for the reason that circulatory disturbances occur in cases of Addison's disease in which the abdominal sympathetic nerves are not involved. A reference to the action of the suprarenal extract will readily explain how loss of circulatory and systemic muscular tone may occur as a result of the absence of the suprarenal secretion. It is not denied, however, from the known interaction between the circulatory apparatus and abdominal sympathetic nerves, that irritation in the latter may lead to marked disturbances in the former.

Pigmentation of the skin and mucous membranes does not occur in every case of Addison's disease. Though prominent when present, it cannot be considered an essential symptom of the disease. A moderately large number of non-pigmented cases have occurred; thus, Greenhow found 29 in a series of 228 cases, and since his monograph appeared other cases have been reported.[1] Pigmentation may appear as the first or as a later symptom of the disease.

The discoloration generally begins on the exposed parts of the body —on the face and neck, backs of the hands, and extensor surfaces of the arms. Thence it extends to other parts of the body—first, and with greatest intensity, to those parts which are normally pigmented; in fact, the pigmentation may be regarded as a pathological exaggeration of a physiological process. Fagge has raised the question as to whether pigmentation would occur if the patient were confined in the dark.

When pigmentation begins it may take the form of a diffused darkening of the skin or be distributed over the surface in patches or streaks, and when it has once begun it generally progressively, though not uniformly, deepens. The intensity of the pigmentation has been observed to vary with remissions and exacerbations of the disease. With the exception of the sclerotics, the skin of the scalp, the palms of the hands and soles of the feet, the whole body may become discolored. It has also been stated that the skin under a beard may escape pigmentation, but that this is not always true is evidenced by a case recently under my observation. The pigmentation is always most intense in those parts of the body normally pigmented—the folds of the axillæ, the areola of the nipple, the external genitals, and the groin. Over the inner surfaces of the arms and thighs the pigmentation is usually less marked or absent.

The color of the pigmented parts varies with the intensity of the pigmentation from a light yellowish or greenish-brown to a dark-brownish or even black tint, forming a strong contrast to the sclerotics. Fagge has compared the color to that of one or another of the colored races of mankind according to the depth of discoloration. While the pigmentation is not uniformly deep in all parts of the body, there is never a sharp line of demarcation between the pigmented and unpigmented portions. The one merges almost imperceptibly into the other. Greenhow has

[1] Fenwick: *Trans. Path. Soc.*, London, vol. xxxiii.

PLATE VIII.

Addison's Disease, Showing Bronzing of Skin, and White Areas of Atrophy. (Personal Observation.)

called attention to darker mole-like spots in pigmented areas which he regards as very characteristic.

The color is often deeper on parts of the skin subjected to irritation or pressure. Thus a distinct line will often be found about the neck, where the collar has pressed; about the waist, where the weight of the clothing has fallen; in men, over the shoulders where the suspenders have passed; and in women, around the legs at the site of the garters. Likewise, the application of a blister will be followed by a deeper pigmentation. It is a remarkable fact that pigmentation of the palms of the hands and soles of the feet does not occur, though they are subjected to the greatest pressure. It is to be noted that pigmentation is less marked in these situations in the colored races of mankind. Scars generally remain unpigmented, but they may become discolored.

The pigmentation sometimes disappears over patches of varying size —leucoderma. This condition is very distinctly shown in Plate VIII. The cause of the disappearance of the pigment is not known.

Some of the *mucous surfaces* undergo pigmentation similar to that of the skin. The surfaces most frequently pigmented are the mucous membranes of the mouth and vagina. The pigmented areas are of a bluish or blackish color. It has been said that the determination of pigment to this or that part of the mucous membrane of the mouth is brought about through irritation of the teeth; thus on the tongue the pigmented areas will be found along its free border, chiefly where it has impinged upon the teeth, and more particularly so where a tooth is decayed and irregular. A line of pigmentation is often found along the margin of the lips where they come together and at the same time press against the teeth. In the vagina the pigmentation occurs chiefly on the mucous membrane of the labia minora and in the vaginal canal proper. Here the pigmentation cannot be explained on the score of irritation.

The alimentary disturbances relate chiefly to anorexia, imperfect gastric digestion, nausea and vomiting, and diarrhœa or constipation. Anorexia and digestive disturbances are often early symptoms. Nausea and vomiting may occur early, though they commonly are late symptoms. Vomiting comes on in almost every case in the later stages, and is rebellious to treatment, if not uncontrollable. The vomiting is particularly distressing because of the already depressed condition of the patient. The vomitus consists largely of mucus and regurgitated bile with the contents of the stomach—partially digested or undigested food. The vomiting may be so severe, so uncontrollable, and so continuous that the patient dies from exhaustion. Cardialgia sometimes occurs.

The bowels are generally constipated from atony of the intestinal wall, probably also from local vascular disturbances. More or less severe diarrhœa may alternate with the constipation.

The *nervous symptoms* are neither pronounced nor numerous, though there may be general depression of the nervous system. The mind usually remains clear to the end, though the memory is often impaired. Death may take place in coma or semi-coma, though in the latter condition the patient will answer questions slowly but pertinently. Headache and vertigo are frequently present. Sight and hearing may be impaired. Pain is often present. It may be located in the loins or in the joints. The lumbar pains are dragging in character, and are the result

of extension of the inflammatory process from the suprarenal capsules to the circumjacent tissues. The arthritic pains are often excruciating, and sometimes resemble rheumatism in that suffering is occasioned by movement or even a jar from striking the bed. Addison's disease has more than once been diagnosed as rheumatism from the location and intensity of the pains. Rheumatism may readily be excluded, however, by the absence of active inflammatory symptoms in the joints. The larger joints, particularly the knees, are the ones most often affected.

Miscellaneous Symptoms.—The temperature in uncomplicated cases of Addison's disease is commonly below normal; sometimes the temperature rises in the later stages of the disease. If Addison's disease is complicated by a disease characterized by pyrexia, the thermometric record will be in accordance. A notable fact in Addison's disease is that patients often retain their subcutaneous fat to the end. Sometimes, however, they become emaciated. Emaciation occurs chiefly in connection with marked gastro-intestinal symptoms—cases in which digestion is imperfect.

Anæmia may be present, but is not a feature of the disease. Thus in two cases under observation during the past summer at Charity Hospital,[1] the counts of red cells were 1,490,850 and 2,484,750 respectively, with hæmoglobin (v. Fleischl), 20 per cent. in the former and 45 in the latter. In the former case the white blood cells were decreased in number, and in the latter slightly increased.[2] Neuman,[3] Tschirkoff,[4] and Cabot[5] have also reported cases with blood counts below normal. Tschirkoff has stated that the only change found in the white cells is a pigmentation with black granules. The number of red cells may vary with improvement or aggravation in the patient's condition. In one of the cases reported by Cabot, the red cells increased from 2,196,000 to 4,700,000 in one month under the exhibition of suprarenal extract. Osler states that the blood count is generally 50 per cent. to 60 per cent. of the normal.

The urine, though generally normal in amount, may be diminished or increased in quantity. It does not, as a rule, contain any abnormal ingredient. Urea is diminished from defective metabolism. Marino-Zucco and Dutto[6] have called attention to the presence of neurin in the urine, to which, in the blood, they attribute the symptoms of Addison's disease, believing the disease to be an auto-intoxication.

Course.—Addison's disease proceeds progressively, though not uniformly, from the incidence of symptoms to a slowly fatal termination. Exacerbations and remissions may recur from time to time. After an exacerbation the patient rarely reaches the state of health possessed before the exacerbations occurred. He may improve temporarily, but the tendency is toward a progressively fatal termination.

The course of any given case will vary according to the symptoms which are most pronounced. Non-pigmented cases occur with moderate frequency and remain non-pigmented to the end of the disease. In

[1] Studied through the kindness of Drs. C. A. Herter and D. H. McAlpin.
[2] Blood counts made by House Physician, Dr. Thomas F. Reilly.
[3] *Deut. med. Woch.*, 1894, 105.
[4] *Zeit. f. klin. med.*, 1891, vol. xix. suppl., Heft. xxxvii.
[5] *Clinical Examination of the Blood.* [6] *Bull. della r. Accad. med. di Roma*, 1891.

other cases pigmentation is the first symptom to make its appearance—at any rate, the first symptom to be observed—and may be the only symptom for months. Later, the constitutional symptoms develop.

However the disease may begin, the majority of cases present a typical picture sooner or later, and proceed with a fair degree of uniformity to a fatal termination.

Duration.—Addison's disease is of variable though long duration. The average duration in a series of cases collected by Wilks was eighteen months. Among the cases of shortest duration is one of a girl who attended school till within a week of her death (Greenhow). On the other extreme, cases have been reported extending over periods of six years (Niemeyer), seven years (Fagge), and ten years (Greenhow). Fenwick,[1] from an analysis of 30 cases, has called attention to the fact that non-pigmented cases are of shorter duration than pigmented. The average of the former in his series was 4.8 months, of the latter 23.6 months.

It is manifestly not justifiable to take the length of time that a case is under observation as a measure of the duration of the disease. The fact that a patient has not yielded to his symptoms or has not consulted his physician does not indicate the absence of symptoms. It only indicates the degree of their severity. And it is probable in the cases of apparently short duration that symptoms have been present for many weeks or months. The condition of the suprarenal capsules often argues for such a belief. As Wilks has said, we have no data from which to estimate the length of time necessary for the destruction of the suprarenal capsules by the tubercular process, but, judging from analogy in other parts of the body, it is probably long. A case reported by the writer was under observation only a little more than three weeks, but the condition of the suprarenal capsules pointed to their complete destruction months before.

Termination.—Death in Addison's disease may occur from progressive asthenia, from exhaustion due to uncontrollable vomiting, in syncope, or from cardiac failure following slight exertion. Sometimes coma or semi-coma immediately precedes the fatal issue, or convulsions or delirium may supervene. Except in coma or delirium the mind remains clear to the end.

Prognosis.—The prognosis in Addison's disease is unfavorable, though the disease is now believed not to be invariably fatal. Scant hope is offered in any case, however, unless there is reason to think that the morbid process in the suprarenal capsules can be arrested. It may be definitely assumed, for the present at least, that complete destruction of the suprarenal capsules will ultimately cause death. A few cases of Addison's disease have been reported as cured, one under antisyphilitic treatment. The number of cases, however, which have recovered is so small as scarcely to influence the prognosis. The large majority of cases terminate fatally, and the prognosis in any given case should be rendered accordingly.

Diagnosis.—When Addison's disease is fully developed the diagnosis is not difficult. Much difficulty, however, arises in the early stages and in those cases which follow an atypical course. The diag-

[1] *Trans. Path. Soc.*, London, vol. xxxiii.

nosis in typical cases rests upon the association of a peculiar pigmentation of the skin and mucous membranes, with progressive asthenia, loss of cardiac and vascular tone, and alimentary disturbances. Such a symptom-complex is not presented by any other disease. With a history of slowly failing strength, gradually developing discoloration of the skin, beginning on the face and hands and most pronounced in situations normally pigmented, associated with weak heart action, a small, easily compressible pulse, a tendency to syncope on assuming the sitting posture or getting out of bed, and perhaps nausea and vomiting, there is no escape from the diagnosis of Addison's disease.

In the early stages, however, before the symptoms have become pronounced, before the discoloration of the skin has begun or as it is just beginning, a diagnosis cannot be made with certainty. At best the diagnosis is only probable, and is made by exclusion. It remains for the subsequent course of the disease to confirm or refute it.

Pigmentation is not an essential symptom, yet from its usual presence the greatest difficulty of diagnosis is offered by non-pigmented cases. It is not an uncommon experience in these cases that the diagnosis is made at the autopsy table. Moreover, in non-pigmented cases the difficulty of diagnosis is still further increased by the apparently rapid course of the malady.

Addison's disease is not the only disease or condition in which pigmentation of the skin occurs. Thus pigmentation may be present in chronic pulmonary tuberculosis, chronic malarial cachexia, pregnancy, chronic uterine disease, tubercular and cancerous peritonitis, melano-sarcoma, syphilis, *diabète bronzé*, so-called vagabond's disease, pancreatic disease, and after the continued administration of arsenic and silver. Again, pigmentation of the skin may result from exposure and occupation.

It is not in all these conditions that a differential diagnosis will have to be made. Difficulty of diagnosis will be offered by the following diseases, however:

It is a question sometimes in chronic pulmonary tuberculosis, from a condition of asthenia out of proportion to the extent of pulmonary involvement, if the suprarenal capsules have not become secondarily invaded, and especially so where there is concomitant pigmentation of the skin of the face. The pulmonary signs do not exclude suprarenal involvement. In these cases a diagnosis cannot be made unless the patient lives long enough for the typical symptoms of Addison's disease to develop.

Tubercular or cancerous peritonitis, with pigmentation of the face and perhaps of the mucous membranes, may simulate Addison's disease to such an extent as to make a positive diagnosis very difficult.

Several cases of melano-sarcoma with intense pigmentation of the skin have been reported, the pigmentation being entirely unassociated with cutaneous tumor formation. In these cases the pigment deposits occupy the same layers of the epidermis as in Addison's disease.[1] If there is a visible or palpable tumor, the diagnosis may be made with little difficulty.

In pancreatic disease with discoloration of the skin, a diagnosis

[1] Wagner: *Archiv der Heilkunde*, Bd. v.

will be possible only after careful and continued observation of the case.

Addison's disease may be differentiated from *diabète bronzé*, a condition of hypertrophic cirrhosis, diabetes, and pigmentation of the skin, by an examination of the urine and liver region.

The diagnosis of Addison's disease in one of the colored races of mankind is a matter of exceeding difficulty.

TREATMENT.—The treatment of Addison's disease may be divided into two parts: (1) treatment with suprarenal extract, and (2) symptomatic treatment.

Until the researches of Schäfer and Oliver made known the function of the suprarenal capsules, the treatment of Addison's disease was wholly symptomatic. During the past few years, however, an attempt has been made to treat the disease with extracts of the suprarenal capsules. The results so far attained have been only partially successful. Moreover, it is difficult to judge of the efficacy of any form of treatment because of the long duration and irregular course of the malady. Ringer and Phear have analyzed the results obtained in the reported cases of Addison's disease treated with suprarenal extract. In some of the cases there has been distinct amelioration of the symptoms with diminution of the intensity of the pigmentation. The improvement, however, has not remained uniform with the continued administration of the extract, and at times, in spite of its exhibition, the cases have grown progressively worse. The results of treatment with suprarenal extract, then, have been rather discouraging, but the treatment should be persisted in until it shall have been proved valueless or positively harmful. Osler has recorded the death of a case while under the exhibition of suprarenal extract, but he expresses the opinion that death was in no way connected with the treatment. Other cases have died with similar symptoms—collapse and delirium—while not under suprarenal extract. Byrom Bramwell believes that the treatment of Addison's disease will not, and cannot, be uniformly successful, because of the double source of origin—glandular and nervous—of many cases of the disease. Manifestly, suprarenal extract will not benefit, or but partially, those cases which owe their origin partly to reflex disturbances from the abdominal sympathetic ganglia and nerves. Addison's disease is only in part analogous to myxœdema, in that the latter is definitely proved to be due to a single cause—a suspension of the secretion of the thyroid gland.

The treatment of Addison's disease with suprarenal extract was begun by hypodermic injections of the extract. The hypodermic method, however, is open to the objections of being troublesome and attended with the danger of pyogenic infection, except in skilful hands. Since the active constituent or constituents of the suprarenal capsules are not affected by the gastric secretion (Schäfer), the glands as such, or preferably in the form of an extract or pill of the dried and powdered glands, may be administered by mouth. When an extract of the suprarenal capsules is used—*e. g.* the glycerine extract—a quantity equivalent to one half of a single gland may be given three times a day, or, if the dried powder be employed, 15 grains (1 gramme) may be given in capsule similarly. In either case it has been found advis-

able to gradually increase the dose. As much as 2 drachms have been administered to a patient not only without harmful, but with distinctly beneficial, result.

The *symptomatic treatment* of Addison's disease consists simply in ameliorating such symptoms as arise. It will not be necessary to keep a patient confined to bed until the later stages of the disease, when the asthenia becomes pronounced and the cardiac action weak, irrespective of the rapidity with which this condition may be reached. When confinement to bed has once become necessary, it is inadvisable to insist on the patient's getting up, or even to allow it, since death may occur from syncope.

In all instances the diet should be nutritious and easily assimilable. A liquid diet is not indicated unless there be gastro-intestinal irritability. Constipation should be treated preferably with calomel, followed by salines, or with salines alone. Drastic hydragogues must not be employed. When diarrhœa is present it may be overcome with opium combined with vegetable or mineral astringents. In every case of diarrhœa it is important to assure one's self that the diarrhœa is not caused by scybalous or other masses of retained fæces.

Vomiting is often, and in the later stages almost always, a difficult symptom to control. One may run the gamut of antiemetics without avail. It will be advisable to begin with the simpler gastric sedatives, as bits of cracked ice, held in the mouth, without swallowing the water; Apollinaris, Vichy, seltzer waters; small doses of bismuth suspended in mucilage of acacia; sodium bicarbonate, etc. If these fail, resort should be had to morphine, cocaine, and cerium oxalate. In all cases of persistent vomiting the diet should be restricted to milk or milk and lime-water or milk and Vichy, half and half, in small quantities at frequent intervals; or peptonized or pancreatized milk may be tried.

Aside from the above details, the treatment of any case of Addison's disease should be conducted along general lines.

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